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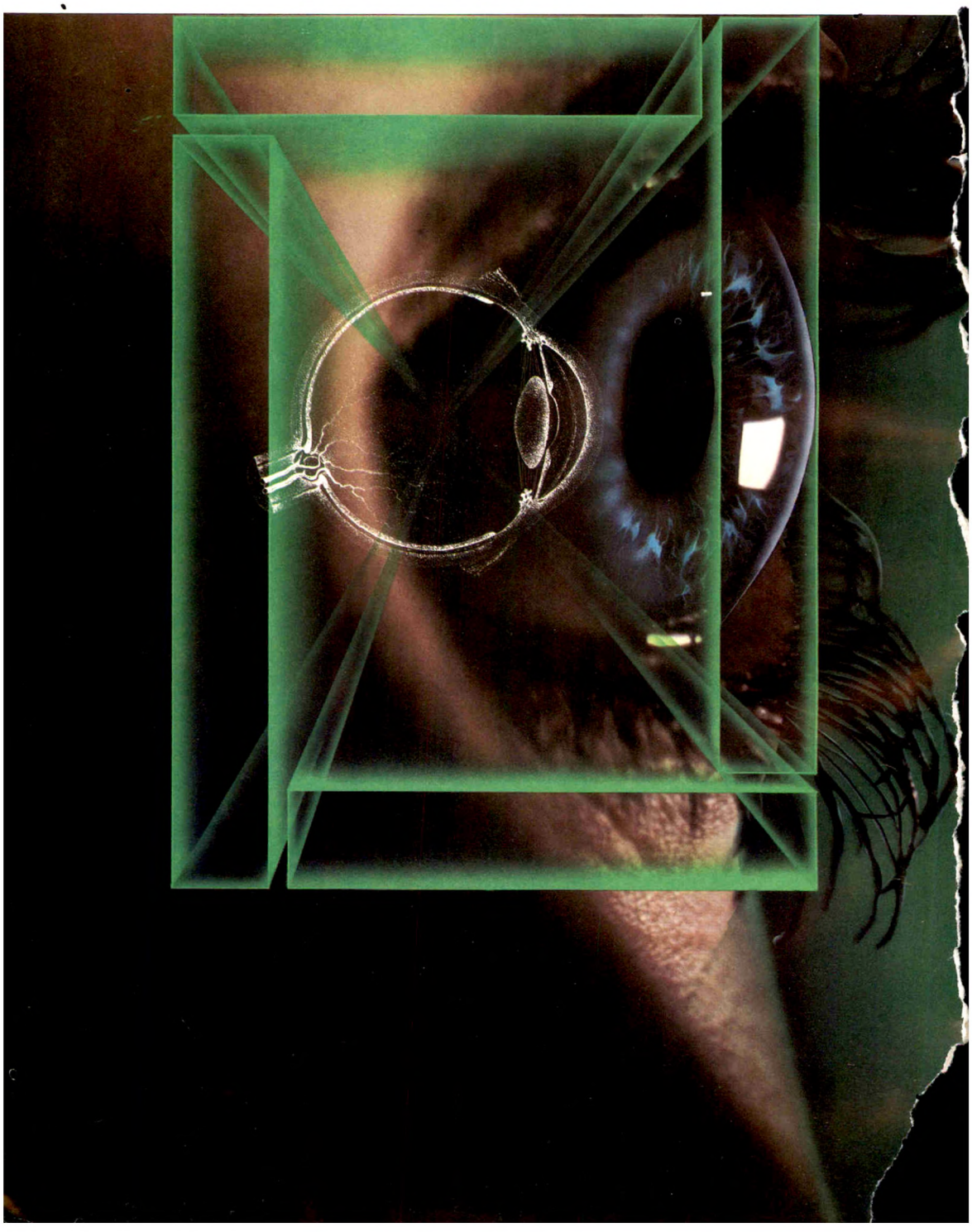
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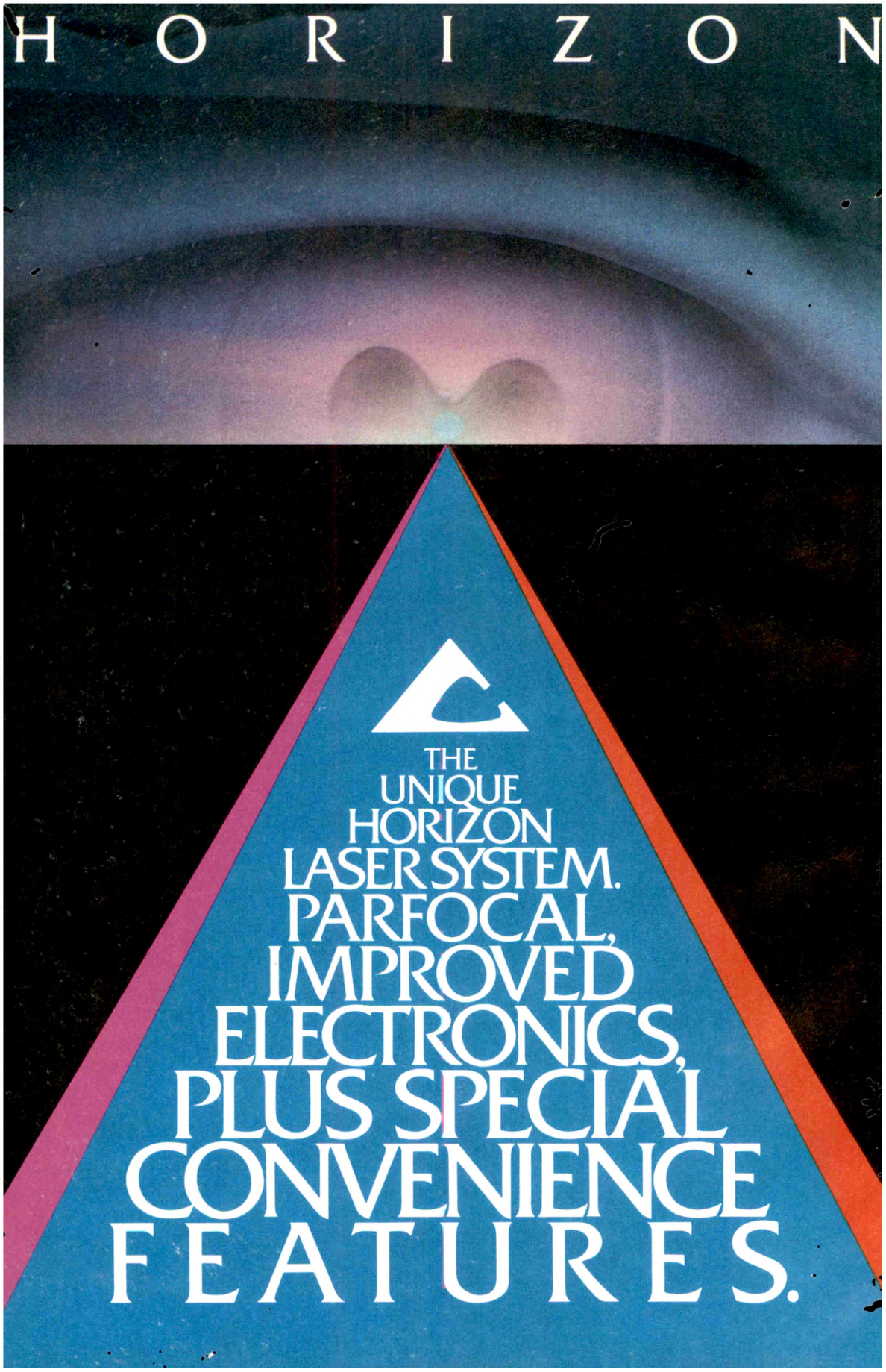
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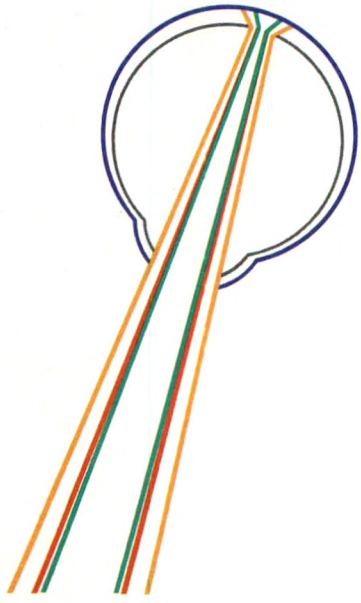
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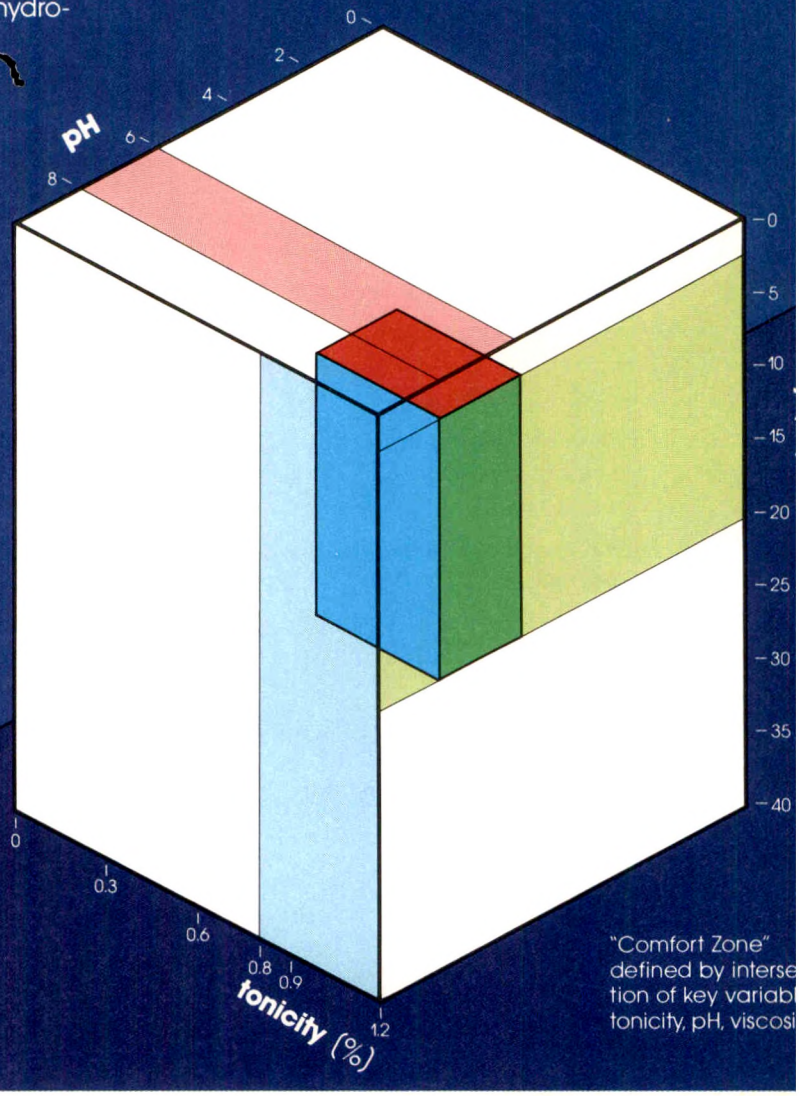
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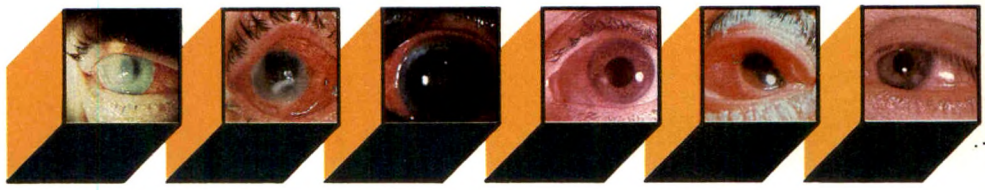
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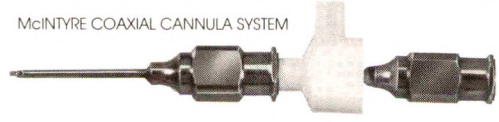
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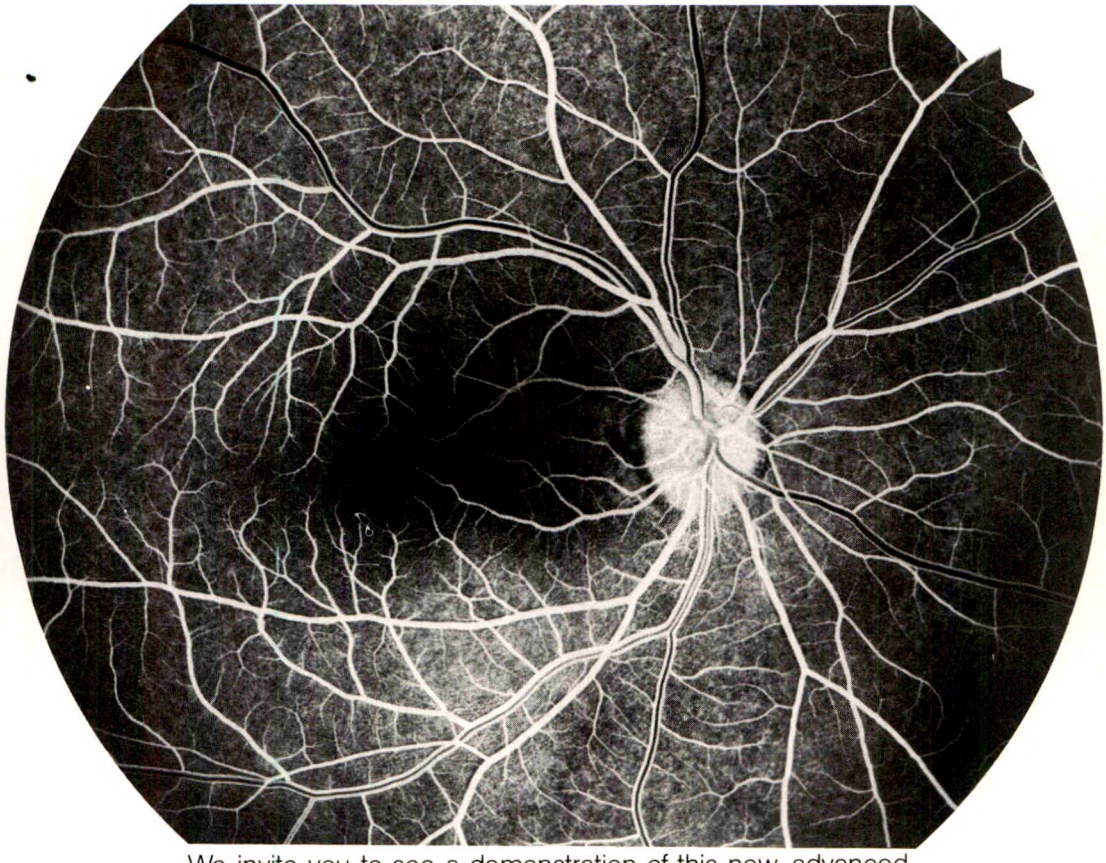
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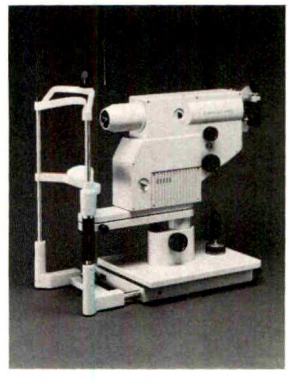
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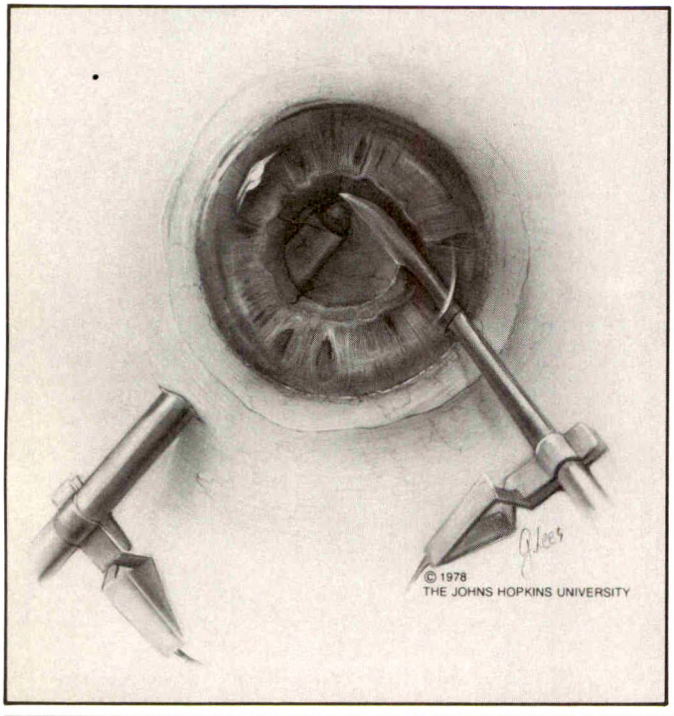
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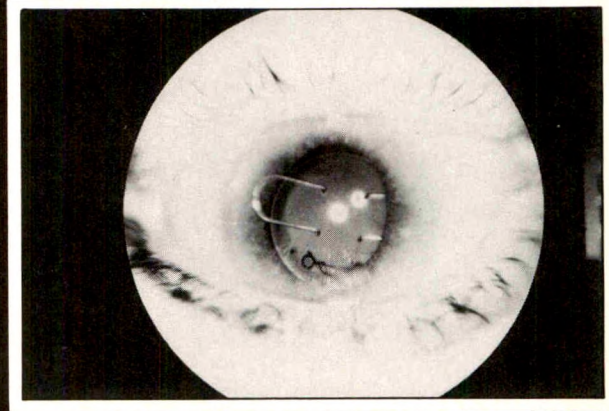


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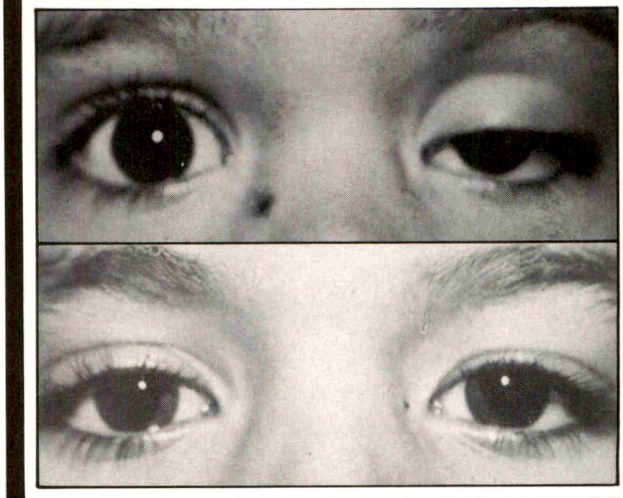


Fig. 13-20. A, Sever unilateral "congenital" ptosis. **B,** Apparent good result by fascia lata brow suspension.

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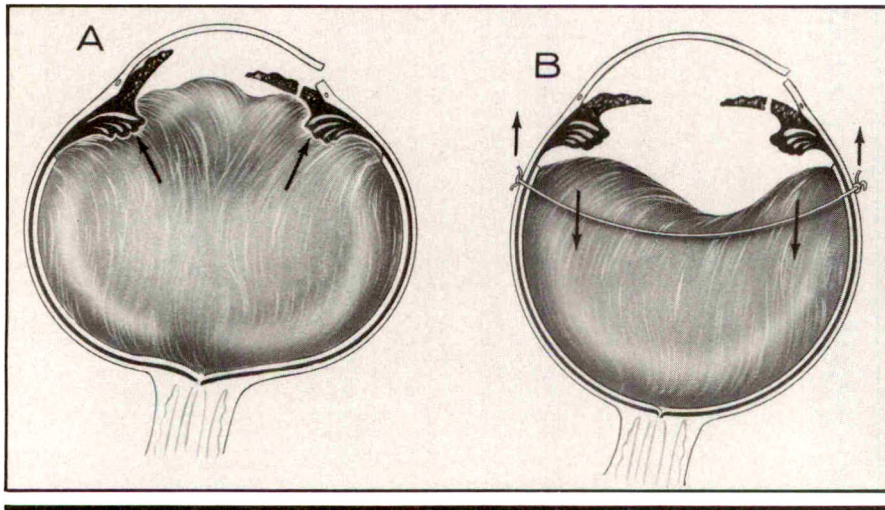
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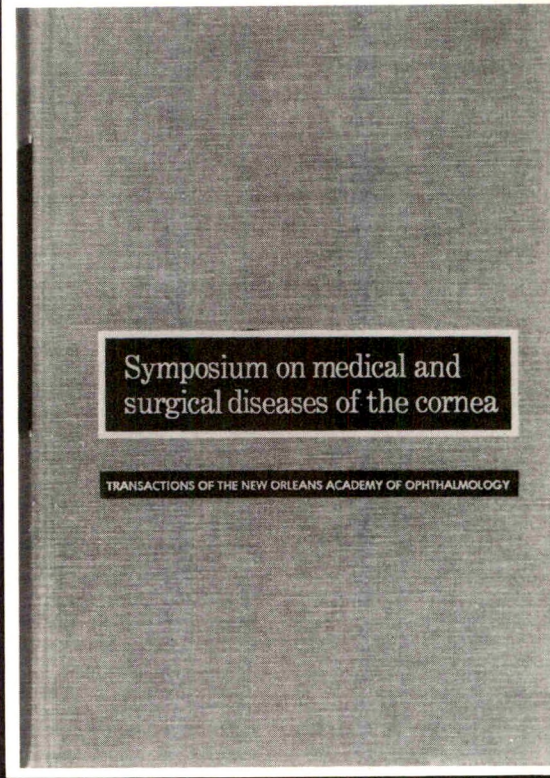
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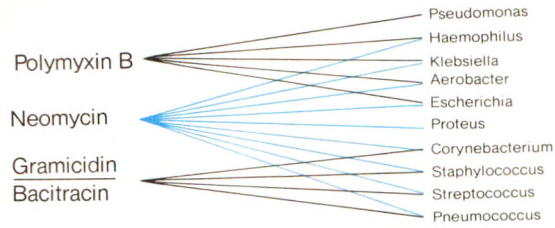
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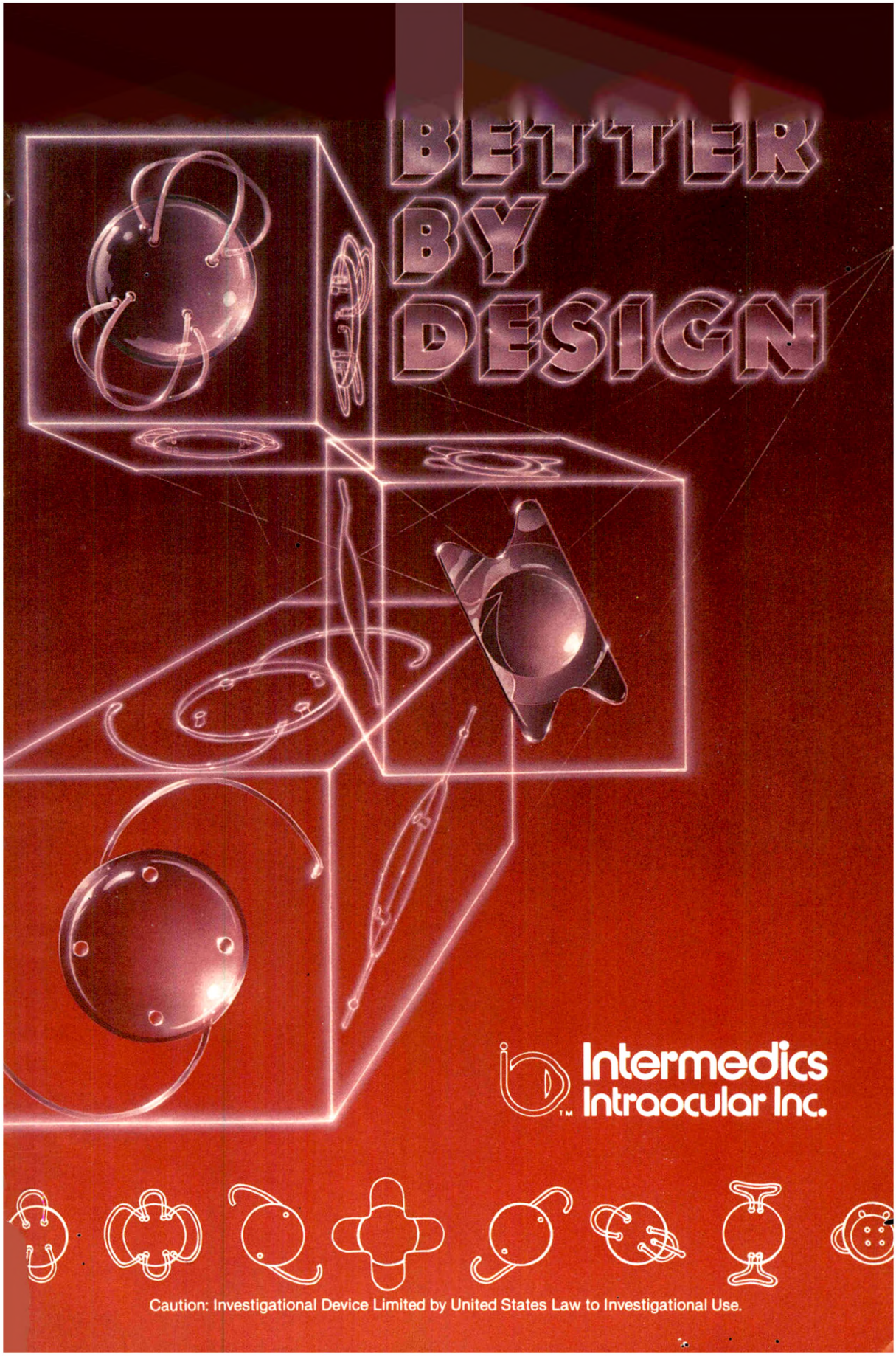
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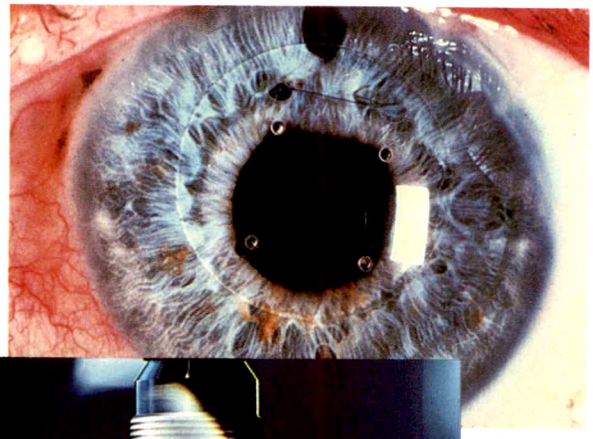
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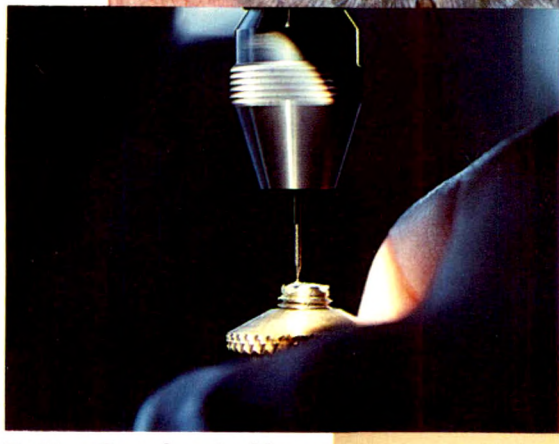
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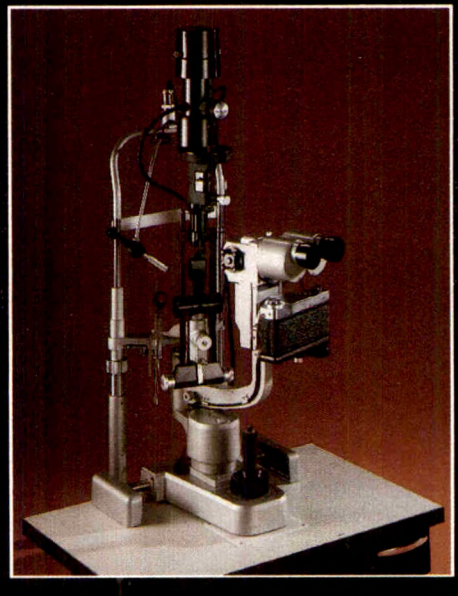
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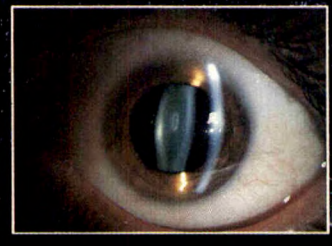
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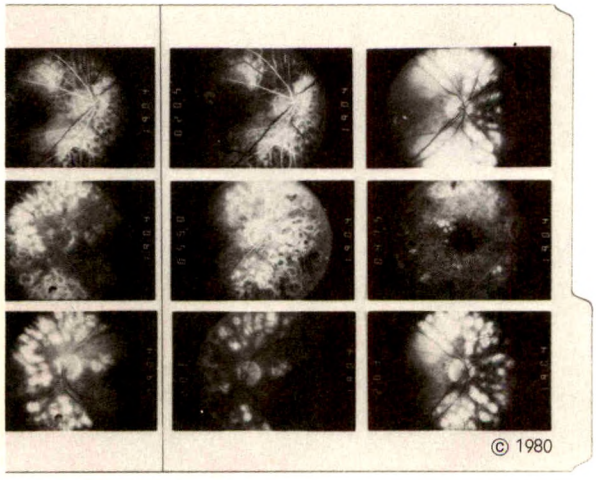
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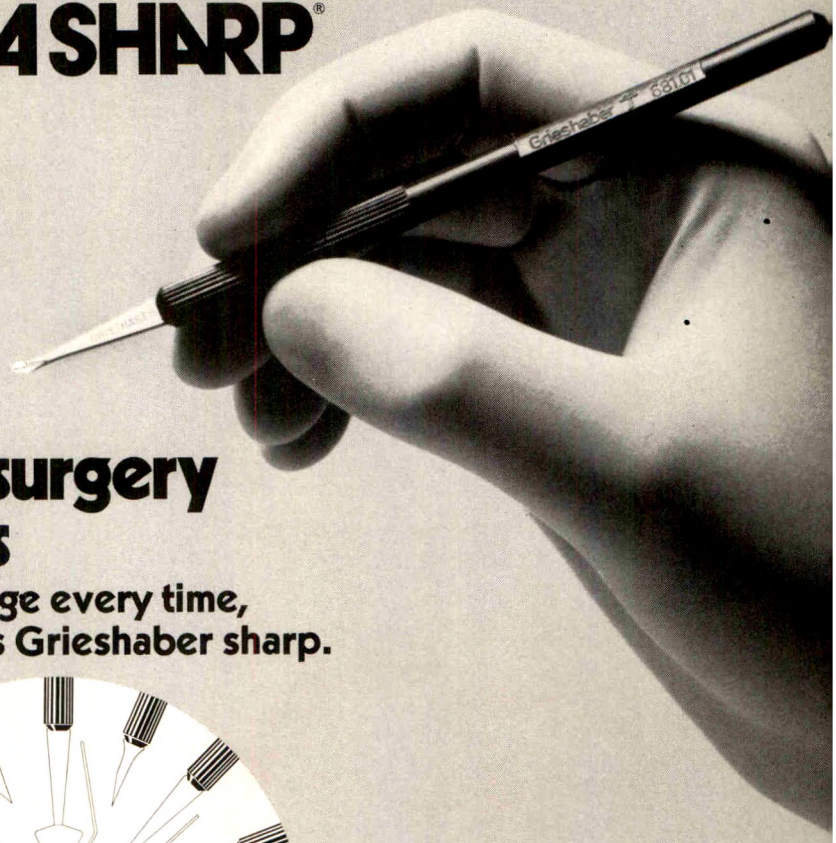
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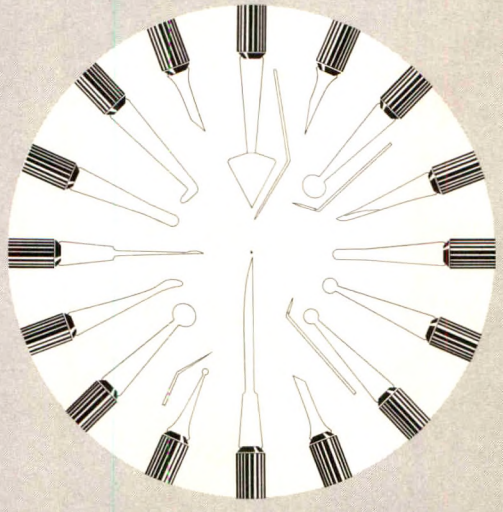
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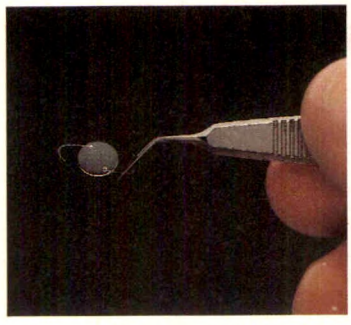
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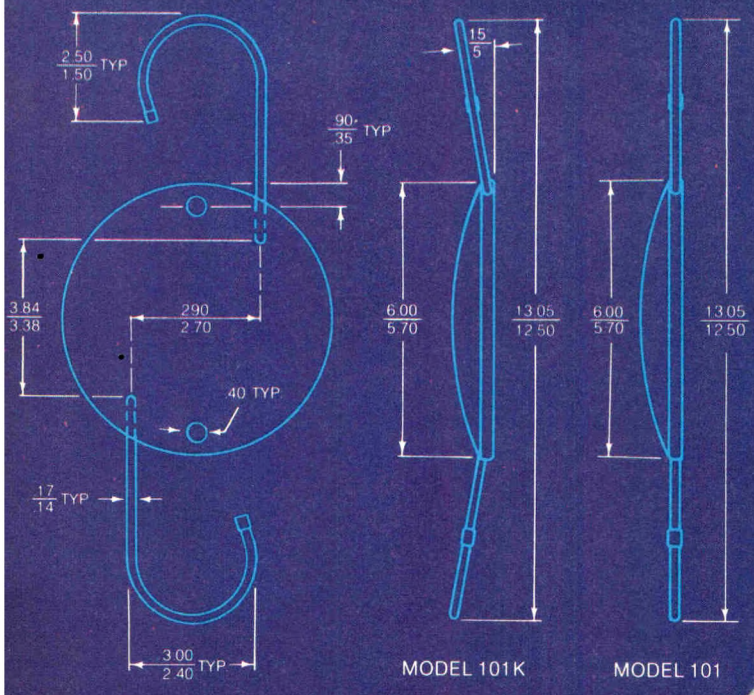


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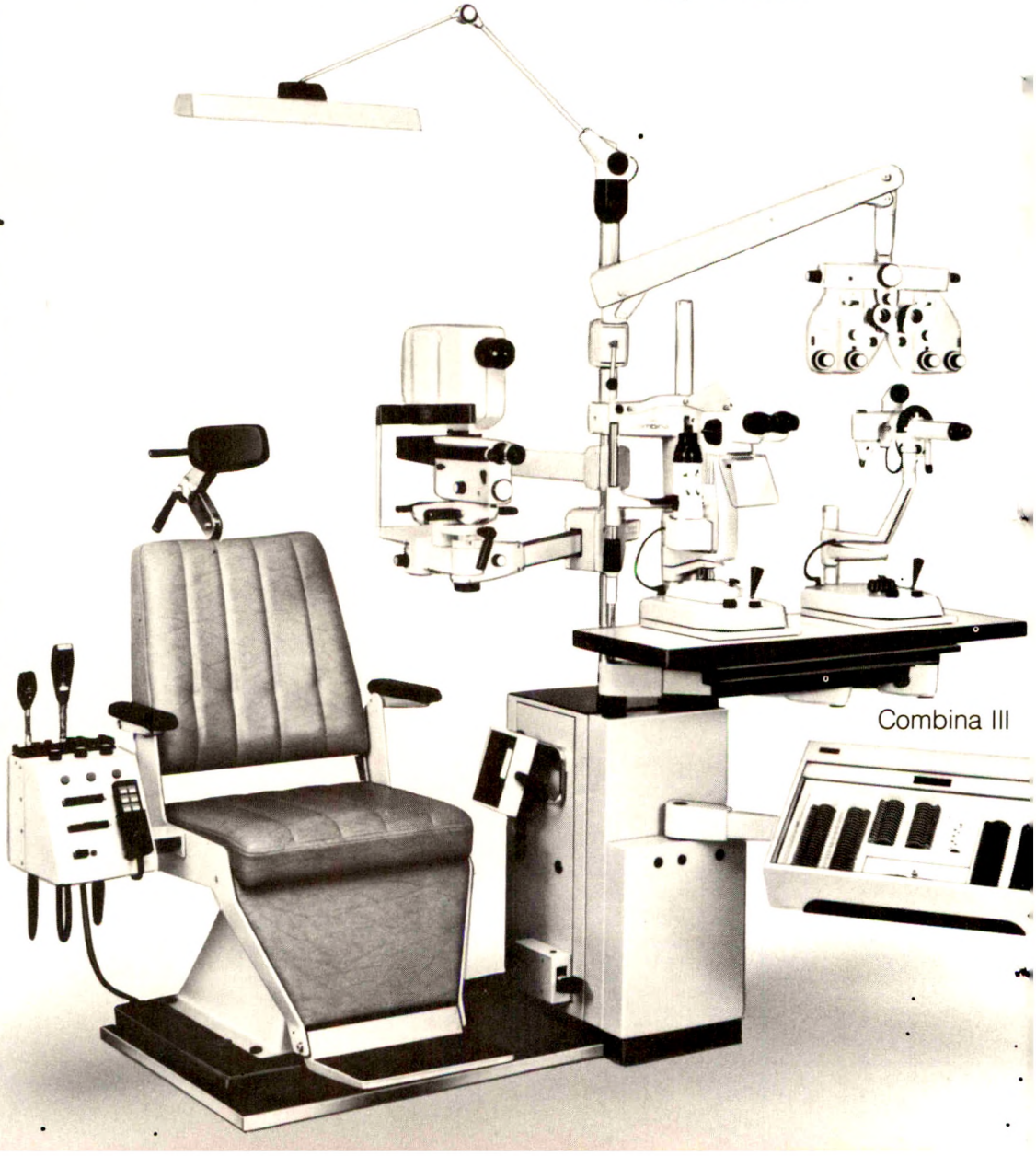
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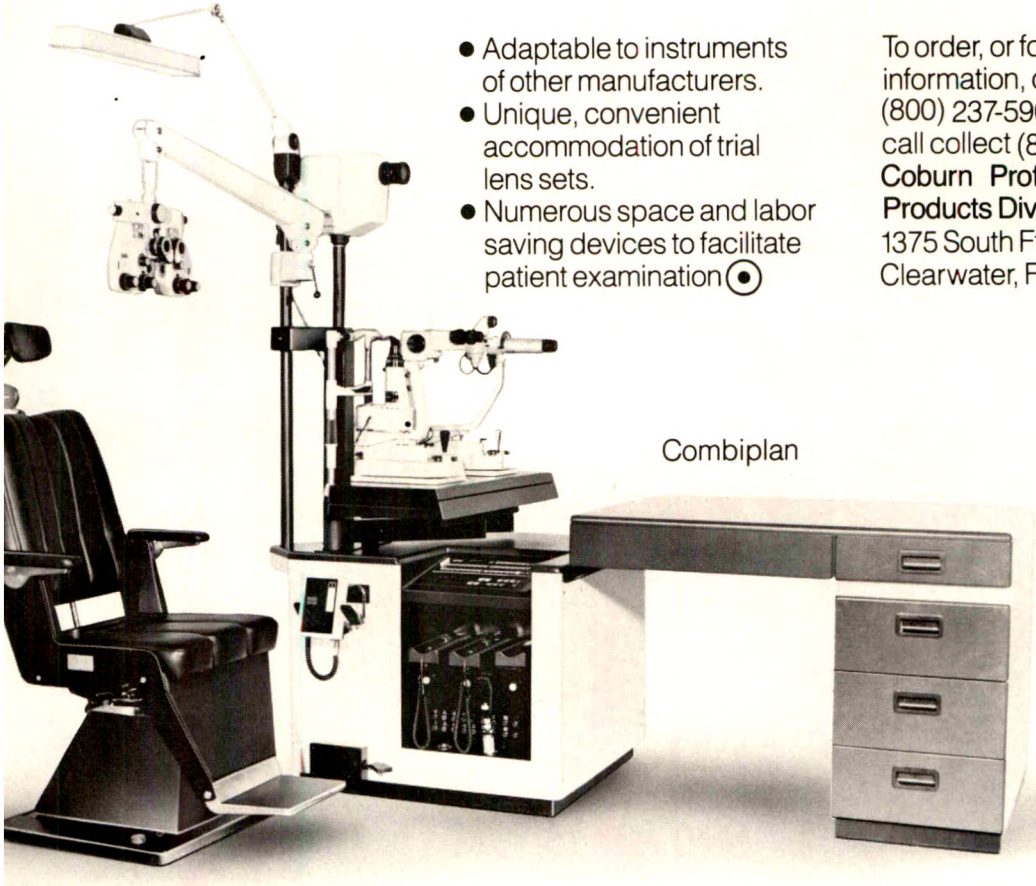
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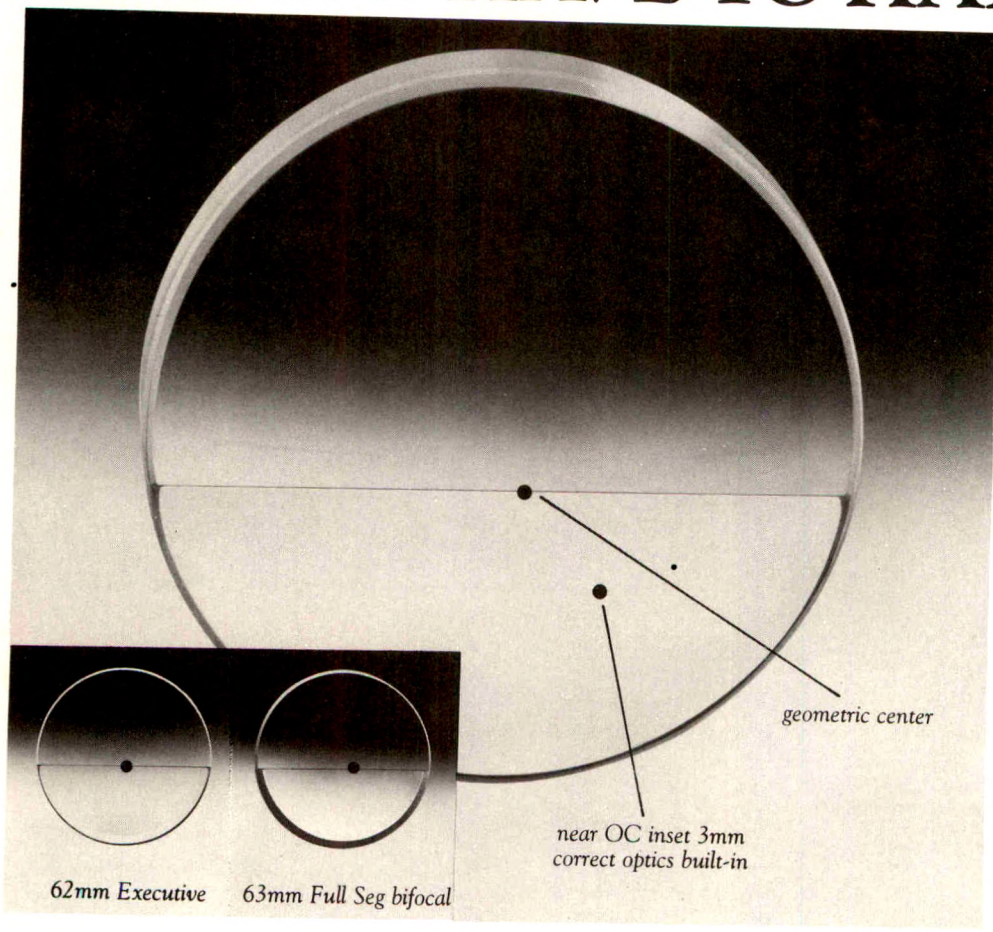


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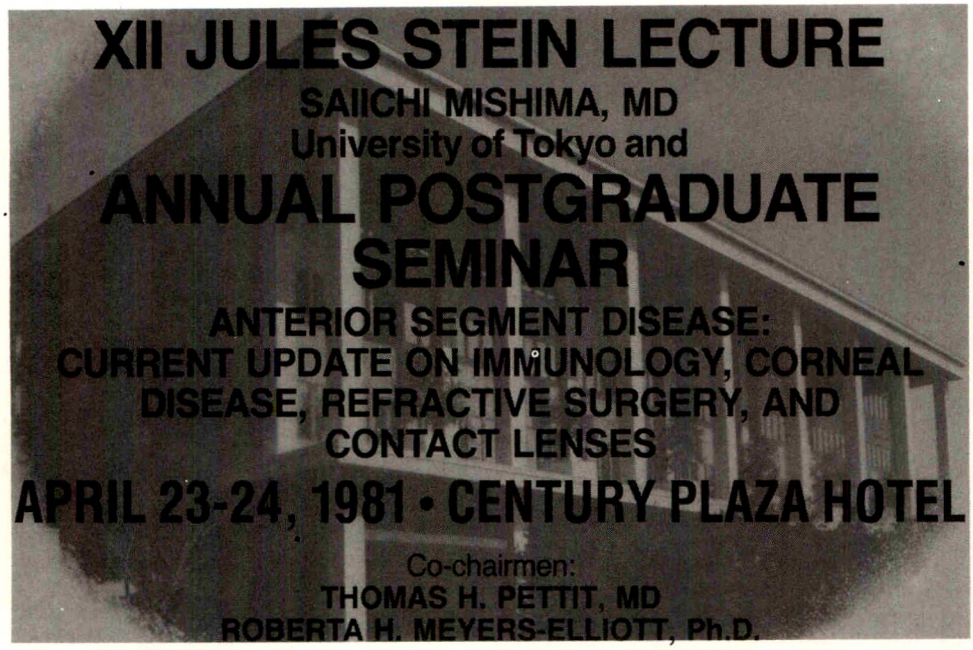
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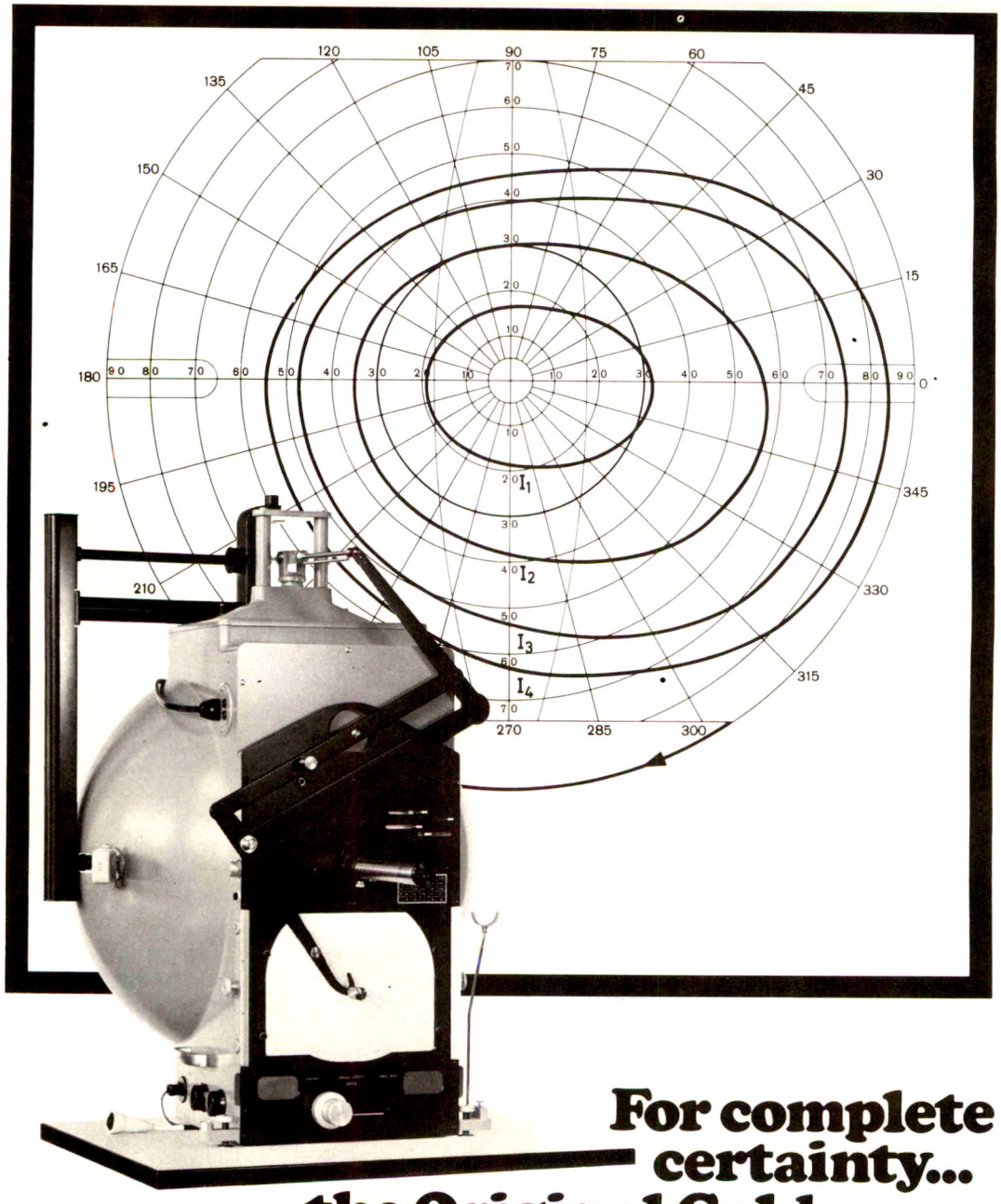
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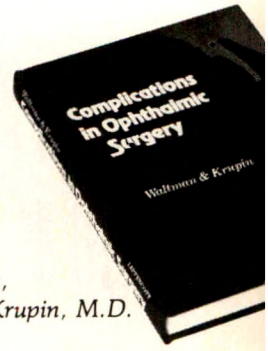
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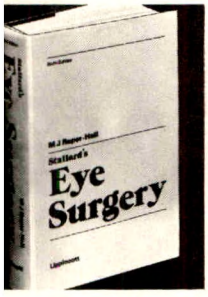
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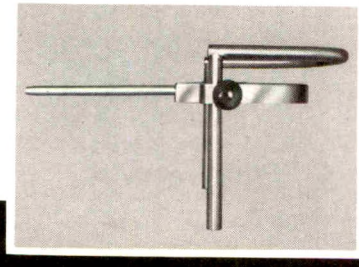
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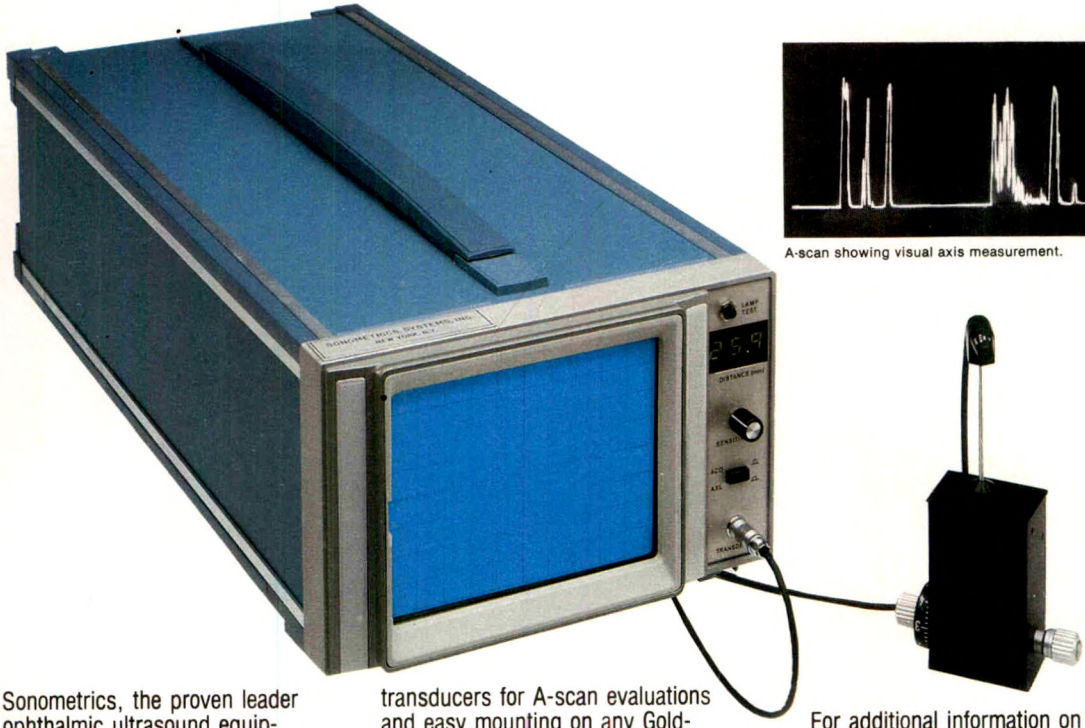
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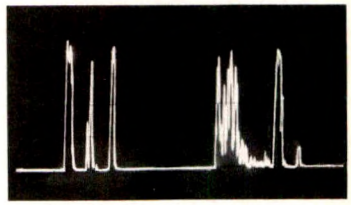
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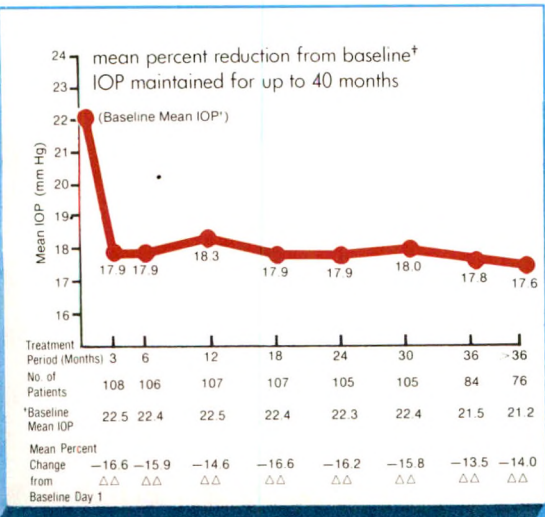
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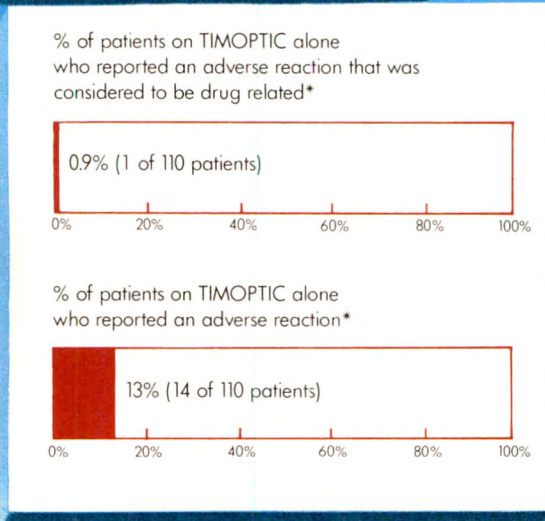


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In patients with a history of severe cardiac disease, pulse rates should be checked.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
WARNINGS: As with other topically applied ophthalmic drugs, this drug may be absorbed systemically.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchospastic disease; sinus bradycardia and greater than first-degree block; cardiogenic shock; cardiac failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients. However, in one long-term study in which 96 patients have been followed for at least 3 years, no significant difference in mean intraocular pressure has been observed after initial stabilization.

Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.
Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.
Animal Studies: No adverse ocular effects were observed in rabbits and dogs administered TIMOPTIC topically in studies lasting one and two years respectively.

Lifetime studies with timolol have been completed in rats at oral doses of 25, 100, and 300 mg/kg/day and in mice at oral doses of 5, 50, and 500 mg/kg/day. In male and female rats and male mice at all dose levels and in female mice at dose levels of 5 and 50 mg/kg/day, timolol demonstrated no carcinogenic effect. There was a slight increase in the incidence of mammary adenocarcinomas in female mice that received 500 mg/kg/day (about 500 times the maximum recommended human oral dose on a mg/kg basis). Timolol caused dose-related elevations of serum prolactin in female mice at doses of 100 mg/kg or more, but only very slight transient elevations were found in male mice at doses of 500 mg/kg. Since numerous studies have demonstrated that drugs which cause elevations of serum prolactin are associated with mammary tumors in rodents, the mammary tumors in the female mice in the highest dosage group of this study were considered to have resulted from an increased serum prolactin. In humans, no such association between serum prolactin and mammary carcinoma has been established. Furthermore, in adult human female subjects who received oral dosages of up to 60 mg of timolol, the maximum recommended human oral dosage, there were no clinically meaningful changes in serum prolactin.
ADVERSE REACTIONS: TIMOPTIC Ophthalmic Solution is usually well tolerated. The following adverse reactions have been reported either in clinical trials of up to 3 years duration prior to release in 1978 or since the drug has been marketed.

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Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

Aggravation or precipitation of certain cardiovascular and pulmonary disorders has been reported, presumably related to effects of systemic beta blockade (see PRECAUTIONS). These include bradyarrhythmia, hypotension, syncope, and bronchospasm (predominantly in patients with pre-existing bronchospastic disease). In clinical trials, slight reduction of the resting heart rate in some patients (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed.

The following adverse effects have been reported rarely, and a causal relationship to therapy with TIMOPTIC has not been established: headache, anorexia, dyspepsia, nausea, dizziness, CNS effects (fatigue, confusion, depression, somnolence, and anxiety), palpitation, and hypertension.
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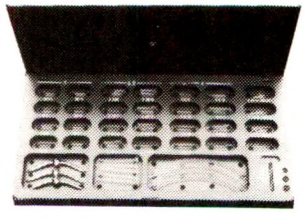
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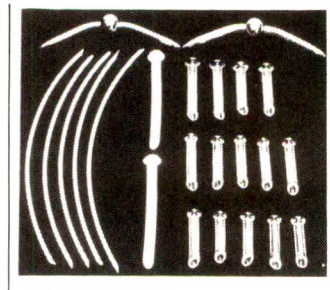
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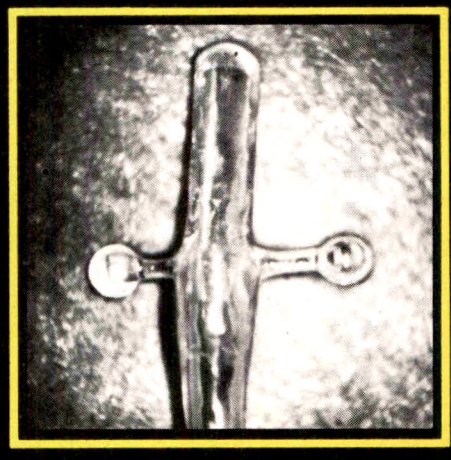
* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
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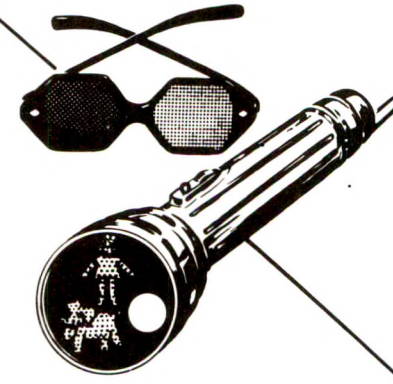
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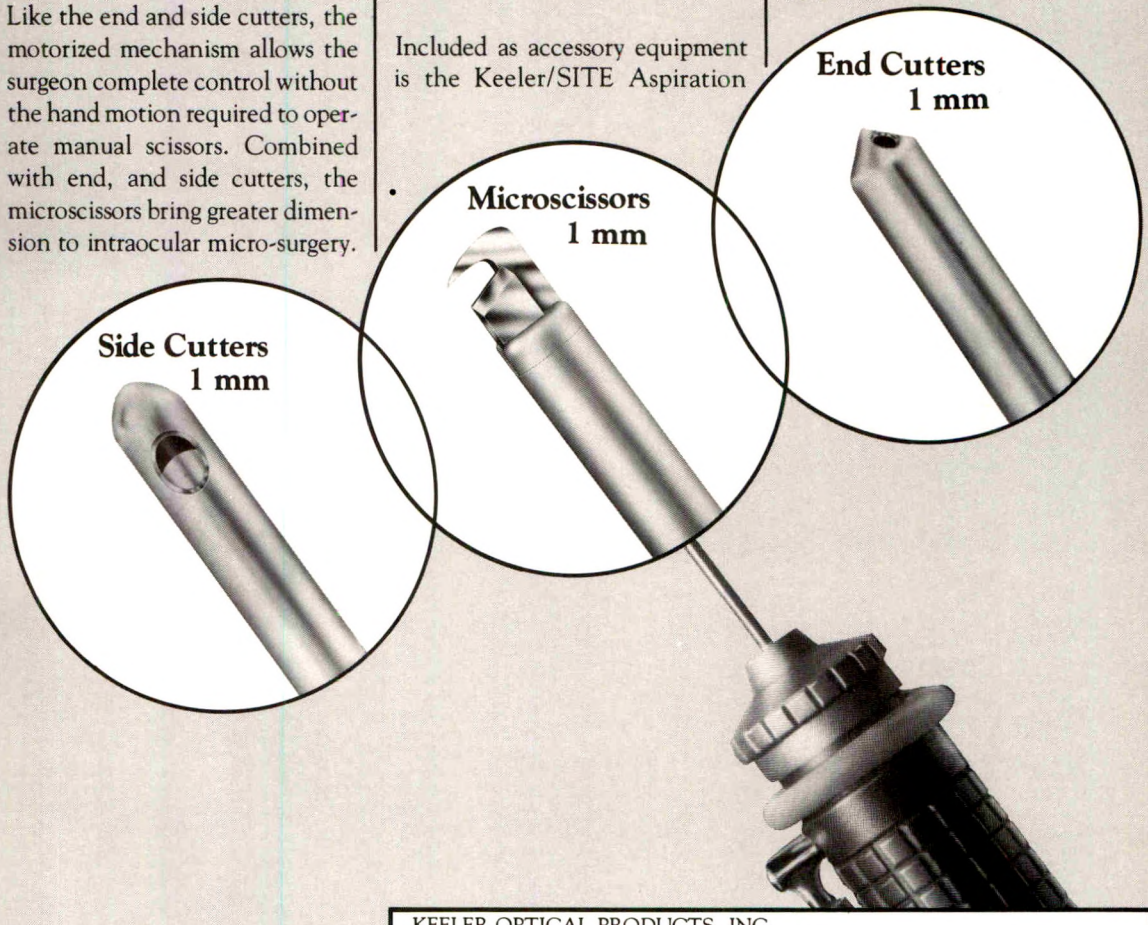
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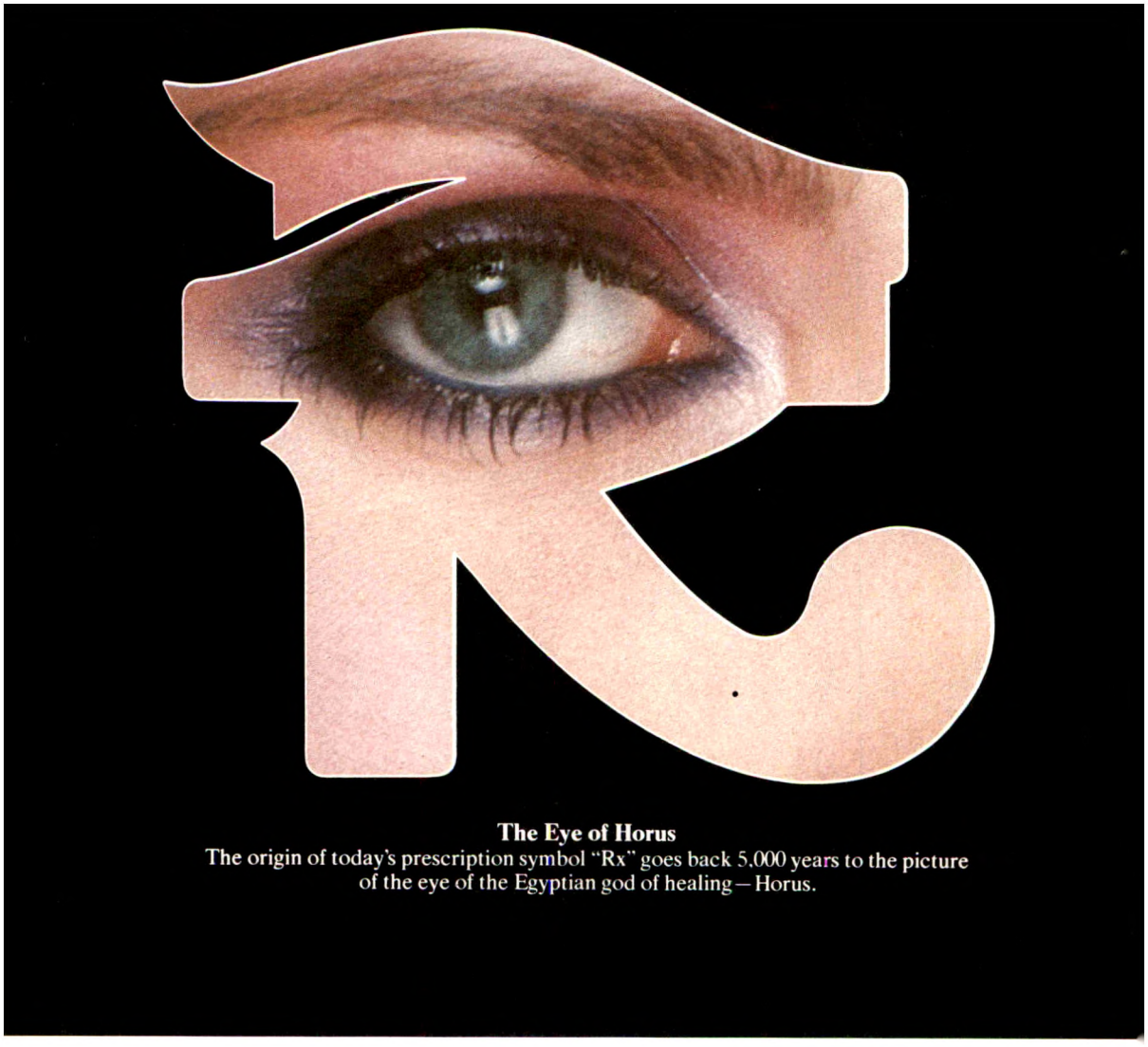
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VIROPTIC sterile opththalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

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During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

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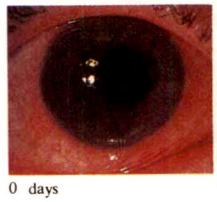
During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

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*Data on file, Medical Division, Burroughs Wellcome Co.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

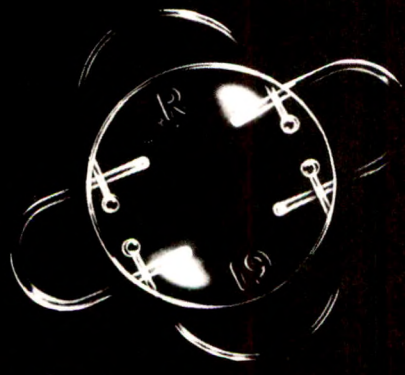
If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

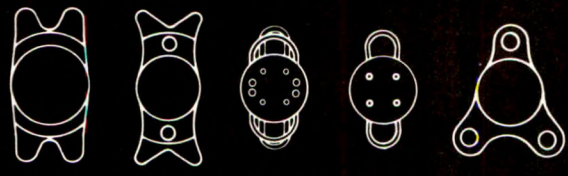
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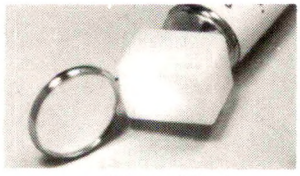
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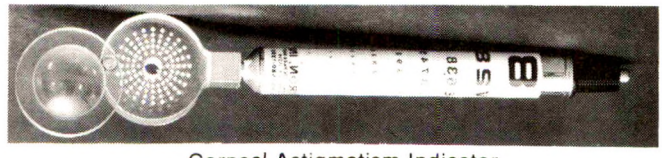
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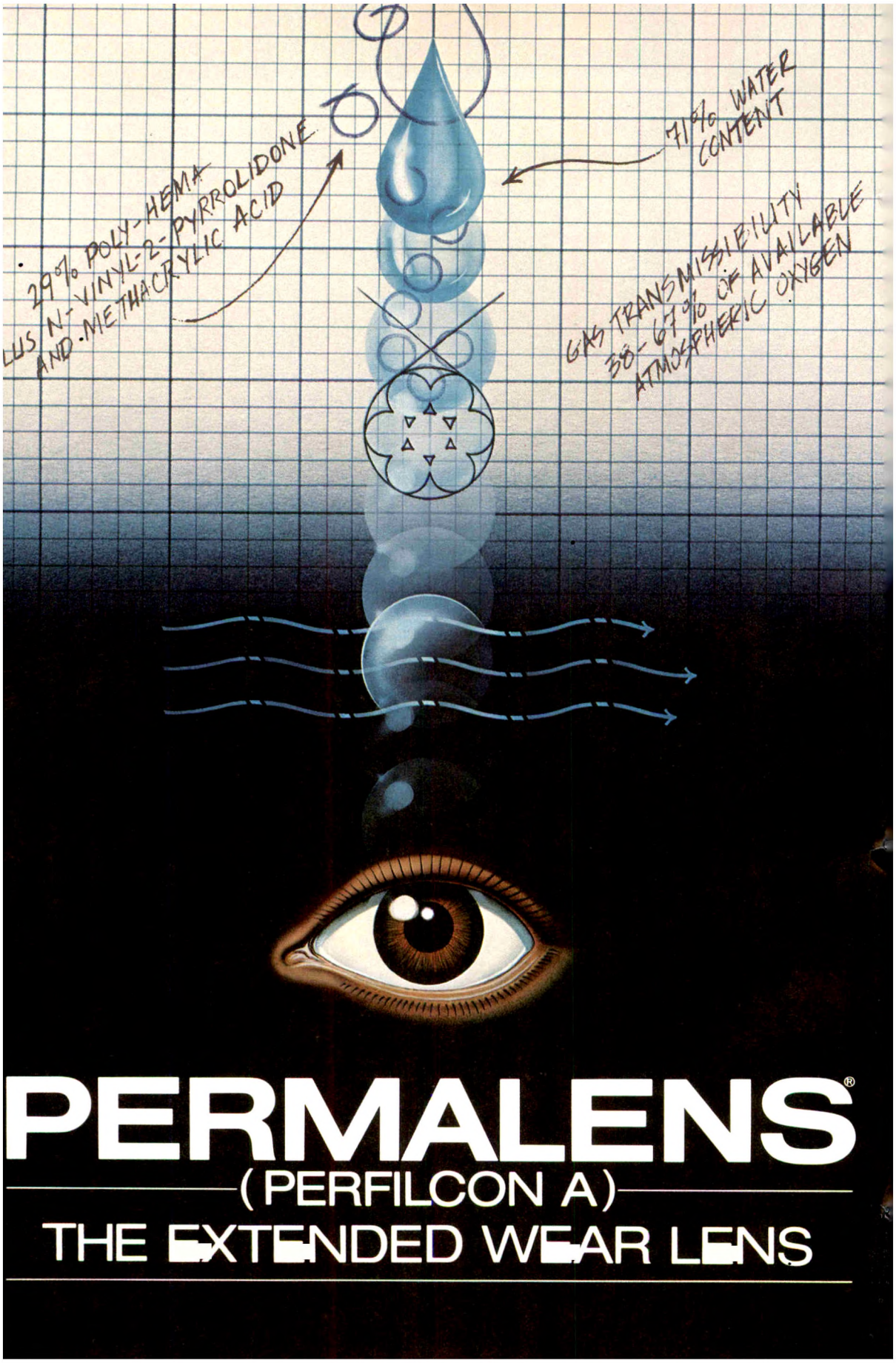
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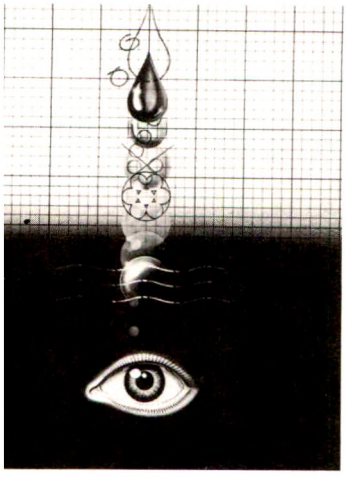
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the wearer's cornea, the lens should be removed and
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2. NORMOL® Sterile Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thimerosal (Lilly) 0.001%, edetate disodium 0.1% and chlorhexidine 0.005%).
3. CLERZ® Lubricating and Rewetting Eye Drops (sterile solution preserved with edetate disodium 0.1% and Thimerosal (Lilly) 0.001%).
4. ADAPETTES® Sterile Lubricating Solution (buffered isotonic aqueous solution containing Adsorbobase® (polyvinylpyrrolidone with other water-soluble polymers), Thimerosal (Lilly) 0.004%, and edetate disodium 0.1%).

No conventional (hard) contact lens solution should be used. Only those solutions specifically recommended by your eye-care practitioner should be used during wear.

WEARING RESTRICTIONS

Permalens® (perfilcon A) Hydrophilic Contact Lenses should not be worn while swimming or in the presence of noxious or irritating vapors.

Lens-Care Regimen
Patients must adhere to the recommended lens-care regimen of **Permalens®** Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections which might result in corneal ulcers.

PRECAUTIONS

Storage
Permalens® Hydrophilic Contact Lenses must be stored in the appropriate storage solution: PERMASOL™ Rinsing and Storage Solution, BOILnSOAK® Sterile Saline Solution, or FLEXSOL® Sterile Disinfection and Storage Solution, depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in one of the following until it returns to a hydrated (soft) pliable state:

1. PERMASOL™ Rinsing and Storage Solution
2. BOILnSOAK® Sterile Saline Solution
3. FLEXSOL® Sterile Disinfection and Storage Solution

PERMA-case™ Contact Lens Storage and Carrying Case should be used for storing lenses.

Hygiene
Hands must be washed and rinsed thoroughly, and dried before handling the lenses. Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Cleaning and Disinfecting
Prior to wearing, **Permalens®** (perfilcon A) Hydrophilic Contact Lenses must be both cleaned and disinfected.

Cleaning: Monthly removal for cleaning is recommended. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Sterile Cleaning Solution. Between regular cleaning periods, CLERZ® Lubricating and Rewetting Eye Drops or ADAPETTES® Sterile Lubricating Solution may be used to maintain lens hydration while being worn.

Routine Thermal Disinfection Method: Permalens® Hydrophilic Contact Lenses may be effectively disinfected using a low-heat disinfecting unit approved for use with hydrophilic contact lenses. After cleaning with PREFLEX® Sterile Cleaning Solution, rinse lenses with PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution, and disinfect in a RINCON* low-heat Thermal Disinfection Unit or other approved unit. The lens storage containers must be emptied and filled with fresh PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfecting system can be used temporarily. After cleaning the lenses according to instructions, fill each chamber of the PERMA-case™ with PERMASOL™ or BOILnSOAK® Bring a pan of water to a rolling boil. Remove the pan from heat. Place the sealed carrying case into hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: USE OF THE RINCON® THERMAL DISINFECTION UNIT SHOULD BE RESUMED AS SOON AS POSSIBLE.

Chemical Disinfection Method: Disinfection with PREFLEX® Sterile Cleaning Solution, FLEXSOL® Sterile Disinfection and Storage Solution and NORMOL® Sterile Rinsing Solution has been shown to be an effective disinfection system. Following each removal, **Permalens®** Hydrophilic Contact Lenses must be cleaned with PREFLEX® Sterile Cleaning Solution and rinsed with NORMOL® Sterile Rinsing Solution. Each time the lens is stored, the PERMA-case™ Contact Lens Storage and Carrying Case must be emptied and refilled with fresh FLEXSOL® Sterile Disinfection and Storage Solution. Fresh FLEXSOL® Sterile Disinfection and Storage Solution must also be used when disinfecting a lens.

WARNING: FLEXSOL® STERILE DISINFECTION AND STORAGE SOLUTION SHOULD *NEVER* BE USED WITH HEAT.

LENS EJECTION

Occasionally, a lens may become partially dehydrated during sleep. This may result in the lens being dislodged from the eye by lid action or rubbing the eyelids. Dislodged or routinely removed lenses should be thoroughly rehydrated in one of the following solutions:

1. PERMASOL™ Rinsing and Storage Solution
2. NORMOL® Sterile Rinsing Solution
3. BOILnSOAK® Sterile Saline Solution

After the lens returns to a soft, supple state it should be cleaned and disinfected prior to replacement on the eye.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses which have been soaked in conventional hard contact lens solutions. Eye irritation may occur within a short time after putting on a lens stored in a solution of improper pH or tonicity. Removal of the lens will relieve the irritation.

Excessive tearing or redness, unusual eye secretions, reduced visual acuity, and photophobia are not normal; if these symptoms occur, the patient should be examined by an eye-care practitioner to determine the cause.

DOSAGE AND ADMINISTRATION

Fitting
Conventional methods of fitting contact lenses do not apply to **Permalens®** (perfilcon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the **Permalens®** Hydrophilic Contact Lens Professional Fitting Guide, copies of which are available from Cooper Laboratories, Inc., 265 N. Whisman Road, Mountain View, California 94043.

WEARING SCHEDULE

Permalens® Hydrophilic Contact Lenses are indicated for extended-wear (24-hour) use, without a gradual daily increase in wearing time required. Cleaning is recommended at monthly intervals. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient.

Lens Care and Handling
Care must be taken on the initial visit to ensure that the patient is supplied with all necessary accessory products and fully understands all care and handling instructions for the lenses. Regular post-fitting visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expiration date of the lens.

Accessories

Patient Care Kit	Cat. #802303
Wearer's Manual	Cat. #802445
PERMA-case™ Contact Lens Storage and Carrying Case	Cat. #802310
FLEXSOL® Sterile Disinfection and Storage Solution*	Cat. #901105
NORMOL® Sterile Rinsing Solution*	Cat. #901275
PREFLEX® Sterile Cleaning Solution*	Cat. #901310
CLERZ® Lubricating and Rewetting Eye Drops	Cat. #801045
PERMASOL™ Rinsing and Storage Solution	Cat. #801310
RINCON® Thermal Disinfection Unit**	Cat. #902385
BOILnSOAK® Sterile Saline Solution*	Cat. #901035
ADAPETTES® Sterile Lubricating Solution	Cat. #901003

CAUTION: Federal Law Prohibits Dispensing Without Prescription

[1]Fatt I, Morris JA: A survey of gas-permeable contact lenses. *The Optician*: 27-36 (November 4, 1977).

*A product of Burton, Parsons & Company, Inc.
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EXTRACAPSULAR CATARACT EXTRACTION

A PRACTICAL WORKSHOP

(INCLUDING POSTERIOR CHAMBER IOL'S)

FRIDAY AND SATURDAY, March 20-21, 1981

(Limited to 40 Participants)



ESTELLE DOHENY EYE FOUNDATION

OBJECTIVES:

1. Review indication, contraindications, complications, pre-op management and surgical techniques, etc. for extracapsular cataract extraction and posterior chamber lenses.
2. Supervised practice surgery on cadaver and animal eyes using various extracapsular techniques and posterior chamber IOL's.
3. Review video tapes of human surgery.

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PROGRAM

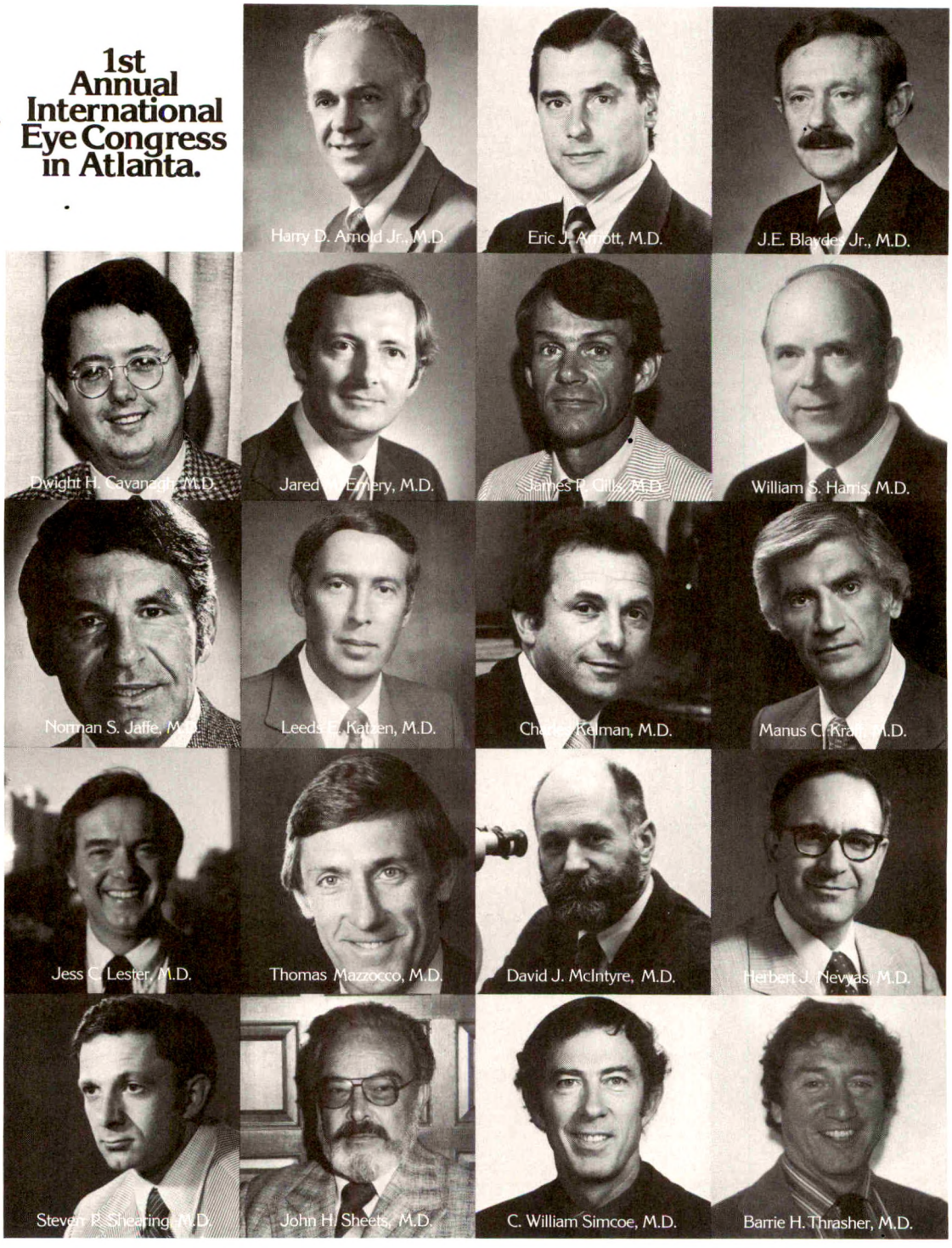
DIDACTIC SESSIONS

History of ECCE Techniques
Extracapsular Techniques
Use of I and A Tip in Planned ECCE
Management of the Posterior Capsule
Problems Related to Late Capsule Clouding
Indications and Selection of Patients
Contraindications and Management
Posterior Chamber IOL's
When to Abort an IOL
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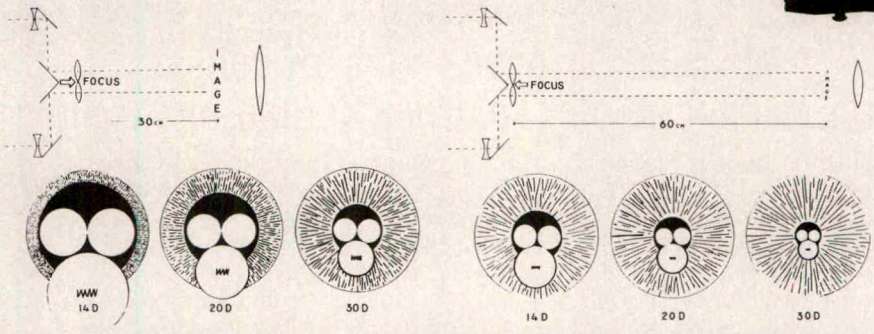
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TOPICS

- REFRACTIVE SURGERY (Friday, March 13)
  Radial Keratotomy (Theory, Animal Studies, Technique, Human Results, Proposed Collaborative Study), Keratophakia, Keratomileusis, Epikeratophakia, Intraocular Lenses
- PEDIATRIC ANTERIOR SEGMENT SURGERY (Saturday, March 14)
  New Approaches to Congenital Cataracts, Subluxated Lenses, Secondary Membranes, Anterior Vitrectomy, Management of Pediatric Ocular Trauma
- UPDATE ON RETROLENTAL FIBROPLASIA (Sunday, March 15)
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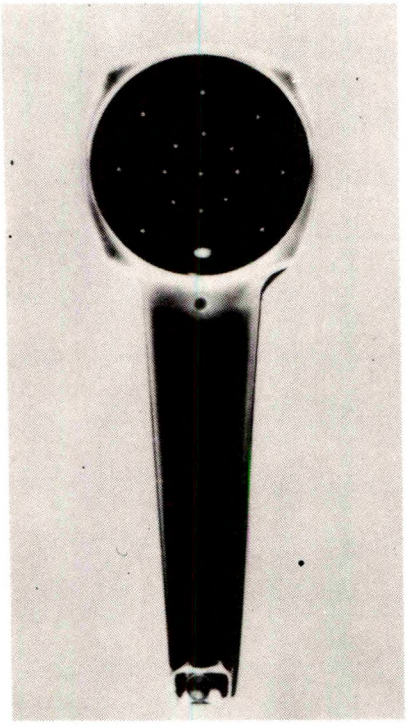
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AMA Category I

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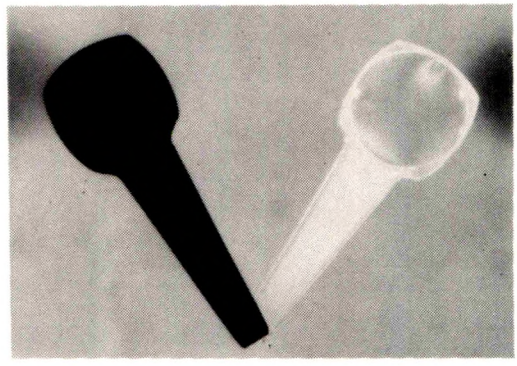
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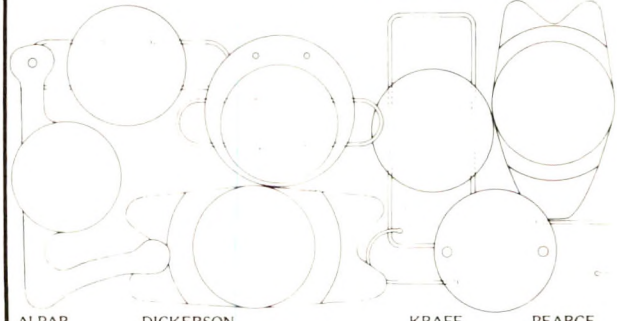
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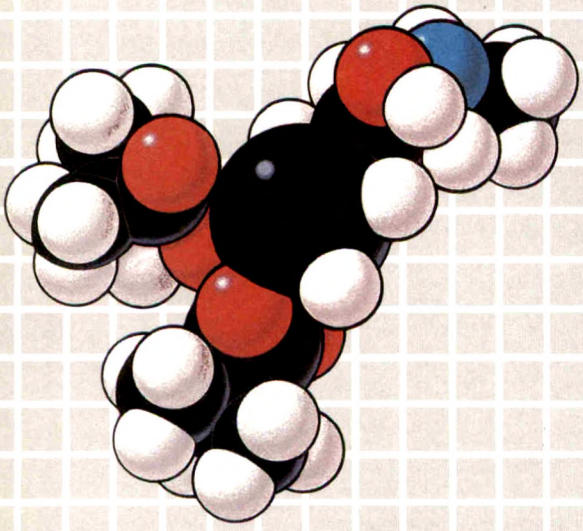
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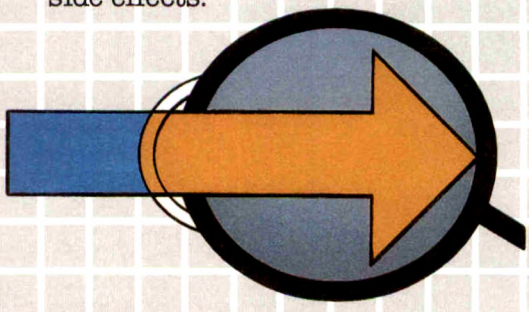
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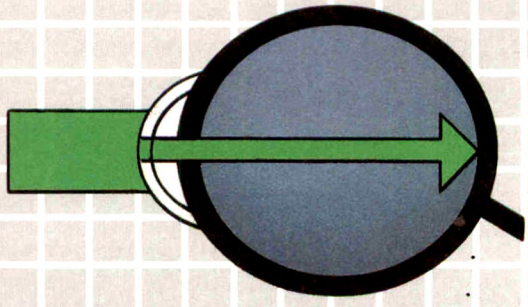
This addition creates a more lipophilic compound which is absorbed better into the eye — 17 times better penetration.[1] Once inside, it is converted to epinephrine by enzymes within the eye. This action makes Propine more potent than comparable concentrations of epinephrine, yet with fewer side effects.



Propine (dipivefrin HCl) molecule depicting addition of the two pivalyl side chains.



Propine (dipivefrin HCl): better penetration with minimal side effects.



Epinephrine: less efficient penetration with greater chance of side effects.

The first prodrug for glaucoma therapy.

Concomitantly with timolol or pilocarpine, or as initial therapy, Propine™ (dipivefrin HCl) provides:

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Therapeutic response comparable to 2% pilocarpine q.i.d.
Reduces mean pressure 20 to 24%
Facilitates outflow and decreases aqueous production

Fewer side effects than epinephrine or pilocarpine

Fewer external side effects than epinephrine.[2,3]
Less chance of cardiovascular effects than epinephrine.[4]
Propine is well-tolerated by patients with demonstrated intolerance to epinephrine.[5]
No miosis as with pilocarpine.

Convenient b.i.d. dosage

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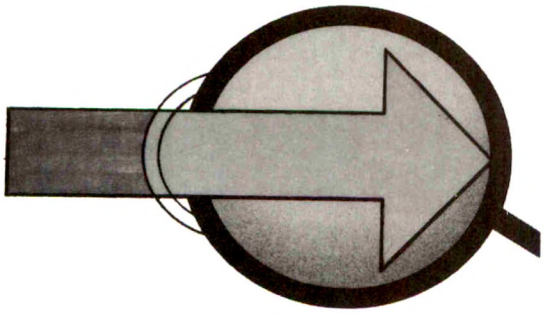
Propine (dipivefrin HCl): the right direction for use

- Concomitantly with timolol or pilocarpine
- Switch epinephrine and pilocarpine users to Propine
- Initial therapy: effective and safe choice for the new glaucoma patient.

Propine™ (dipivefrin HCl)... another advancement in research and development by the leader in ophthalmics.

References:
1. Mandell AI. et al. Dipivalyl epinephrine: A new prodrug in the treatment of glaucoma. Ophthalmology 1978 Mar;85(3):268-275.
2. Kohn AN. et al.Clinical comparison of dipivalyl epinephrine and epinephrine in the treatment of glaucoma. Am J Ophthalmol 1979;87:196-201.
3. Kass MA. et al. Dipivefrin and epinephrine treatment of elevated intraocular pressure: a comparative study. Arch Ophthalmol 1979 Oct;97(10):1865-1866.
4. Allergan Report Series No. 168, 1980.
5. Allergan Report Series No. 169, 1980.

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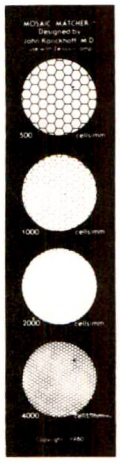
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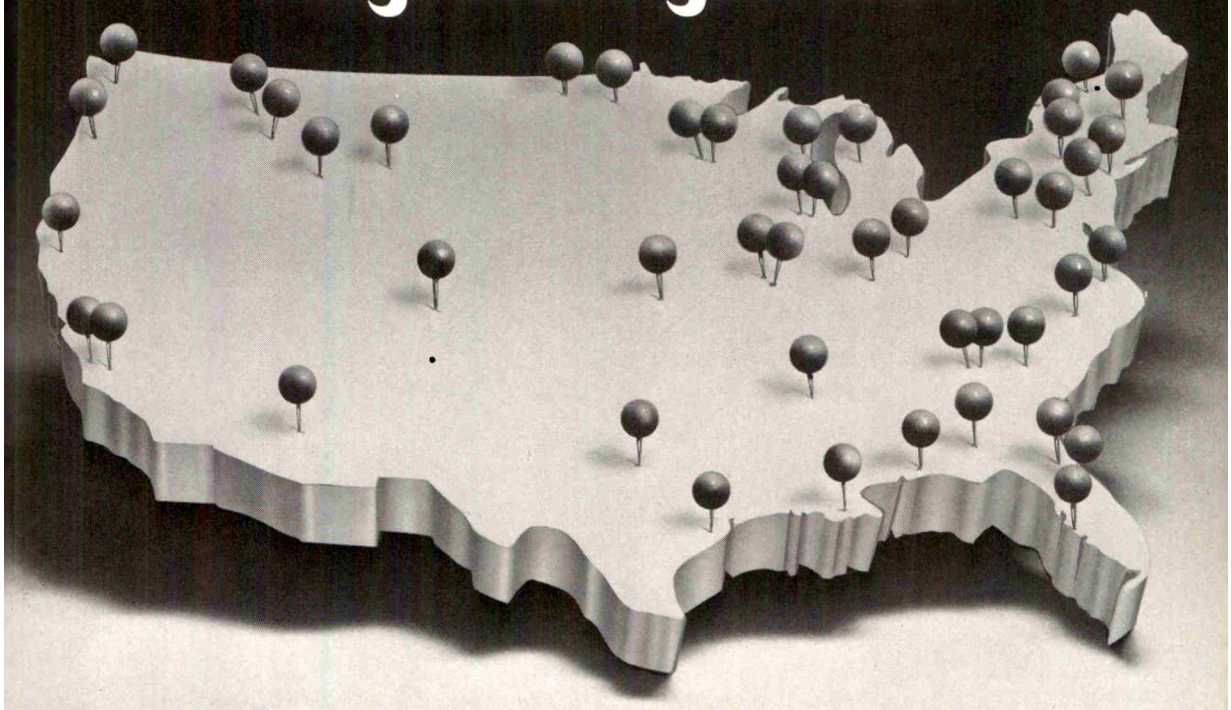
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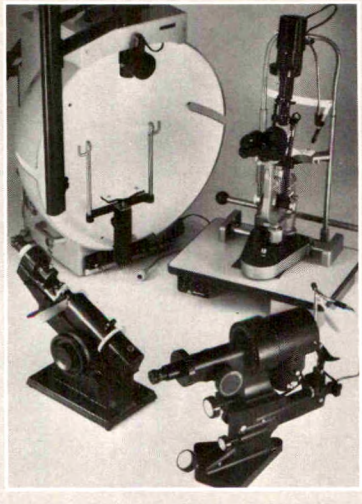


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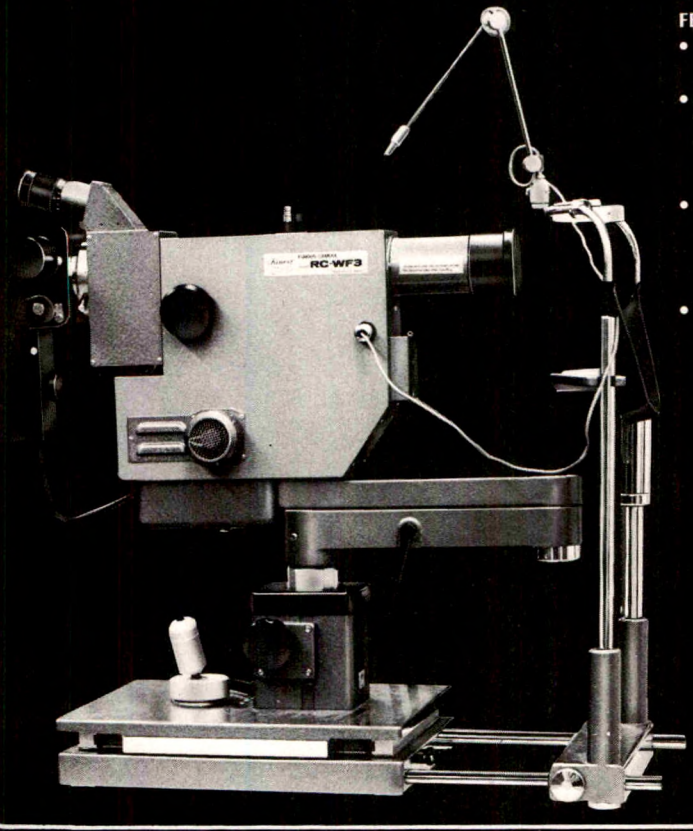
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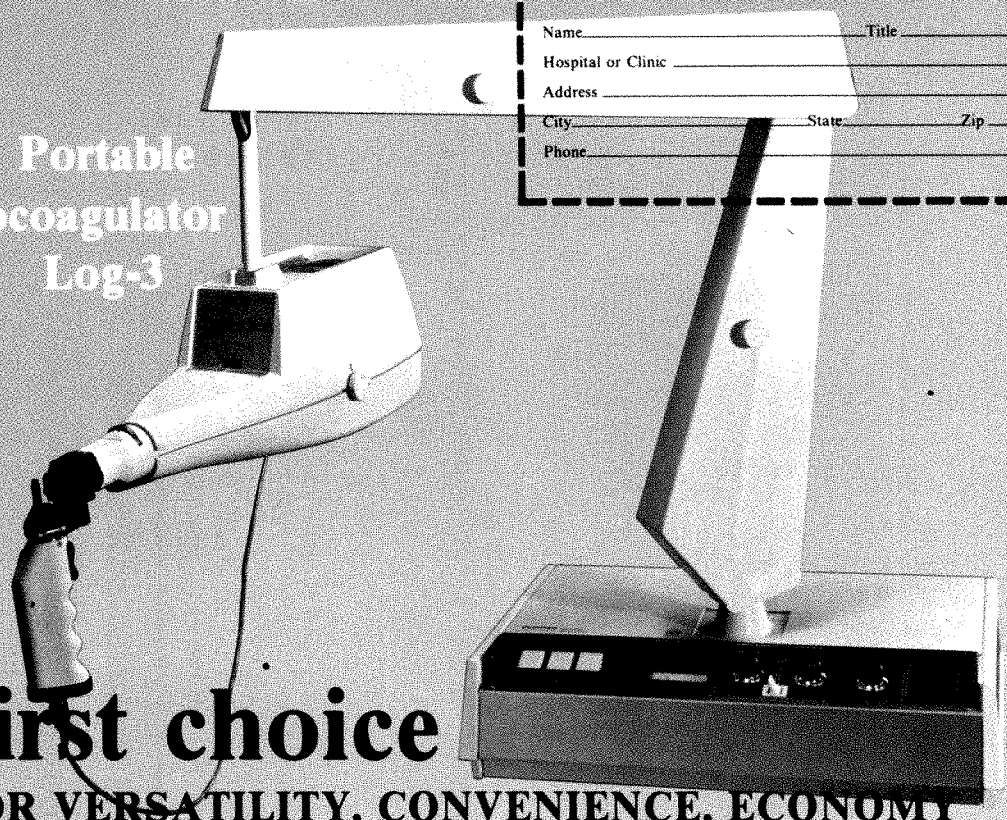
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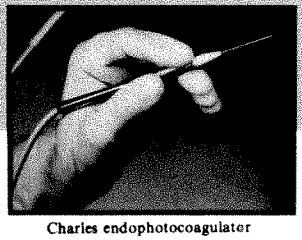
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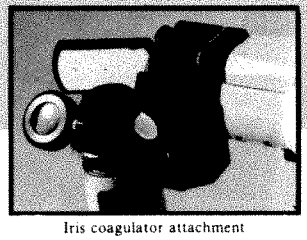


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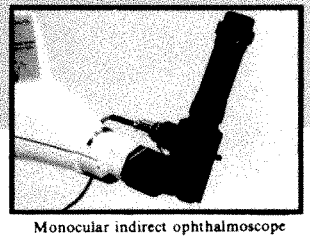
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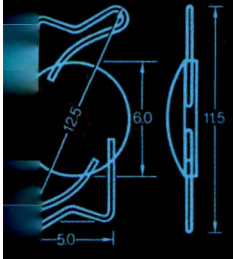
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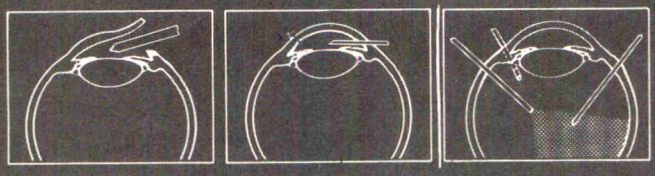
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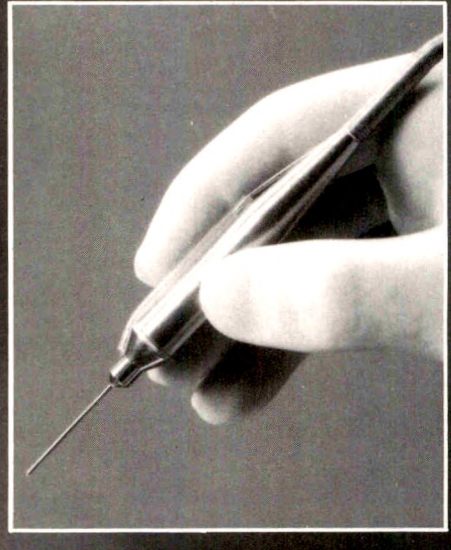
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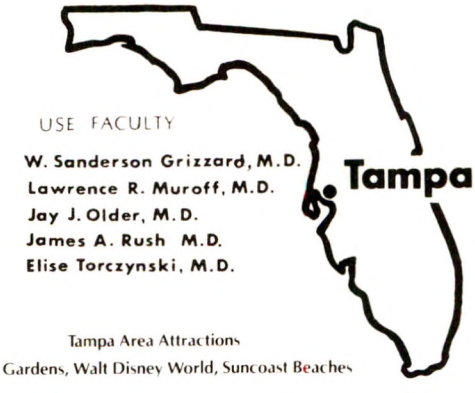
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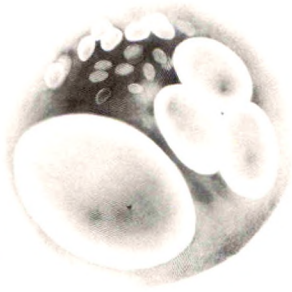


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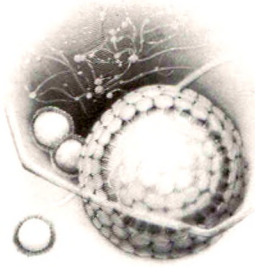
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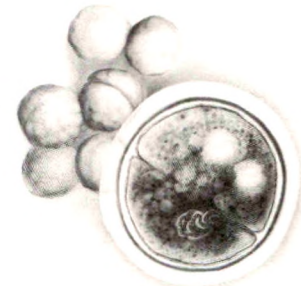
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REFERENCE: [1]Antibacterial Agents. Monograph I. *Office Seminars in Ophthalmology.*® Irving H. Leopold, ed. Allergan Pharmaceuticals, 1976, p. 5.

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CONGENITAL ADDUCTION PALSY AND SYNERGISTIC DIVERGENCE

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There have been four reported cases of congenital unilateral paralysis of adduction associated with simultaneous bilateral abduction on attempted gaze into the field of action of the paretic medial rectus muscle.[1-4] The apparent medial rectus muscle palsy has been attributed to internuclear ophthalmoplegia or selective destruction in the medial rectus muscle nucleus. The peculiar abduction movement, the most striking feature of this syndrome, has been attributed to anomalous connections between the abducens (cranial nerve VI) nuclei[1,2] or to unopposed action of the lateral rectus muscle, allowing further contraction of the oblique muscles and causing the globe to become more abducted.[3] We report a new case of congenital adduction palsy and simultaneous divergence and offer an analysis of the clinical findings and electromyographic data which suggest a relationship to Duane's retraction syndrome.

CASE REPORT

A 7-year-old boy was examined here in 1971 because of a variable exotropia present since his birth. The patient had had strabismus surgery in 1968, at which time preoperative forced ductions while the boy was anesthetized disclosed increased resistance to adduction but not to abduction. At that time, the right lateral rectus muscle insertion was described as fibrous and elongated with an inelastic fibrous band attached to the sclera. The lateral rectus muscle was disinserted, the fibrous band excised, and the lateral rectus muscle recessed to the equator. The medial rectus muscle, described as "tight," was resected 6 mm.

We did not examine him again until 1978. Visual acuity was R.E.: 6/9 (20/30) with $-1.00\ -3.00 \times 15$ and L.E.: 6/7.5 (20/25) with $-4.75\ -1.00 \times 160$. Intraocular pressure in the right eye, 15 mm Hg in the primary position, increased to 25 mm Hg as the patient attempted levoversion, producing abduction of the right eye. The intraocular pressure was 15 mm Hg in the left eye in both positions. Results of other examinations, including an otolaryngologic examination, audiometry, and X-ray films of the cervical spine and skull, were normal.

In the primary position (Fig. 1), there was a 30-diopter exotropia of the right eye. On attempted levoversion both eyes abducted; the maximum divergence of the eyes exceeded 90 diopters. The right eye abducted as soon as the left eye abducted past the midline. There was slight narrowing of the right palpebral fissure with some depression of the right eye on attempted levoversion. On dextroversion, the left eye adducted normally, with the right eye abducting less than with levoversion. There was slight underaction of the elevator and depressor

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Fig. 1 (Wilcox, Gittinger, and Breinin). Extraocular movements in nine fields of gaze. The right eye has the most marked lateral movement on attempted levoversion, but also abducts on dextroversion to a lesser degree.

muscles in the right eye. The right eye's maximally adducted position was 15 degrees temporal to the midline. The pupils did not react to changes in the position of the eyes. The globe did not retract. Initial and subsequent forced ductions, done while the patient was anesthetized with topically applied proparacaine, still showed increased resistance to adduction.

The left eye converged normally. The right eye remained stationary in its exotropic position until the target was 3 to 4 cm from the nose, at which point it abducted further. When we tested asymmetric convergence by bringing a target in along the visual axis of the left eye, we elicited nystagmoid movements in that eye with an apparent fast phase to the left. At the same time, a nystagmus with a fast phase to the right appeared in the right eye.

Optokinetic nystagmus was unusual in that, as the drum was rotated to the patient's right, the eyes followed, but at the point where a refixation saccade should have taken place, the right eye abruptly jerked further into abduction. The left eye made the expected refixation saccade to the left. Rotating the drum to the patient's left produced simultaneous divergence of both eyes. The refixation saccade in the left eye appeared normal, and the right eye made only small movements.

Electromyographic examination—Electromyography of the ocular muscles, performed while the patient was anesthetized with topically applied proparacaine (Fig. 2), showed cocontraction of the right medial rectus and the right lateral rectus muscles. Rotating the optokinetic drum to the patient's right produced simultaneous maximal innervation of both lateral recti muscles, the electromyographic equivalent of synergistic divergence.

The electromyographic findings in the present case showed decreased amplitudes for both the medial and lateral recti muscles of the right eye. The alterations in amplitude could have been the result of the structural or innervational abnormality, the effect of previous surgery, or an artifact of sampling within the limits of the clinical situation. The innervational pattern is paradoxical, showing increased firing of the right lateral rectus muscle on left gaze. Calling this condition medial rectus muscle palsy seems technically inappropriate because electromyography demonstrated both innervation of the right medial rectus muscle and maintenance of a normal pattern of reciprocal innervation with respect to gaze (Fig. 2). Limitation of adduction of the right eye apparently resulted from anomalous and greater innervation of the antagonistic right lateral rectus muscle. The resulting simultaneous contraction of both lateral recti muscles on left gaze produced the divergence movement (Fig. 2). The increase in intraocular pressure in the right eye found on levoversion may have been the result of this cocontraction of the lateral and medial recti muscles or of the mechanical restriction observed. Lack of globe retraction, as observed here, has occurred in other cases of cocontraction.[5,6]

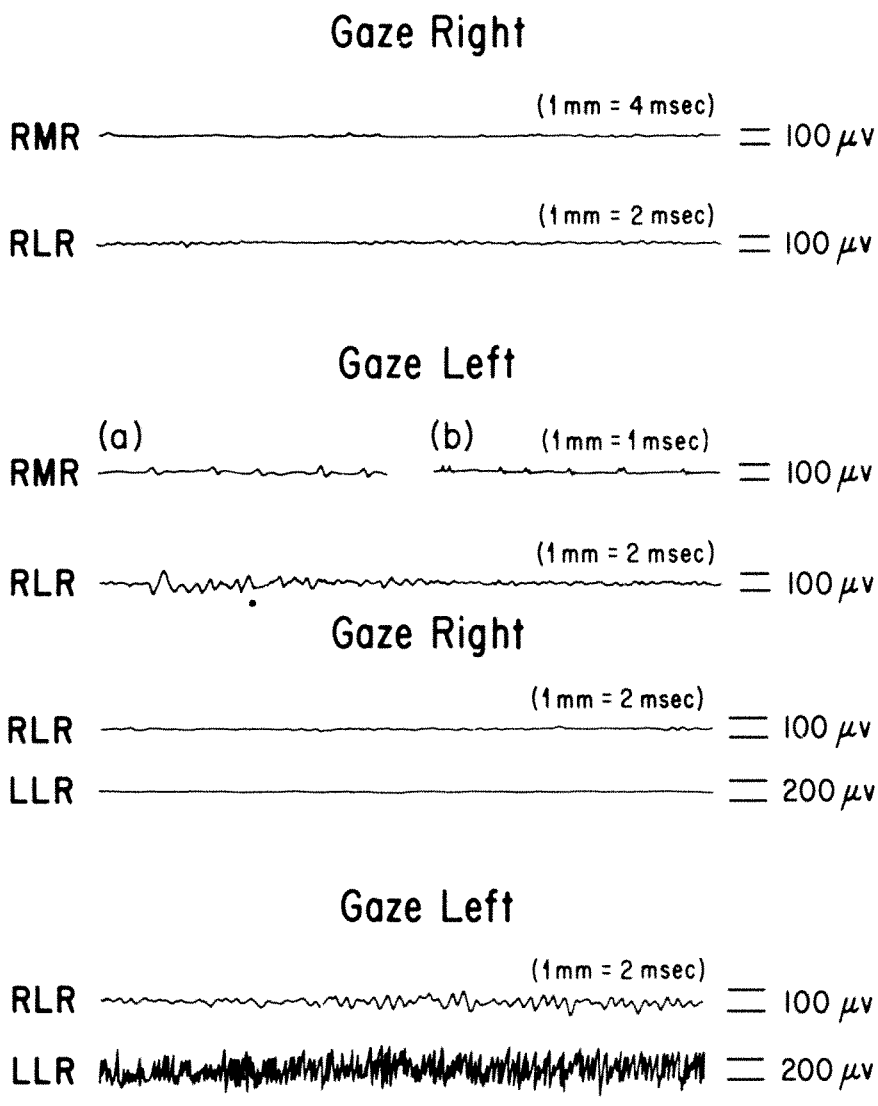


Fig. 2 (Wilcox, Gittinger, and Breinin). Ocular electromyogram. Top, On right gaze the right medial rectus muscle (RMR) and the right lateral rectus muscle (RLR) show reciprocal but reduced amplitudes. On left gaze there is augmentation of right medial rectus muscle with single units (a) and polyphasic spikes (b). Comparing the simultaneous innervation of the right medial and lateral recti muscles on left gaze demonstrates the cocontraction nature in the right eye. Bottom, Simultaneous recordings from both lateral recti muscles show marked coinnervation with left gaze.

Discussion

The clinical findings in this patient were similar to those in previously described patients, although, to our knowledge, this was the first case where the apparent medial rectus muscle palsy involved the right eye. Comparing clinical and electromyographic findings in simultaneous divergence to those seen in Duane's syndrome suggests that synergistic divergence should be considered a variant of Duane's retraction syndrome.

Cocontraction, an innvervational anomaly violating Sherrington's law, is a regular feature of Duane's syndrome[7-11] and has also been described in aberrant regeneration of the third cranial nerve, dorsal midbrain syndromes (such as Parinaud's and Koerber-Salus-Elschnig syndromes), and, possibly, internuclear ophthalmoplegia.[7,12,13] The two major hypotheses regarding the coactivation observed in Duane's syndrome are an anomaly of peripheral innervation and a supranuclear brain-stem disorder. The explanation of Duane's retraction syndrome on the basis of a simple congenital anatomic defect of muscle is not tenable. The distinction between these two mechanisms cannot be made by electromyography alone. Electromyography measures only end-organ activity, and this can be altered by either peripheral or central mechanisms.

The importance of electromyographic data in the understanding of variations of innervation is exemplified by the findings in typical Duane's syndrome, a paresis of abduction with retraction of the globe on adduction. According to Huber's[7] classification (Duane's syndrome Type I), the medial rectus muscle innervation is normal and the lateral rectus muscle innervation on abduction is defective. The decreased innervation on attempted abduction explained the paresis observed; the greater innervation on adduction produced retraction. Blodi, Van Allen, and Yarbrough,[8] however, reported two cases of typical Duane's syndrome in which there was normal innervation of the lateral rectus muscle and contraction of the medial rectus muscle on both abduction and adduction. The mechanism of abduction paralysis in these cases appeared to be the cocontraction of the medial rectus muscle. Thus, two different innervational patterns produced the same clinical syndrome.

Figure 3 shows one possible explanation of the various types of Duane's syndrome (as defined by Huber[7]) and of congenital adduction palsy with synergistic divergence. Peripheral misdirection of the oculomotor (III) or abducens (VI) nerve is the essential ingredient. Anomalies of peripheral oculomotor nerves have been observed pathologically. Hoyt and Nachtigaller[14] collected a number of reports of such anomalies and suggested that substitute innervation of the lateral rectus muscle by an extra branch of the oculomotor nerve (III) could explain Duane's syndrome. Peripheral innervation can vary both quantitatively in the amount of innervation and qualitatively in the origin of the nerve fibers. In Huber's classification, the innervational pattern described as Duane's syndrome Type II is the most similar to that observed in simultaneous divergence. In Duane's syndrome Type II there is an absence of adduction and normal inhibition and excitation in the medial rectus muscle; however, the lateral rectus muscle fires both on adduction and abduction. Thus, the contraction of the medial rectus muscle is counterbalanced by cocontraction of the lateral rectus muscle, causing the apparent adduction weakness. A similar pattern of innervation was present in our case of simultaneous divergence. The striking feature, however, was that, unlike Duane's syndrome Type II, there was a quantitatively greater innervation of the right lateral rectus muscle compared to the right medial rectus muscle on levoversion, thus producing abduction of the right eye at the same time as normal abduction of the left eye.

Duane's syndrome Type III (absent adduction and limitation of abduction) is clinically similar to simultaneous divergence. In Duane's syndrome Type III, the lateral and medial recti muscles behave electromyographically as one muscle and fire on both abduction and adduc-

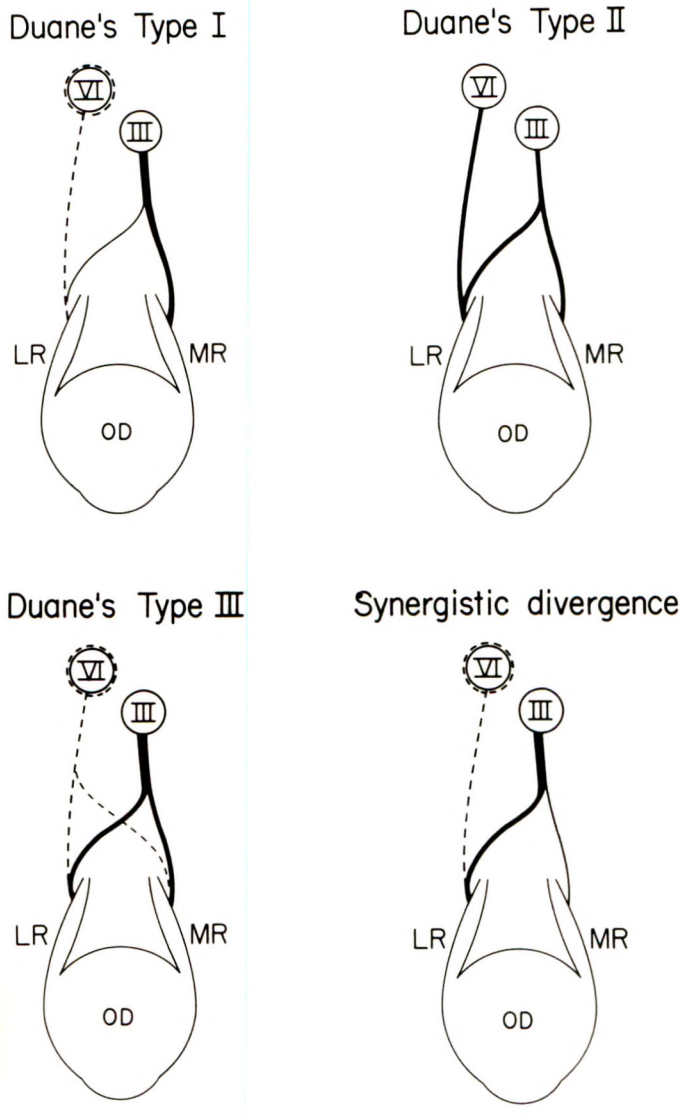


Fig. 3 (Wilcox, Gittinger, and Breinin). Anomalies of peripheral innervation that may explain Duane's syndrome. Lateral (LR) and medial (MR) recti muscles of the right eye and the medial rectus muscle subnucleus (III) and sixth nerve nucleus (VI) with their nerves are shown. The thickness of the line representing the nerve is intended to represent quantitative innervation. Dashed lines indicate hypoplasia or absence of the nerve.

tion. If this is peripheral, both muscles must be innervated by both the oculomotor (III) and abducens (VI) nerves (Fig. 3). In Duane's syndrome Type II and in simultaneous divergence, only the lateral rectus muscle needs dual innervation. This again demonstrates that comparisons among the cocontraction syndromes must take into account both the motility defect observed and the innervational pattern found electromyographically.

A central or supranuclear lesion has also been postulated in Duane's syndrome but the nature of the brain-stem disorder has not been delineated. The proposed mechanisms for other abnormal synkineses are relevant. Associated movements or abnormal synkineses, both congenital and acquired, occur particularly in muscles innervated by the oculomotor, facial, and trigeminal nerves (cranial nerves III, VII, and V).[15,16] Such phenomena as aberrant regeneration of the third

cranial nerve or hemifacial spasm have usually been presumed to be the result of peripheral misdirection of nerve fibers occurring after injury. Ferguson,[17] however, suggested that hemifacial spasm is a central, nuclear or supranuclear, reaction to peripheral axotomy with resulting physiologic changes at the level of the nerve cell body and its dendrites. This results in alteration of the neuron's synaptic milieu. Loewenfeld and Thompson[18] suggested a similar mechanism for the cyclic spasms occasionally observed in congenital third nerve palsies. Wartenberg[15] was the first to argue that aberrant regeneration is itself a central release phenomenon.

A combination of a peripheral lesion and a central synaptic reorganization could also explain synergistic divergence and other variants of Duane's syndrome. Injury to or absence of the sixth nerve early in development could result in reorganization of the synaptic connections of the sixth nerve nucleus and the subnucleus of the third nerve subserving the lateral rectus muscle.

Hepler, Hoyt, and Loeffler[19] suggested that another congenital paradoxical synkinesis, Marcus Gunn (jaw winking) syndrome, is mediated by inhibitory interneurons in the midbrain. Interneurons arising in the sixth nerve nucleus decussate and pass rostrally via the medial longitudinal fasciculus to provide the primary neural input for the contralateral medial rectus muscle motor neurons.[20] The pathways for inhibitory input to the ipsilateral medial rectus muscle are less well understood,[21] but the loss of inhibitory input has been suggested as the cause of the abduction nystagmus regularly observed in internuclear ophthalmoplegia.[22] A sixth nerve lesion in an immature nervous system might result, therefore, in paradoxical excitation of the ipsilateral medial rectus muscle, the cocontraction observed in Duane's syndrome Type I. Similar mechanisms could explain other innervation patterns of Duane's syndrome, including congenital paralysis of adduction with simultaneous divergence. Either central response to a peripheral lesion or peripheral reinnervation producing Duane's syndrome could explain why congenital sixth nerve palsies are so rare compared to congenital third and fourth nerve palsies. A more complete understanding of the retraction syndromes can only be obtained with more clinicopathologic correlations. In the absence of such correlated material, various explanations of cocontraction syndromes—peripheral, central, or a combination of the two—are possible. Whatever mechanism is correct, we think it reasonable to consider synergistic divergence a variant of Duane's retraction syndrome.

Summary

Electromyography of the medial and lateral recti muscles in the right eye of a 7-year-old boy with congenital variable exotropia demonstrated cocontraction. This explained the observed simultaneous abduction on attempted levoversion. Abduction of the right eye increased intraocular pressure to 25 mm Hg from 15 mm Hg. This synergistic divergence should be considered a variant of Duane's syndrome, and, like Duane's syndrome, may occur as the result of anomalies of the peripheral oculomotor nerves or of central synaptic alterations, or of a combined central and peripheral mechanism.

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ACQUIRED CYCLIC ESOTROPIA IN AN ADULT

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Cyclic ocular motor phenomena are most often congenital or the result of acquired neurologic disease. Such conditions include cyclic oculomotor palsy,[1] periodic alternating nystagmus,[2,3] periodic mydriasis,[4,5] migrating pupil,[5] periodic head turns in congenital nystagmus,[6] and cyclic esotropia.[7-18] We studied a patient with previously normal ocular motility who developed cyclic, 48-hour period esotropia in conjunction with spontaneous retinal detachment and subsequent surgery when the patient was 62 years old.

CASE REPORT

A 67-year-old woman had alternate-day esotropia. An ocular examination done when she was 39 years old had disclosed a visual acuity of R.E.: 6/6−4 (20/20−4) and L.E.: 6/7.5+2 (20/25+2) with $-11.25 + 0.25 \times 100$ degrees. An external examination, biomicroscopy, and ophthalmoscopy disclosed small posterior subcapsular cataracts and myopic optic disk configuration. She had not had diplopia or manifest tropia on examination of muscle balance. Another examination in 1971 had given the same results except for a slight increase in myopia.

In April 1974 she suddenly lost visual acuity in the right eye and was hospitalized for treatment of a retinal detachment. She underwent a scleral buckling procedure which involved a 360-degree encircling element, cryotherapy of retinal holes, and draining of subretinal fluid. The lateral and medial recti muscles were isolated and used during the procedure for traction but no muscles were disinserted. The patient had a satisfactory postoperative course with reattachment of the retina. However, in August 1974, she underwent cryoretinopexy of the right eye for new retinal holes. The patient did well postoperatively, recovering her previous visual acuity.

In April 1975, she reported that her right pupil became dilated every other day. The right pupil was 2 mm larger than the left pupil but had normal light and accommodative responses. She was examined at three-month intervals during the next 18 months but there was no evidence of extraocular muscle imbalance. In April 1976, however, she had right-sided esotropia of 25 to 30 diopters and right-sided hypertropia of 10 to 12 diopters, findings verified at three other examinations during the year. She reported "seeing double every other day." Alternate-day examinations showed esophoria of only 4 diopters on her "good" day but esotropia of 25 diopters and right-sided hypertropia of 8 diopters on her "bad" day. An examination in May 1978 gave similar findings.

Reevaluation in May 1979 showed esotropia of 30 diopters and right-sided hypertropia of 12 diopters on her esotropic day. On alternate days she had persistent mild right-sided blepharoptosis and a sluggishly reactive pupil 4 mm in diameter. The left pupil was 3 mm in diameter and reacted normally to light. Both pupils dilated normally with hydroxyamphetamine. The patient said that if she became excessively tired or had more than two drinks on the evening of her "bad" day, she experienced some "turning in" on the good day.

She had not had a squint or strabismus as a child. Review of her earlier records disclosed no ocular motility disturbance but did show severe myopic refractive error. Her daughter had had surgery for large-angle esotropia at 7 years of age without subsequent recurrence or complaint of diplopia.

METHODS

We recorded eye movements with an infrared reflectance technique, with a bandwidth of 140 Hz.[19] The subject viewed an array of light-emitting diodes mounted on an arc 1.52 m away. A headrest and chin support maintained head stabilization. Calibration was carried out on one eye at a time, with the other occluded, so that any fixational ambiguities caused by diplopia were eliminated. We recorded saccadic refixations on both "good" (orthotropic) and "bad" (esotropic) days to targets at 5, 10,

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15, and 20 degrees to the right and left of center.

Results

The patient maintained a 48-hour cycle during 13 months of observation, documented by still photography (Fig. 1), videotape recordings, 16-mm motion pictures, clinical examinations, and quantitative eye-movement recordings. Analysis of the eye-movement records disclosed normal saccadic velocity-amplitude relationships to amplitudes of 15 degrees. Frequent glissadic eye movements[20] were present in the right eye. We found normal saccadic dynamics on both orthotropic and esotropic days. Figure 2 shows a series of 10-degree saccades on an orthotropic day for both eyes. Figure 3 shows the results for an esotropic day for both eyes. Because the patient had diplopia on esotropic days, binocular viewing produced ambiguous results. Alternate covering and uncovering during steady fixation produced slow drifts with the characteristics of vergence or glissadic movements.

Discussion

Cyclic esotropia is rare; published reports suggest that it occurs in one out of 1,000 to 30,000 cases of childhood strabismus. Most patients have congenital strabismus and regular rather than intermittent cycles of esotropia at ages 4 to 10 years.[7,9,13] The cycles are usually alternate-day,[8,9] but one- and four-day periods have also been reported.[7,13] Cases acquired in childhood after head injuries have been documented.[12-14] Most children improve after extraocular muscle surgery but a number have developed the condition after strabismus surgery.[10,15] Others have noted a change after refractive correction[13] or a gradual change to persistent deviation in six to 12 months.[9]

The present case was unique because onset occurred in adult life after surgery and because the pupil and eyelid were involved first. Another unusual feature was that fatigue and alcohol prolonged the esotropic half of her cycle and shortened the orthotropic portion of the cycle. The usual periodicity resumed the next day.

We did not study the cyclic pupillary phenomena ourselves because they were not present at the time of our examinations. The description suggests that there was periodic sympathetic hyperreactivity during her esotropic day or that there was some cyclic oculomotor nerve dysfunction. If the third nerve had been injured at the time of surgery, initial postopera-

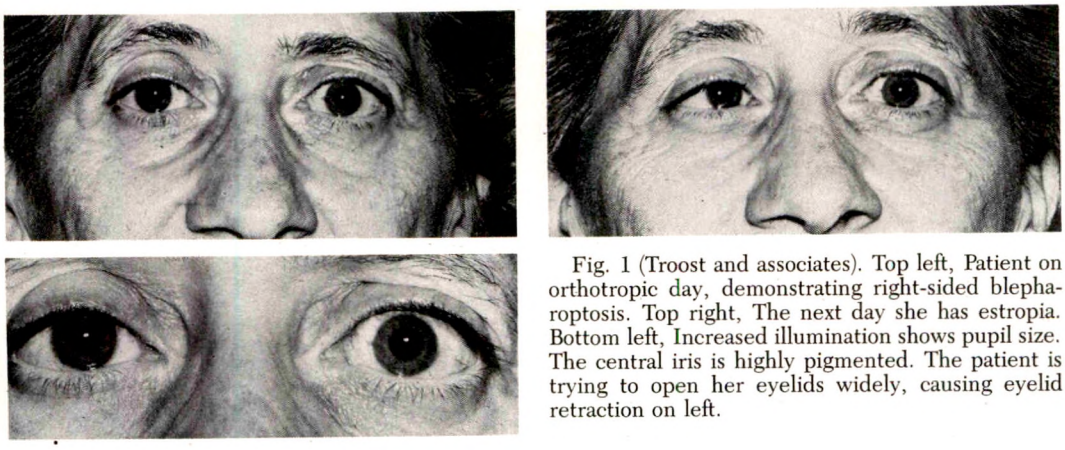


Fig. 1 (Troost and associates). Top left, Patient on orthotropic day, demonstrating right-sided blepharoptosis. Top right, The next day she has estropia. Bottom left, Increased illumination shows pupil size. The central iris is highly pigmented. The patient is trying to open her eyelids widely, causing eyelid retraction on left.

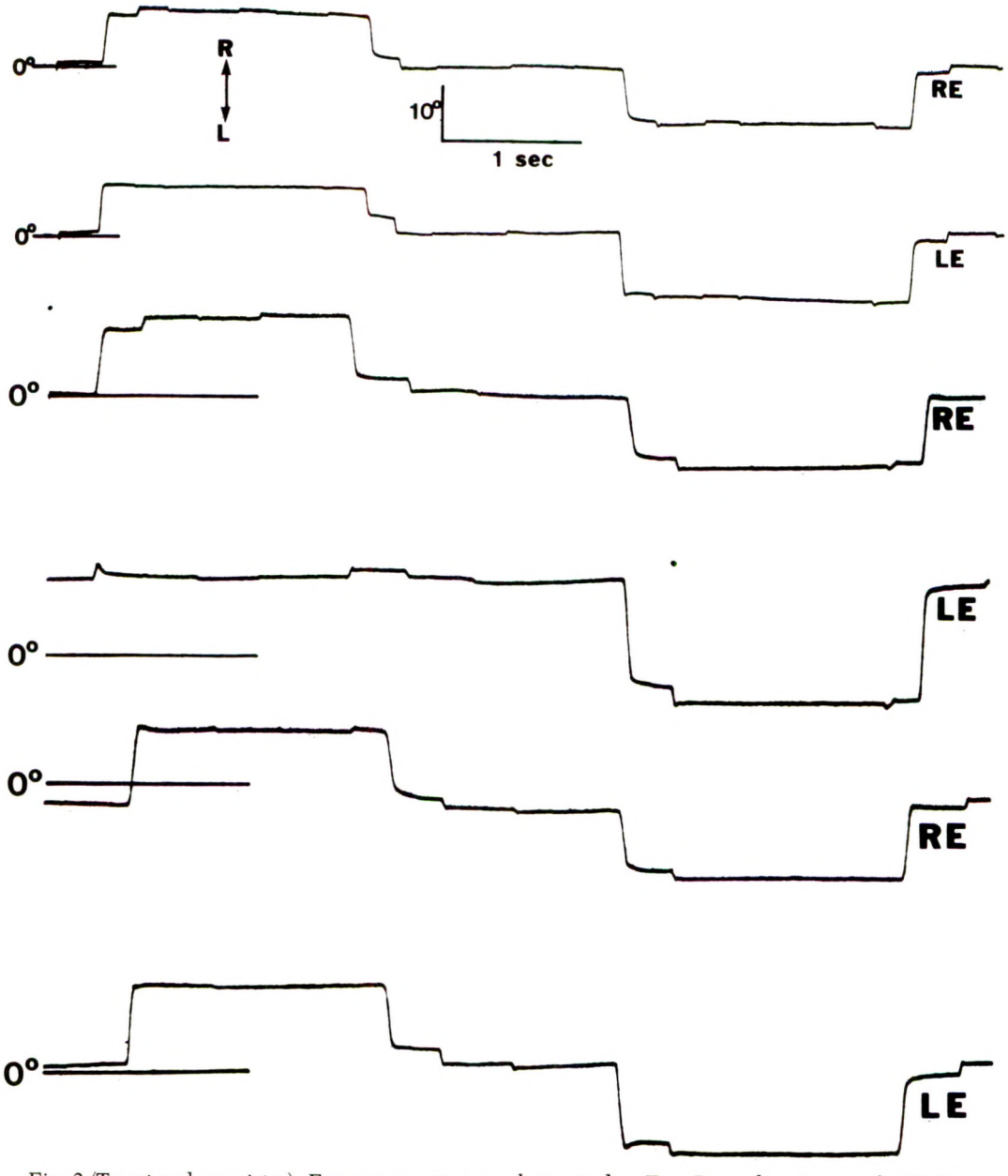


Fig. 2 (Troost and associates). Eye movements on orthotropic day. Top, Binocular viewing of ±10-degree targets. Upward deflection indicates rightward movements and downward deflection, leftward movements. Movements are essentially normal. Middle, Viewing with the right eye shows a 10-degree esophoria. Rightward refixation produces only a phasic movement in the covered eye; refixation to the left produces a larger saccade in the covered eye than in the viewing eye, but still leaves the left eye short of the target. Bottom, Viewing with the left eye shows 3-degree esophoria. Saccades are now comitant in both directions.

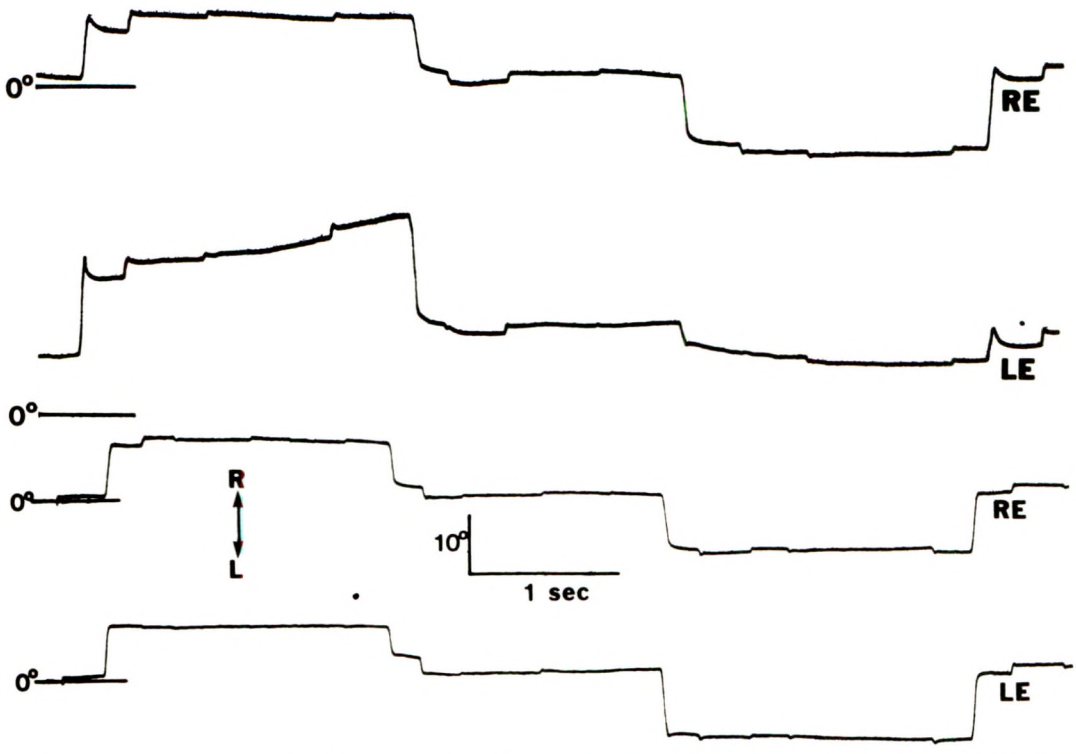


Fig. 3 (Troost and associates). Eye movements on esotropic day. Top, Viewing with the right eye. The left eye moves further than the right eye and drifts in on right gaze, but is underactive on left gaze. Bottom, Viewing with the left eye. The right eye is 10 degrees esotropic. Again, with the left eye viewing the movements are grossly comitant.

tive diplopia and oculomotor defect might have been expected in addition to blepharoptosis. She had no signs of third nerve regeneration. If sympathetic hyperreactivity was present, blepharoptosis was unexpected. No obvious explanation accounts for all the findings.

Because the patient had esophoria (Fig. 2) on her orthotropic day and her daughter had strabismus, one might argue that the initial visual afferent defect after retinal detachment unmasked her phoria, that is, her latent deviation decompensated to a manifest one. This does not explain her alternating cycle. Other patients have had decreased visual acuity during their strabismic days or have had amblyopia in one eye.[7]

Circadian rhythmicity occurs in a variety of physiologic processes, including intraocular pressure, temperature, headache, psychological states, and the like.[8,9] Among the suggested causes of cyclic esotropia are biologic clocks,[8] cerebral damage,[9] prior encephalitis,[17] and trauma.[14] In congenital nystagmus, periodic null shifts have caused head turns,[6] and such mechanisms have been used to explain periodic alternating nystagmus.[2] Although the periods in these disorders are hours or minutes, they do reflect regular periodic behavior in eye-movement control.

Even though our patient showed no additional neurologic or neuro-ophthalmologic abnormalities, we speculated that the initiating peripheral ocular event later produced secondary central nervous

system adaptive changes in the oculomotor control system.[21] Quantitative eye-movement recordings offer some support for this idea. The normal saccadic velocities on all recordings support the concept that no persistent peripheral dysfunction occurred. Saccadic slowing has been reported in partial sixth nerve injury and extraocular muscle injury.[22,23] There were significant eye-movement differences when we tested binocular and monocular viewing. Binocular viewing on the orthotropic day produced a virtually normal record except for the tendency of adducting saccades in the right eye to be glissadic. However, when the right eye was used, the left eye turned in 10 degrees; it did not move when the gaze was shifted to the 10-degree right target. Looking at the 10-degree left target produced proper fixation in the right, viewing eye, but moved the left (covered eye) 16 degrees, leaving it 4 degrees short. When the right eye was occluded, it turned in 3 degrees but when refixations were made with the left, viewing eye, the right eye moved comitantly. This difference is striking. It was somewhat variable; during viewing with the left eye, the covered right eye was initially turned in about 5 degrees and did move somewhat when small refixations were made.

One explanation for the unusual behavior on the orthotropic day is that a tendency to turn in persisted during monocular viewing with the right eye. The innervation needed to maintain primary position, in the absence of a fusional stimulus, drove the yoked left eye medially while it was covered. This does not account for the lack of comitance. Nevertheless, the range of abnormalities present on a day when the patient appeared to have normal ocular motility was striking. The responses on an esotropic day were somewhat less remarkable. During monocular viewing, the occluded eye turned in about 10 degrees. Again, asymmetries of movement were more pronounced during viewing with the right eye, but movements to the left were too great and those to the right too small—the opposite of the findings on the orthotropic day.

We can not explain all the oculomotor abnormalities disclosed by the eye-movement recordings. Even on an orthotropic day, her oculomotor system functioned in a grossly abnormal manner as soon as binocular vision was interrupted, supporting the idea that cyclic esotropia consists of strabismus interrupted by periodic intervals of fusion, rather than normal function intermixed with periods of disturbance.

Summary

A 67-year-old woman developed periodic alternate-day esotropia, documented with videotape, 16-mm film, and quantitative eye-movement recordings, after retinal detachment surgery. The eye movements suggested that central reprocessing and adaptation to a peripheral defect was in part responsible for this adult form of acquired cyclic esotropia.

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OCULAR TORSION AND THE PRIMARY RETINAL MERIDIANS

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When the head is tilted the eyes are said to undergo a rotation in the opposite direction around the visual line in order to maintain the original angle of the retina in space.[1] This countertorsion is called static when it persists at a given angle of steady head tilt and dynamic when it occurs during head-tilt.[2,3] According to published reports the extensiveness of static torsion varies from zero to 90 degrees. However, most researchers found static countertorsion to be about 10% of large static head-tilts.[4-11] Torsion is, therefore, considered an imperfect or partial compensatory mechanism for retinal tilt, with further adaptation occurring in the central nervous system.[12] Dynamic torsion is said to have a larger amplitude than static torsion.[4] In dynamic countertorsion, the eyes are said to lag behind the head movement and then to undergo a series of intermittent, saccadic, small-amplitude torsional movements in the direction of the head-tilt. The eyes are said, however, not to catch up with the head, but to leave a persistent static countertorsion, related in amplitude to the degree of head-tilt.[13]

A vestibulo-ocular reflex is considered responsible for eye torsion associated with head-tilt. This reflex is thought to be involuntary, and independent of vision and of impulses arising from neck muscle receptors.[1-3] For static torsion, the afferent reflex impulses are thought to arise from the otolith organs, with the right and left organ each contributing one half of the response.[1,4,14] More extensive dynamic torsion is said to result from stimulation of both the otolith organs and the anterior and posterior semicircular canals.[4] The finding that simultaneous stimulation of the anterior and posterior semicircular canals brings about ocular torsion appears to support this.[15]

Ocular torsion has been reported to be associated with both symmetrical and asymmetrical convergence.[16-18] For each 10 degrees of convergence, there is said to be one degree of extorsion in the horizontal plane; and the amount of extorsion is said to increase with convergence in elevation and decrease with convergence in depression. Convergence has also been found to be unassociated with torsion.[19]

In previous experiments on the function of the extraocular muscles,[20] I was unable to measure static ocular torsion in human subjects or rhesus monkeys as other researchers have done. Moreover, I believe that the existence or nonexistence of ocular torsion in humans during head- and body-tilt, and in normal version and vergence eye movements, is a central issue in oculomotor physiology. The resolution of this question is important for the interpretation of clinical oculomotor defects and of experimental findings in vestibular physiology. To reinvestigate this question, I measured the effects of static and dynamic head- and body-tilt and of convergence upon ocular torsion. This article describes my methods and the results for 15 normal experimental subjects and for 400 patients without neurologic or extraocular muscle abnormalities.

Definitions—The center of rotation of the head is the origin of a rectangular coordinate system that is drawn with the head in its "normal" erect position and with its horizontal axes parallel to the terrestrial horizon. In Figure 1, X′ desig-

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nates the horizontal head axis, Z′ the vertical axis, and Y′ the anteroposterior axis. The center of rotation of the eye is regarded as a fixed point in the orbit which has zero velocity during rotation of the eye.[16] This point is also the origin of a rectangular coordinate system in which X is the horizontal, Z the vertical, and Y the anterposterior axis. These orbital axes are parallel and fixed in regard to the head axes.

Listing's plane is the XZ plane.[16,21,22] It corresponds to an X′Z′ head plane that includes the centers of rotation of both eyes. The primary retinal plane is fixed in the eye and is defined anatomically by the center of ocular rotation, the fovea, the retinal raphe, and the visual line. The primary retinal meridian is contained within the primary retinal plane (RMF in Fig. 1, A). It is the same as the so-called horizontal retinal plane, but only when the eyes fixate the horizon with the head erect.

Listing's Law states that "every movement of the eye in which the line of fixation travels from the primary position to any other position is equivalent to a simple rotation around an axis perpendicular to the meridional plane that passes through the final position of the line of fixation."[22] Said more simply, all eye movements can be considered to occur around axes in Listing's plane. Oblique ocular rotations around axes in Listing's plane produce an apparent torsion (pseudotorsion) because of the projection of lines from a spherical surface onto a flat plane.[16,21,22]

Ocular torsion takes place only when the eye rotates around any axis outside of Listing's plane. It takes two forms: wheel-torsion when the eye rotates around the visual line, and eccentric-torsion when the eye rotates around any other axis outside of Listing's plane. Eccentric-torsion is a compound movement in which torsion is combined with both vertical and lateral ocular displacement. Pathologic ocular torsion is termed cyclotropia.

Material and Methods

Apparatus—A biomicroscope (slit lamp) was used, with an adjustable slit of light that could be rotated 180 degrees. It

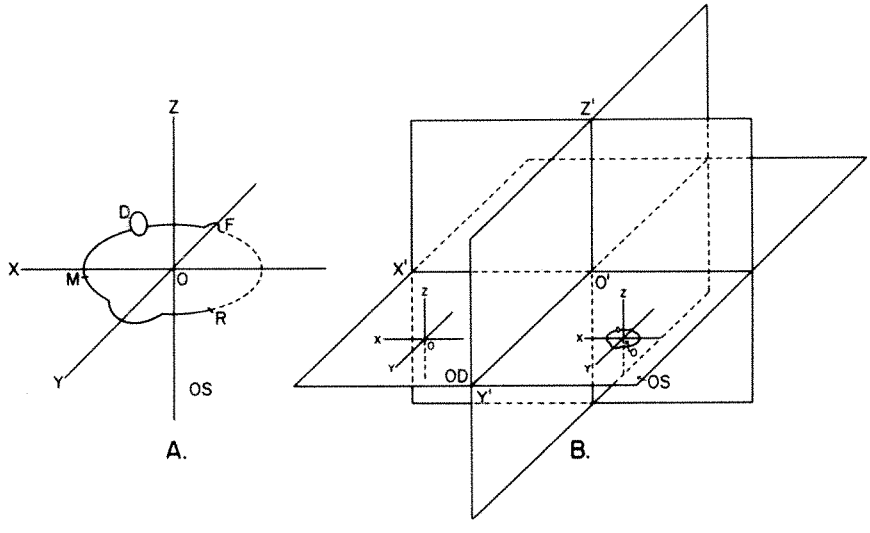


Fig. 1 (Jampel). The reference system for eye position (see text). A, Horizontal schematic section of the eye and the rotational axes. B, Relationship between the ocular and head coordinate systems. The line RFM represents the primary retinal meridian; D, optic disk; F, fovea; FR, retinal raphe (dashed line); OD, right eye; OS, left eye.

was modified so that either ciné or still photographs could be taken through its optical system. The head-brace and chin-cup of the photo slit lamp were replaced by a square bar, held firmly in a lathe-vise, so that it could be moved accurately in the X, Y, and Z spatial planes, corresponding to the head planes. A dental impression bite-bar was fitted perpendicularly to the end of the metal bar, so that it could be rotated around its anteroposterior axis (Fig. 2). Fixation points were placed on and around a circular glass plate and frame that covered and protected the objective lenses of the photo slit lamp.

A modified bite-bar, firmly attached to the adjustable lathe-vise, was used without the slit lamp to ciné photograph eye movements during dynamic head- and body-tilt. The bite-bar was attached to a round metal beam 20 inches long. At the end of the beam a perpendicular bar, containing fixation targets which could be slid toward or away from the subject along the round metal beam, was attached. The bite-bar and attachments were light enough in weight so that the subject could twist the bite-bar with his mouth as he tilted his head at different velocities. The ciné camera was fitted with a 50-mm lens with an extension tube, so that the eye would be magnified and fill about one third of the 16-mm picture frame.

Procedures—In the first group of experiments, 2% pilocarpine was instilled in the experimental subject's eyes 30 minutes before the test to eliminate pupillary movements and to stabilize iris markings. A piece of white adhesive tape was placed just below the lower orbital rim of the subject's eye. On top of this tape a piece of narrower calibrated yellow adhesive tape with a sharp edge was placed. The bite-bar was used to fix the subject's head in the erect position in front of the photo slit lamp. The bite-bar was carefully kept as perpendicular as possible to the optical axis of the photo slit lamp and to the atlanto-occipital articulation, so that when the head was

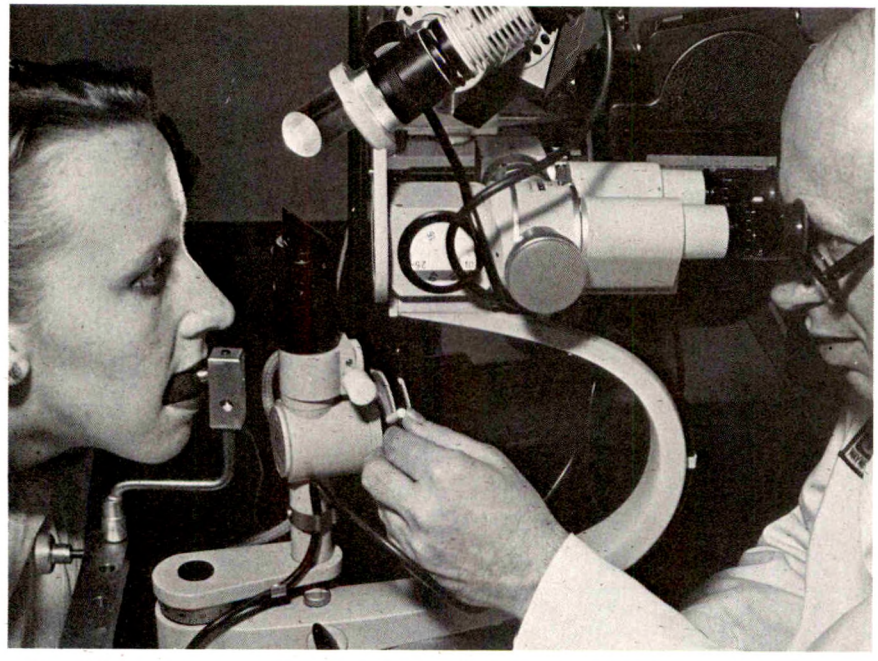


Fig. 2 (Jampel). The apparatus used (see text). The investigator is manipulating the size and the position of the slit and monitoring fixation. The results are being recorded with a ciné camera (upper right, behind investigator).

tilted it remained in a frontal plane. The illuminated biomicroscopic slit was narrowed to 0.1 mm or less and rotated so that it corresponded exactly with the sharp upper edge of the yellow adhesive tape below the subject's eye. For precision this was done under a ×10 or ×40 magnification (Fig. 3, A). The position of the illuminated slit thus represented the exact position of the head (±0.2 degree). The subject was told to maintain fixation on the central mark on the protective glass plate covering the objective lenses of the slit lamp. The slit was then moved upward onto the surface of the iris and placed across identifiable iris markings or through the center of the pupil (Fig. 3, B). Still and ciné photographs of the position of the slit on the iris were now taken at ×6, ×10, and ×16 magnifications, using a built-in electronic flash or focal illumination. The slit was then moved downward again from the iris, to doublecheck its alignment with the edge of the yellow tape. To record the effects of head-tilt, the head was then rotated around the axis of the bite-bar into different positions of increasing inclination from the erect position and, for each position, the slit was rotated to again match the edge of the yellow tape. It was then moved onto the iris across the pupil as before, and repeat photographs were taken. Each step of the experiment was performed under the direct visual control of the investigator.

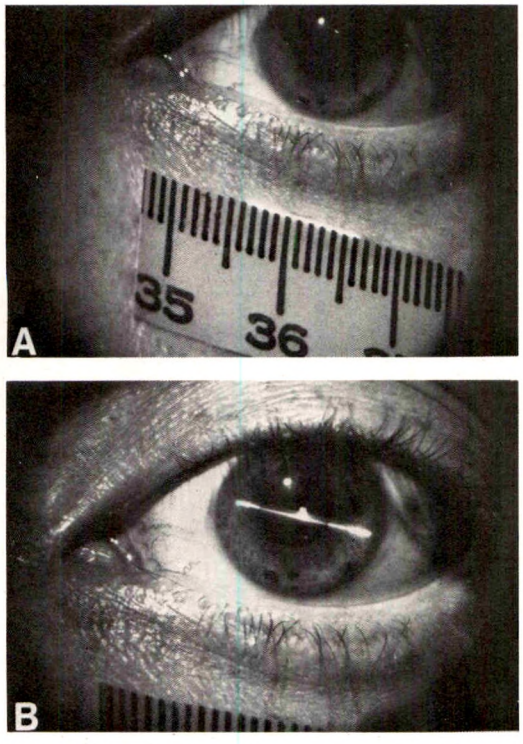


Fig. 3 (Jampel). Part of the experimental technique (see text). A, The calibrated tape is on the orbital rim. The illuminated slit is made to coincide with the edge of the tape under ×10 magnification, and, therefore, represents the position of the head. When the head is tilted, the slit is rotated to again coincide with the edge of the tape. B, The slit has moved upward onto the iris to be photographed.

A simple modification of the above method can be used for large numbers of clinic patients. An ordinary slit lamp is used without a bite-bar, and no pilocarpine is instilled. Adhesive tape with a sharp edge is placed on the patient's cheek bone, forehead, or side of the nose. The patient's head is then tilted within the slit lamp head-brace or chin-cup into various inclined positions. For each photograph, the slit is rotated to first match the edge of the adhesive tape and then brought onto the iris across the center of the pupil.

To observe whether torsion was associated with convergence or with oblique conjugate eye movements, the slit was kept either vertical or horizontal, and iris markings were identified with the slit. The first picture was then taken. After convergence or version of the eyes, the slit was moved to correspond to the previously identified markings and then rephotographed.

A second method was used to determine whether ocular torsion was associated with convergence. After baseline ciné pictures were taken, an accommodative target was slowly moved toward the patient's eyes in a parasagittal plane. This target moved in the visual line of one eye so as to produce asymmetrical convergence, that is, the homolateral eye did not move while the contralateral eye

converged. When the target was about 10 inches from the eye, it was moved slowly across the visual field in a frontal plane, producing a combination of conjugate gaze and convergence. The eye movements were ciné photographed during convergence with the eyes in horizontal and in vertical gaze.

In the second group of experiments, a local anesthetic was instilled in the subject's eyes; a calibrated tape was placed beneath the orbital rim and a piece of eggshell membrane, measuring about 1 × 3 mm, was placed on the cornea. The subject had to be able to inhibit blinking while biting the bite-bar and tilting the head at different velocities and amplitudes. During this time the eyes were fixated on a target that moved with the same velocity as the head. Ciné photographs of these movements were taken for analysis at 50 frames per second.

Measurements—The ciné photographs were analyzed, picture by picture, with a back-projecting motion-analyst ciné projector. Tracing paper was placed over the projected ciné frame on the translucent glass. A tracing of the tape edge, iris markings, and slit edge on the iris or eggshell membrane was drawn. The next frame was then projected and the tracings superimposed on the projection, to determine correspondence.

Comments—These methods did not use after-images or electronic monitoring but relied on direct observation and objective measurements. Fixation was controlled so that the eye could be photographed in the same position in the orbit for any degree of head-tilt; the head was tilted in a fixed frontal plane. If fixation and head position are not rigorously controlled in such experiments, so-called pseudotorsion may be measured, which is a major artifact in such measurements. This phenomenon is best explained by Alpern[16] and is shown in Figure 4. Additional artifacts may appear because the eyeball is a complex ellipsoid and not a perfect sphere, the corneal curvature and thickness vary on the eye surface, and the iris is not a flat plane, but has a conical shape.

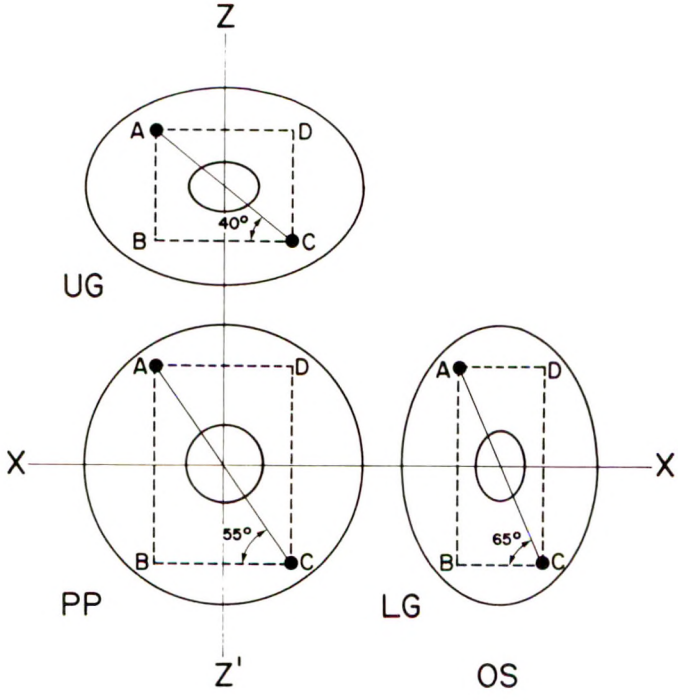


Fig. 4 (Jampel). Pseudotorsion may be understood without resort to mathematics. The large circle represents the iris and the small circle the pupil. The line AC connects two iris markings (darkened circles). When the eyeball rotates laterally (LG) or upwards (UG) two sides of the rectangle ABCD become foreshortened when viewed from the front (in LG, AD, and BC, and in UG, AB, and DC). This causes angle ACB to appear larger in LG and smaller in UG. In LG, extorsion and in UG intorsion appears to have taken place. OS, left eye.

RESULTS

Static torsion—Fifteen subjects with normal extraocular movements were studied by the photo slit lamp technique. In these subjects no static torsion could be measured for head- or body-tilts that were not readily explicable by the artifacts mentioned previously. This was true for both eyes and for any fixation point that maintained the eye in a stable orbital position for any head- or body-tilt. Two subjects were ciné photographed while standing upright and standing on their heads without the bite-bar or slit lamp. Comparison of the photographs frame-by-frame disclosed no difference in the eye positions elicited in these two postures. Also, no static torsion was observed with head-tilt while the subjects were prone, lying on their sides, or supine. The method was used without the bite-bar and photography in 200 patients (ages 7 to 87 years) during routine ocular examinations. No ocular torsion was found at head-tilts of about 30 to 60 degrees in these patients that was not also explicable by artifacts (Figs. 5 and 6).

Dynamic torsion—Eight subjects with normal extraocular movements were studied with the corneal eggshell membrane technique. Head-tilts ranging up to 135 degrees and with velocities from 3 to 70 degrees per second were ciné photographed at 20 and 50 frames per second. Analysis was limited by the fact that the method did not allow the measurement of eye movements at less than 20-msec intervals. However, the following was apparent: During head-tilts of more than 3 degrees per second the eyes underwent intermittent, saccadic, small-amplitude torsional movements that preceded the head movements. In between these saccades and at the end of the head-tilt, the eyes and head were aligned (±0.1 degree), disclosing no persistent torsion.

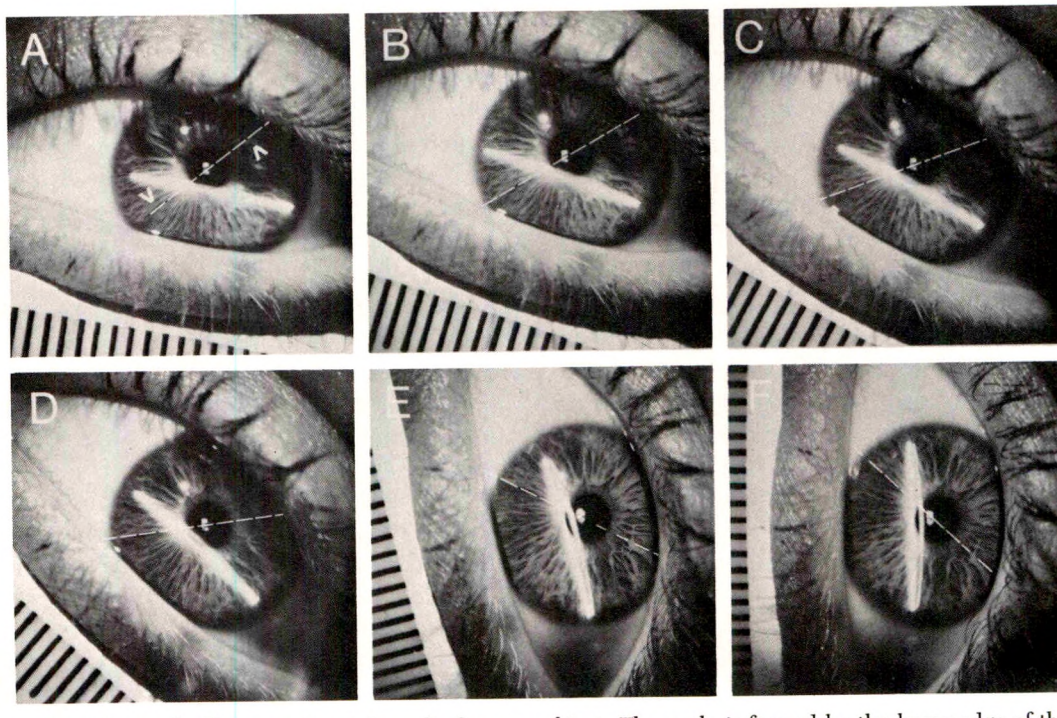


Fig. 5 (Jampel). The experimental results for one subject. The angle is formed by the lower edge of the illuminated slit on the iris and a dashed line drawn through distinct iris markings (A, arrowheads). The acute angle measures 49 degrees and is the same in all frames. If intorsion took place the acute angle would become larger. In A the head is rotated 11 degrees; in B, 19 degrees; in C, 30 degrees; in D, 43 degrees; in E, 77 degrees; and in F, 89 degrees.

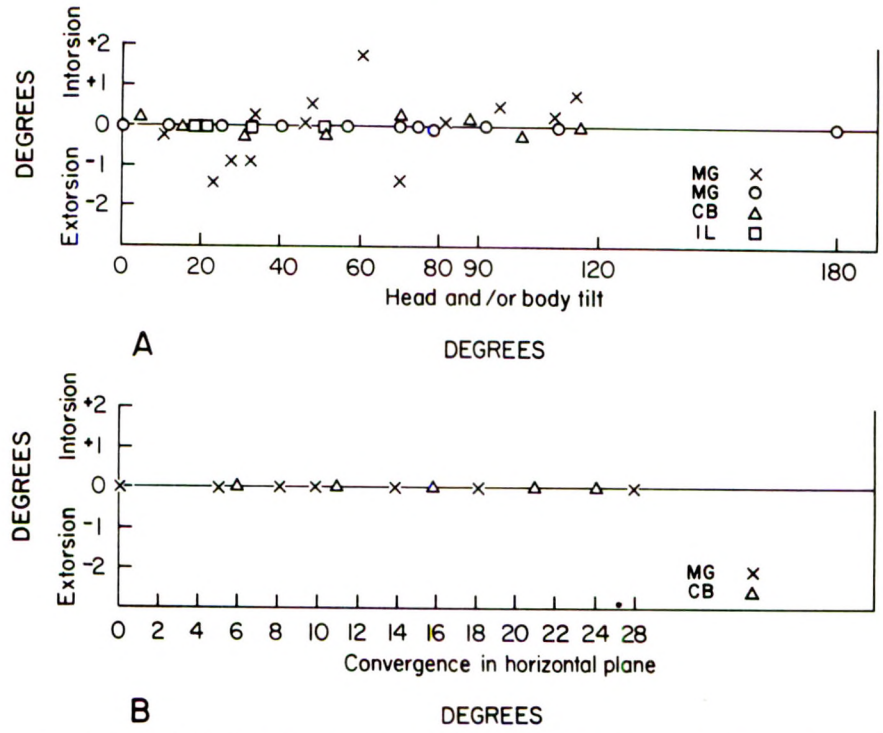


Fig. 6 (Jampel). Static torsion in head-tilt and convergence. Because the results were negative, a graph of only a few subjects is shown. The symbols X and O represent an experiment on the same subject at different times. In the first experiment, X, fixation was imperfect and pseudotorsion was measured.

These torsional saccades accelerated to a velocity of at least 100 degrees per second in about 20 msec, preceded the head by 2 to 3 degrees, and then decelerated to the velocity of the head-tilt. In other words, the torsional eye movements did not lag behind the head movement, as had been expected. On the contrary, the eyes moved ahead of the head during the saccades, and the head caught up in the intervals between the saccades. Ocular fixation did not affect the saccades and none were photographed when the head tilted at more than 60 degrees per second. The number of torsional saccades occurring during any head-tilt movement was related to the amplitude and velocity of the head movement. Occasionally, with the head in a static tilted position, spontaneous torsional saccades were observed that were in excess of the head-tilt (Figs. 7 to 9).

Torsion associated with convergence—Fifteen normal subjects were studied extensively with both the photo slit lamp and the eggshell membrane techniques. Symmetrical and asymmetrical horizontal convergence were studied. Convergence was also studied with the head flexed and extended, with base-in and base-out prisms, and with subjects prone, on their sides, supine, and standing on their heads. In none of these positions was torsion found that was not explicable by artifacts (Fig. 6). Comparison of the positions of iris and conjunctival markings showed no difference between the location in the ocular adduction produced by conjugate horizontal gaze and that produced by convergence. In vertical convergence, the eyes were in oblique gaze positions and the phenomenon of pseudotorsion was measured. These measurements agreed with those taken with the eyes in conjugate oblique gaze. Two hundred consecutive patients (ages 2 to

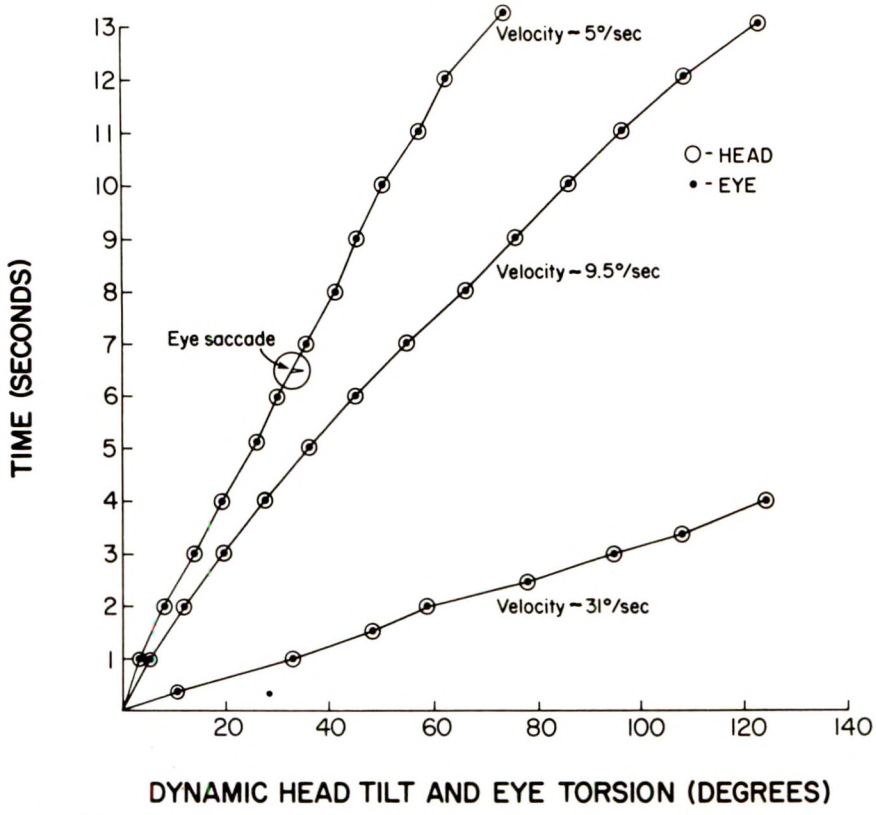


Fig. 7 (Jampel). The response of the eye during head-tilt. The head and the eye remain aligned except for intermittent, small-amplitude, torsional saccadic eye movements. The small circle encloses one of these saccades; the other saccades are not shown. For a head-tilt of 120 degrees there are about 12 saccades.

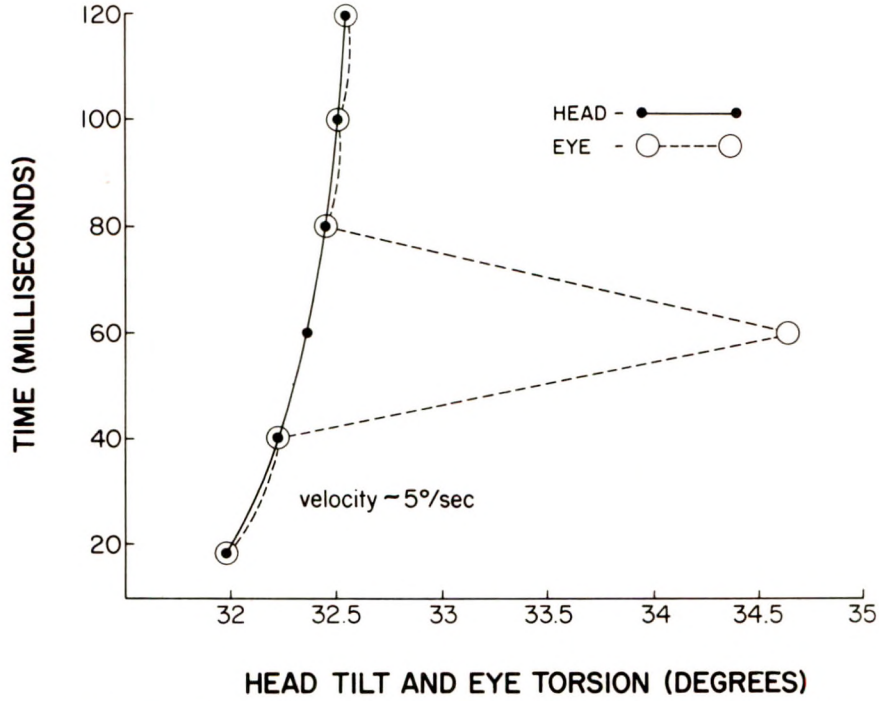


Fig. 8 (Jampel). The details of one saccade. The eye precedes the head by accelerating to at least 100 degrees per second for about 20 msec and then decelerating in about 20 msec to the velocity of the head. The amplitude of the saccade is at least 2.2 degrees.

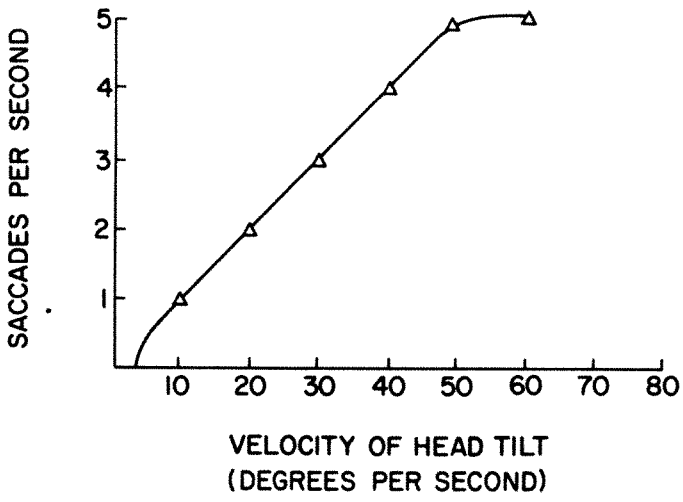


Fig. 9 (Jampel). The number of saccades is dependent on the velocity and the amplitude of the head-tilt. When the head tilts at more than 60 degrees per second no saccades are seen.

74 years) with clinically normal eye movements were examined during the course of routine ocular examinations with a slit lamp and magnification up to ×40. In no case was torsion observed in symmetrical or asymmetrical convergence. Also, in no instance was cyclophoria observed in the patients with the routine cover-uncover and alternating-cover tests.

Discussion

My measurements on normal human subjects failed to show static ocular torsion in head- and body-tilts of up to 180 degrees, at least none within the limits of accuracy of the method (less than ±0.1 degree). Nor was there measurable static ocular torsion upon convergence of the eyes that could not be explained as artifactual. Also, my present and previous experiments and those of others further confirm that no torsion occurs in oblique conjugate gaze.[21-24]

Ciné analysis of eye movements during dynamic head-tilt showed the occurrence of intermittent, saccadic, small-amplitude, torsional movements. These saccades preceded the head-tilt and the eyes did not lag behind the head as reported by others.[13] After each torsional saccade, alignment of the eyes and head were reestablished, and at the cessation of the head-tilt no persistent torsion was measurable. The function of these torsional saccadic movements may be to facilitate alignment of the eyes with the head by anticipating the final head position.

My findings are at variance with those of other investigators.[1-15] I believe these differences are caused by experimental artifacts, especially those that produce pseudotorsion (Fig. 4). Such artifacts may result from the use of after-images, corneal contact lenses, or insufficient control of head position and eye fixation. The possible artifacts associated with the many techniques for measuring ocular torsion are discussed in detail by Howard and Evans.[25] My ciné methods are direct and objective, require no complex head- and body-holding devices, provide for monitoring of eye and head position with magnification, and allow the results to be ciné projected for evaluation and measurement by many observers.

Ocular torsion, in addition to occurring during head-tilt, occurs under pathologic conditions that disturb the integrative mechanisms of the brain, and under special experimental conditions that elicit ocular adjustments to factors not usually present in the normal visual environment. Thus, I have observed ocular torsion (cyclotropia) in certain types of extra-

ocular muscle paralysis, strabismus, and abnormal ocular movements. These torsional movements are usually associated with both vertical and lateral eye movements (eccentric-torsion) and are rarely wheel-like. Also, torsion has been produced experimentally by rotating optokinetic disks,[26,27] by complex visual targets that induce cyclophoria,[28] and by visual feedback training.[29] Optokinetic responses, in general, are attempts to maintain a stable visual scene in the face of environmental movement relative to the position of the head. Cyclophoria is a compensating motor response to torsional retinal disparities. Visual feedback training enables the eyes to realign after-images presented to the retina at different angles. These phenomena are neuromuscular mechanisms whereby the brain adapts to or learns to cope with torsional visual sensory imbalances and to reestablish alignment. The point is that the absence of static ocular torsion is the normal steady state of the eyes. The brain strives to achieve this state by overcoming whatever obstacles are placed in its path.

I believe that the absence of static ocular torsion is a central factor in the normal oculomotor physiology of man. In addition to being involved in moving the eyes, the oculomotor system is involved in maintaining the retinas in a constant relationship with each other and with the brain during all head and gaze positions, and in the face of whatever sensory or motor obstacles may arise. This constancy may be stated as a generalization: the primary retinal meridians become aligned with each other in the normal individual during binocular fixation regardless of the position of the head in space and the point of ocular fixation. As a corollary: the primary retinal planes develop a constant angular relationship with a fixed horizontal head (brain) plane, regardless of the position of the head in space and the direction of gaze.

The consensus of vestibular physiologists is that ocular countertorsion is a vestibulo-ocular reflex elicited mainly by the otolith organs that function to maintain the original angle of the retinas in space with regard to the horizon.[1-3] But how can this consensus be valid if there is no static torsion in man? Is it not possible that head- and body-tilt cause simultaneous excitation and inhibition of different parts of the vestibular apparatus in such a way as to neutralize any signals that might produce static ocular torsion? The vestibular apparatus in man may be concerned primarily with providing information to the brain as to the position of the head with respect to gravity and the horizon. The eyes, in turn, are then stabilized to a reference plane in the brain and not to the horizon.

In the clinical analysis of oculomotor defects the oblique extraocular muscles are supposed to produce ocular torsion which increases as the eye adducts.[30] But if there is no static torsion normally associated with head-tilt, with convergence, and with oblique conjugate gaze, then the oblique muscles cannot normally be torters. From this and other experimental work,[31] I have concluded that the main function of the oblique muscles is to inhibit ocular torsion rather than produce it, and thereby to help maintain the alignment of the primary retinal meridians during all head and gaze positions.

SUMMARY

I measured ocular torsion during and after head- and body-tilt, ocular convergence, and conjugate gaze. Extensive examination on 15 normal human subjects and additional observations on 400 consecutive patients without extraocular motor defects failed to show static ocular torsion. During dynamic head-tilt, intermittent, small-amplitude, saccadic, wheel-like torsional eye movements were recorded as moving faster than the head so that the eyes preceded the head. Between these intermittent saccades,

and at the end of the head-tilt, there was no residual ocular torsion.

The absence of static ocular torsion allows for formulation of a generalization linking the oculomotor to the visual sensory system. The primary retinal meridians become aligned with each other in the normal individual during binocular fixation, regardless of the position of the head in space or the point of ocular fixation.

The vestibular and oculomotor systems function to inhibit rather than produce ocular torsion; the eyes are oriented to a plane in the brain rather than to the horizon. In particular, the oblique muscles function mainly to maintain the alignment of the primary retinal meridians and, therefore, to inhibit torsion in the normal state.

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MYELINATED RETINAL NERVE FIBERS

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Myelinated nerve fibers of the retina and optic nerve head are common, clinically conspicuous, and scientifically provocative developmental anomalies. Recognizing these properties, we report data derived from our clinicopathologic study of myelinated nerve fibers located in the sensory retina and optic nerve head.

In the course of normal prenatal development, myelination of the optic nerve begins at the lateral geniculate body, progresses toward the eye, and terminates posterior to the lamina cribrosa of the sclera before birth.[1-3] As a developmental abnormality, however, myelination occasionally extends anterior to the lamina cribrosa along nerve fibers of the optic nerve head (defined as the intraocular portion of the optic nerve anterior to the lamina cribrosa) and the sensory retina. The retinal myelination may be continuous with or completely separate from the optic nerve head.

Myelinated nerve fibers of the retina are reported in 0.3% to 0.6% of ophthalmic patients, are more common in male than in female patients, and are bilateral in about 20% of affected individuals.[4-7] Ophthalmoscopically, the myelin sheaths surrounding nerve fibers of the optic nerve head and retina have an opaque white appearance. Clusters of these opaque white fibers produce gray or dense white patches.

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Pathologic features of myelinated retinal nerve fibers were described by Virchow[8] in 1856, and additional anatomic characteristics of the myelin sheaths were noted thereafter by several authors.[9-11] Significantly, oligodendroglia, thought to be responsible for myelination, are normally present in the optic nerve head but are not present in the retina.[9-11] Thus, despite considerable study, the pathogenesis of myelinated retinal nerve fibers has not been established.

In general, previous reports concerning clinical and pathologic features provided substantial information, but raised numerous questions regarding myelinated fibers of the retina and nerve head. Consequently, we carried out this clinicopathologic study to determine the incidence of myelinated nerve fibers in the retina and optic nerve head, to establish the gross, microscopic, and ultrastructural characteristics, and to evaluate the clinical manifestations of the abnormality.[12] In studying clinical manifestations, we focused on myelinated retinal nerve fibers associated with ipsilateral myopia, amblyopia, and strabismus.[13]

SUBJECTS AND METHODS

We used 3,968 autopsy cases in which eyes were available for study, and a clinical series of 32 patients with myelinated nerve fibers of the retina.

The 3,968 consecutive autopsy cases excluded stillbirths but included all other autopsy cases. Both eyes were available from all cases, so we examined a total of 7,936 eyes obtained at autopsy.

Eyes were removed as soon as possible after death, fixed in formalin for seven days, and maintained in 50% ethanol thereafter. Gross sectioning, macroex-

amination, and recording of data were done by standard methods.[14] We supplemented standard staining of microsections with Masson's trichrome, Luxol fast blue, Weil's stain, and PAS preparations. We sectioned five lesions in a step-serial fashion with India ink scleral marks as guides.

To evaluate the integrity of the lamina cribrosa in eyes with retinal and nerve head myelination, we sectioned three eyes with myelinated nerve fibers of the retina and optic nerve head in a step-serial fashion in the coronal plane through the lamina cribrosa. For electron microscopy, tissues were dehydrated with ethyl alcohol, postfixed in osmium tetroxide, and embedded with Araldite. We cut ultrathin sections with an ultratome, stained them with uranyl acetate and lead, and examined them with an electron microscope.

Using gross diagnosis and appropriate histologic confirmation, we determined the prevalence of myelinated nerve fibers. For each significant gross feature we performed an analysis of variance, by eye and by lesion, to establish prevalence and range of gross morphologic characteristics.

Thirty-two patients with myelinated nerve fibers of the retina and optic nerve head in one or both eyes were examined and followed up for a maximum of 12 years. We obtained a detailed ophthalmic history and performed a complete ophthalmic examination, including biomicroscopic examination, tonometry, indirect ophthalmoscopy, and appropriate ocular fundus biomicroscopy, on every patient. Additionally, we took ocular fundus photographs of every patient and performed fluorescein angiography and visual field studies on most patients.

RESULTS

Autopsy series—Myelinated nerve fibers of the retina, identified grossly and confirmed microscopically whenever appropriate, were present in 39 of the 3,968 consecutive autopsy cases (0.98%). Myelinated nerve fibers were least common in the first decade of life, most common in the ninth decade, and distributed evenly throughout the other decades. The difference between the small incidence of myelinated nerve fibers in the first decade and the greater distribution thereafter was statistically significant (based on Fisher's exact test), but the differences in incidence in the second through ninth decades were not statistically significant.

In the autopsy series there were 2,201 males (56%) and 1,767 females (44%) (Fig. 1). Among the 39 cases with myelinated nerve fibers, there were 23 males (59%) and 16 females (41%). Therefore, the ratio of affected males to examined males was 23:2,201 or 1.0%; the ratio of affected females to examined females was 16:1,767 (0.90%). There was no statistically significant difference between the sexes in the development of myelinated retinal nerve fibers. Similarly, with 22 right eyes (52%) and 20 left eyes (48%) affected, there was no statistically significant preference for either side.

In all autopsy cases, both eyes were available for study. Of the 39 cases with myelinated nerve fibers of the retina, one eye was affected in 36 cases (92.3%) and both eyes were affected in three cases (7.7%). Thus, myelinated nerve fibers were present in 42 of the 7,936 eyes (0.54%) in this series.

Among the 42 eyes with myelinated fibers, 37 eyes (88%) contained a single lesion and five eyes (12%) contained two separate and distinct patches of myelinated nerve fibers. No eye contained more than two patches of myelinated nerve fibers. Consequently, a total of 47 lesions were available for study.

We characterized patches of myelinated retinal nerve fibers as continuous with the optic nerve head or as discontinuous (when an area of normal-appearing

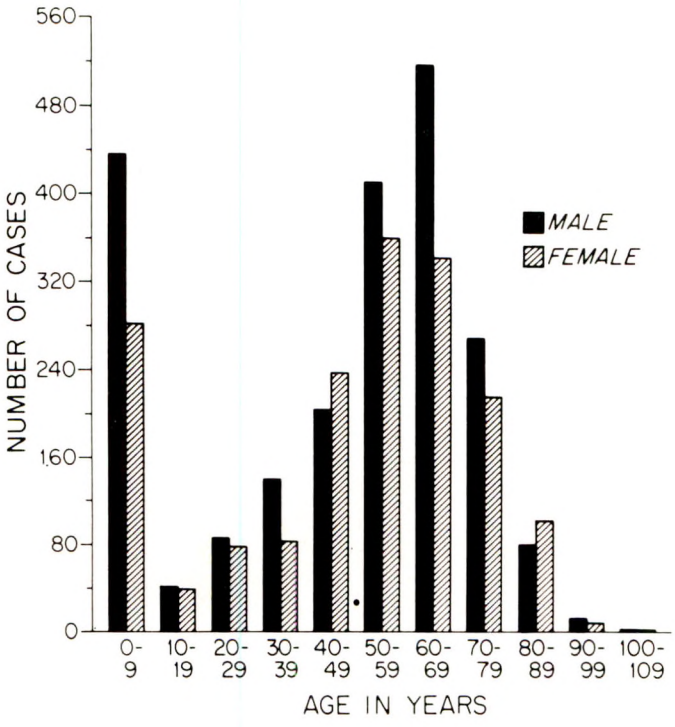


Fig. 1 (Straatsma and associates). Age and sex distribution of 3,968 consecutive autopsy cases. In all cases, both eyes were available. Therefore, 7,936 autopsy eyes were examined in this study.

retina separated the optic nerve head and the lesion). Continuous lesions were present in 14 eyes (33%) (Fig. 2), including both eyes of the three bilaterally affected cases in this series. Continuous lesions generally extended onto the optic nerve head, varied greatly in overall size, and differed markedly in the extent of nerve-head circumference involved. The inferior temporal and the inferior nasal sectors of the nerve head were most commonly affected, but there was no statistically significant difference among quadrants and all were involved.

Discontinuous lesions were present in 28 eyes (66%), including all of the five eyes in which there were two separate lesions. Discontinuous lesions were generally located posterior to the equator, differed greatly in size and other characteristics, and were present in all quadrants extending from the nerve-head border. With the nerve head as the central landmark, the involvement was most common in the inferior temporal quadrant, but there was no statistically



Fig. 2 (Straatsma and associates). Myelinated retinal nerve fibers continuous with optic nerve head in a 63-year-old man (× 12).

significant difference among quadrants and all were involved.

All lesions identified grossly as myelinated nerve fibers appeared as white or gray-white striated patches that corresponded in shape to the distribution of retinal nerve fibers, were sharply demarcated, and possessed frayed or feathered borders related to the various lengths of the individual myelin sheaths. Continuous lesions extended onto the optic nerve head and adjacent retina for various distances, generally appeared as dense white patches, were wedge-shaped to correspond with nerve fiber distribution, partially obscured underlying retinal vessels, and displayed frayed distal margins.

Discontinuous lesions were separated from the nerve head but were located posterior to the equator. They generally appeared as white or subtle gray-white, wedge-shaped patches with conspicuously frayed distal borders (Fig. 3). In some lesions there was subtle opacification of

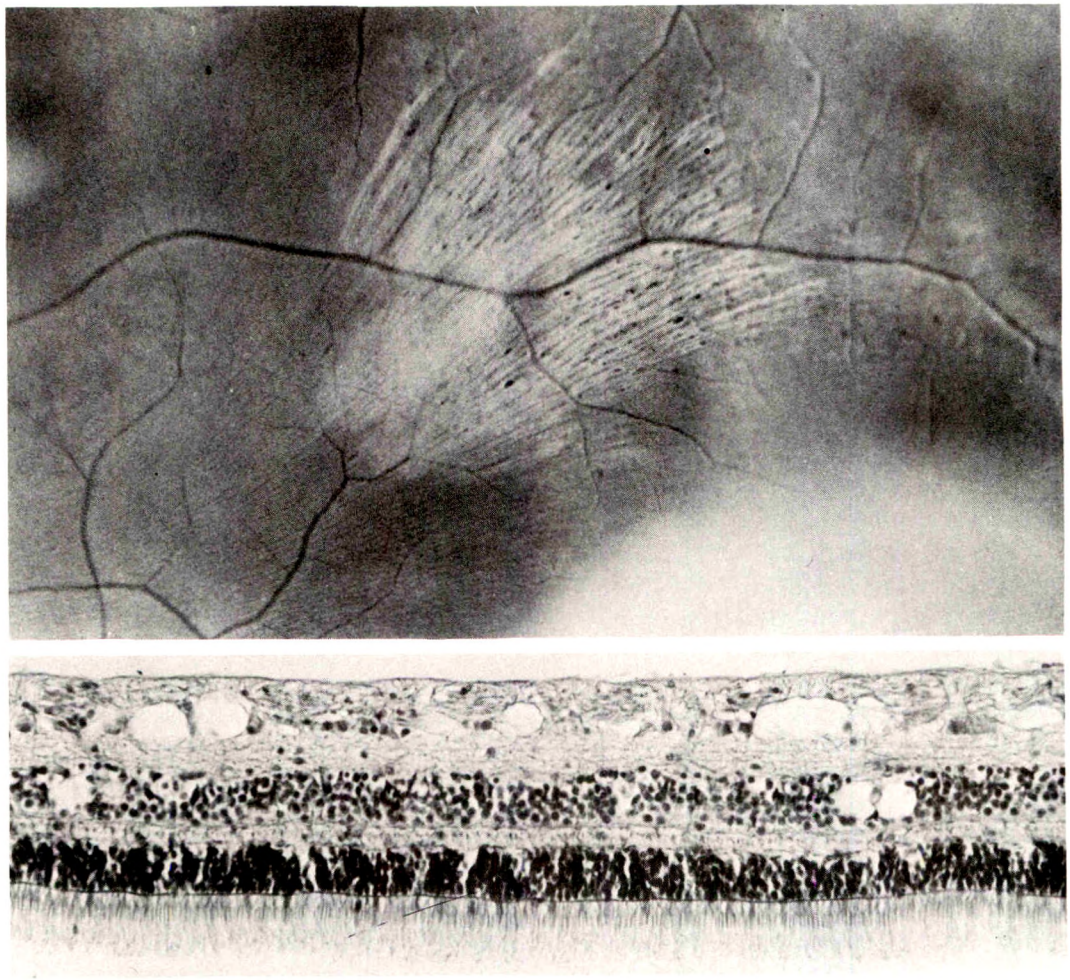


Fig. 3 (Straatsma and associates). Top, Myelinated nerve fibers of retina of a 74-year-old man are discontinuous with optic nerve head and are located temporal to macula and slightly inferior to horizontal raphe. Tiny cysts are scattered throughout the more peripheral portion of the patch (× 4). Bottom, Microscopic section through lesion shows a few microcysts and degenerating oligodendrocytes in the inner and middle layers of retina (hematoxylin and eosin, × 250).

the nerve fiber layer so that retinal vessels and choroidal features were not obscured. It was easy to overlook lesions when they were distal to the nerve head, where nerve fibers were less numerous. Occasionally, microcystoid degeneration of the inner retina was evident within the patches of myelinated nerve fibers, but there were no pigmentary, vascular, or other associated morphologic abnormalities.

Microscopically, the stained sections showed subtle lesions, displaying only increased eosinophilia and, in some instances, increased cellularity (Fig. 4). The cells in the latter lesions resembled oligodendroglia with dark round nuclei smaller than those of the accessory astrocytes normally present. With special stains, a variable proportion of the nerve fibers in a lesion stained positively for myelin. In occasional lesions, more ganglion cells were displaced into the inner nuclear layer than into adjacent uninvolved areas of the retina.

Special stains such as Luxol fast blue and Weil's stain demonstrated myelin most conspicuously. Interestingly, eyes with the continuous type of myelination did not demonstrate staining for myelin in the region of the lamina cribrosa (Fig. 4). Apart from the cylindrical myelin sheaths surrounding the axons and other features already described, we found no associated histologic abnormalities in the retina, the optic nerve head, or other ocular tissues.

We sectioned three eyes with continuous patches of myelinated nerve fibers involving the retina and optic nerve head in step-serial fashion in the coronal plane through the lamina cribrosa. These eyes showed an intact lamina cribrosa.

Correlative electron microscopy provided more detailed information regarding myelinated nerve fibers in two eyes. A typical discontinuous lesion showed characteristic myelin sheaths composed of concentrically laminated lipoprotein membranes encasing the axons (Fig. 5).

Clinical study—We reviewed the cases of 32 patients with myelinated nerve fibers of the retina. Although these physician-referred patients did not form a random sample, we studied them in order to correlate the study of eyes obtained at autopsy with the clinical characteristics of living patients and to clarify some of the varied clinical manifestations. Significantly, four patients, including one patient with leukocoria who had been referred because retinoblastoma was suspected, had ipsilateral extensive myelinated retinal nerve fibers, myopia, amblyopia, and strabismus.[13] The other 28 patients with myelinated nerve fibers had been referred for unrelated disorders. This series included one set of phenotypically identical twins with bilateral myelinated retinal nerve fibers.

There were 21 male patients and 11 female patients, ranging in age from 2 to 76 years at the initial examination (mean age, 49 years). The observation period ranged from a single consultation to 12 years.

We examined both eyes in all but one patient. Retinal myelination was bilateral in five of the 31 patients (17%) who had both eyes. The 37 affected eyes varied in refractive error from a spherical equivalent of −10.00 to +4.50.

Four patients (12%) with unilateral myelinated retinal nerve fibers had ipsilateral extensive myelination of retinal nerve fibers, anisometropic myopia, strabismus, and amblyopia that resulted in visual acuity of 6/60 (20/200) or worse, despite occlusion therapy. This syndrome is described in detail elsewhere.[13] In all other affected eyes, visual acuity was 6/9 (20/30) or better unless impaired by an unrelated disease.

Of the 37 affected eyes, 35 (94%) contained a single lesion. The lesion was

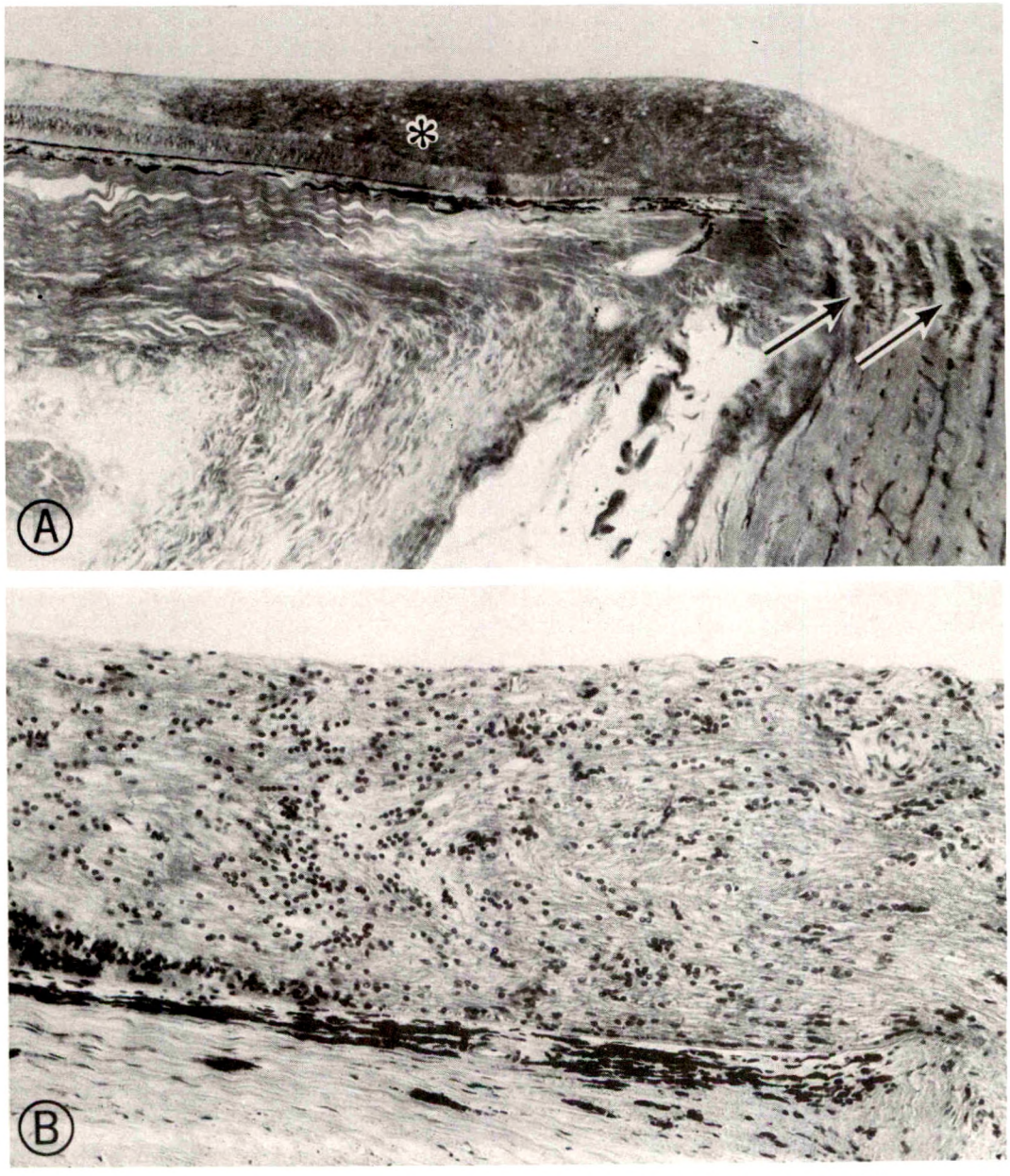


Fig. 4 (Straatsma and associates). Top, Microsection of optic nerve head and adjacent eye wall in an eye with continuous myelination. Nerve bundles transmigrating the lamina cribrosa (arrows) and within the optic nerve head are unstained, whereas those of the optic nerve and the superficial layers of parapapillary retina (asterisk) stain positively for myelin (Luxol fast blue, × 32). Bottom, Oligodendrocytes (small dark nuclei) within the patch of myelinated nerve fibers (hematoxylin and eosin, × 250).

continuous with the optic nerve head in 30 of the 37 eyes (81%), including both eyes of the five bilaterally affected patients in this series. Arising most commonly from the inferior or superior pole of the nerve head, the patches generally extended onto the nerve head, varied greatly in size, and possessed frayed distal margins (Fig. 6). Continuous lesions appeared as white striated patches

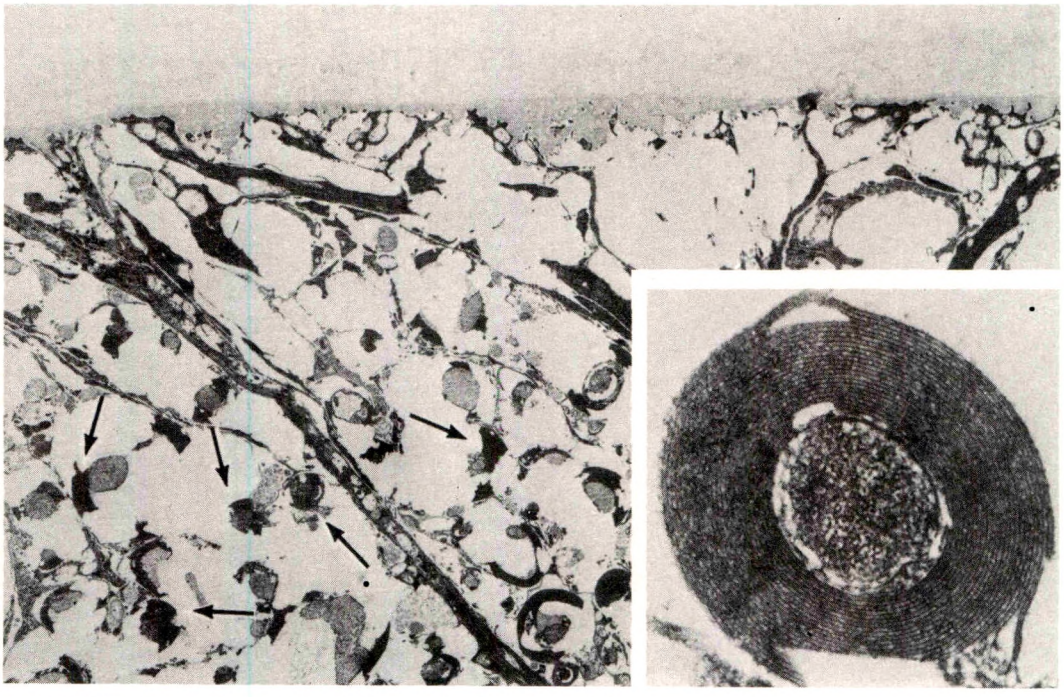


Fig. 5 (Straatsma and associates). Ultrastructural features of discontinuous patch of myelinated nerve fibers. Despite severe autolysis, characteristic myelin profiles are present in nerve fiber layer (arrows) (× 1,750). Inset, Detail of myelin sheath encasing axon (× 48,000).

that corresponded in shape to the distribution of retinal nerve fibers (Fig. 7). When bilateral, the continuous patches of myelinated nerve fibers usually were similar but somewhat asymmetrical. The identical twins had bilateral continuous patches of myelinated retinal nerve fibers. The myelination in each of the affected eyes of the twins was generally symmetrical in size and location but did not take the form of a mirror image.

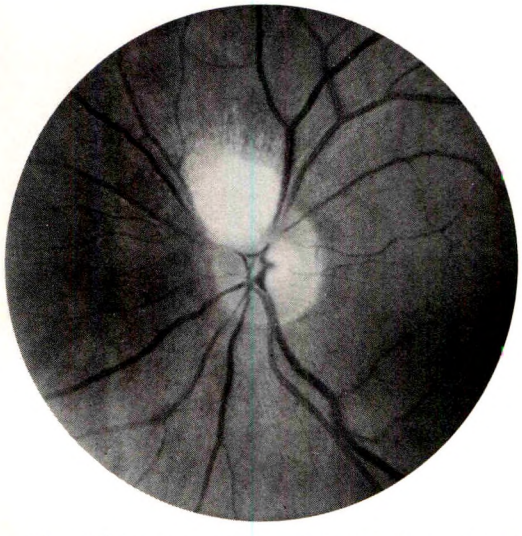


Fig. 6 (Straatsma and associates). White striated patch of continuous myelinated nerve fibers from the left eye of a 57-year-old woman.

There were discontinuous patches of myelinated nerve fibers, separated from the optic nerve head by an area of normal-appearing retina, in seven of the 37 eyes (19%). These lesions appeared as white or gray-white striated patches that corresponded in shape with the course of normal nerve fibers (Fig. 8). Located posterior to the equator and varying substantially in size, the discontinuous patches demonstrated frayed or feathered borders, particularly at the distal margin; these lesions were bilateral in one patient (Fig. 9). Additionally, we found microcystic degeneration in the inner layers of the retina in some discontinuous patches of myelinated retinal

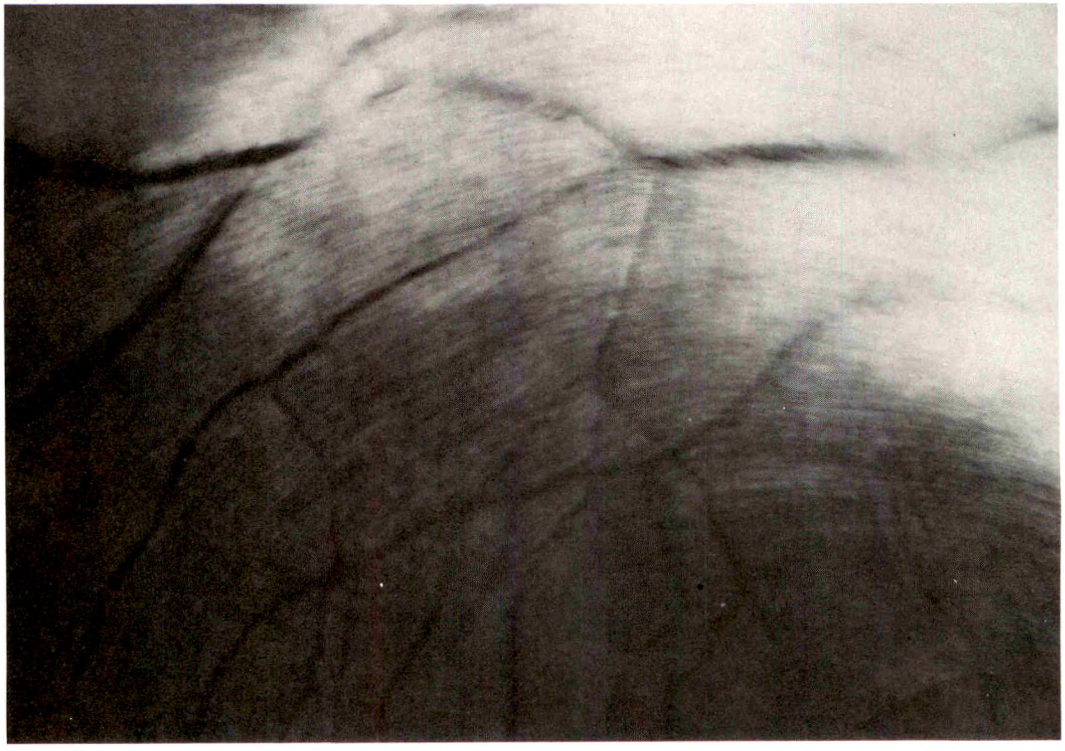


Fig. 7 (Straatsma and associates). Distal border of myelinated nerve fibers continuous with optic nerve head, superior to macula in the right eye of a 28-year-old man.

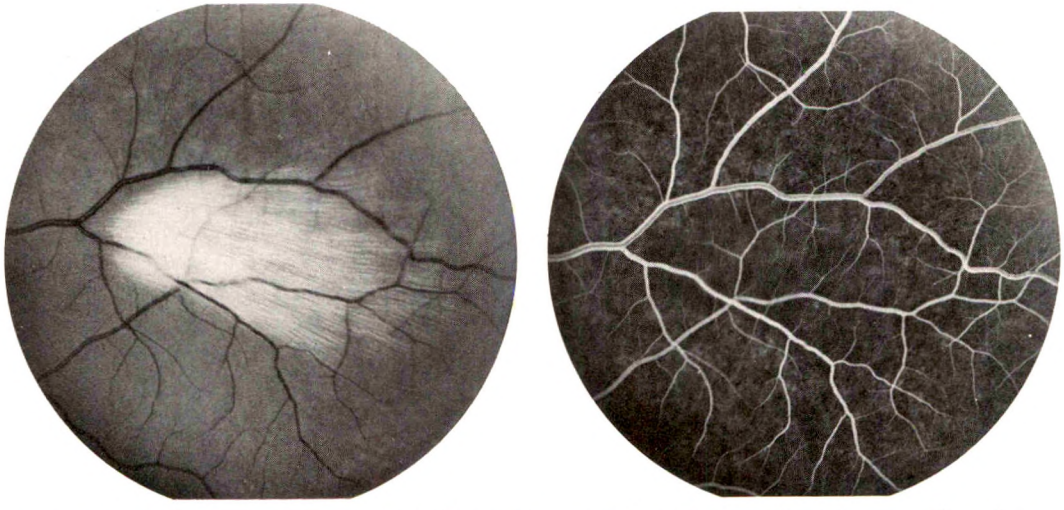


Fig. 8 (Straatsma and associates). Left, Gray-white striated patch of myelinated nerve fibers from a 37-year-old man are discontinuous with optic nerve head and show frayed distal border (photographed with red-free light). Goldmann visual field demonstrated an inferior nasal relative scotoma to I_3 isopters, corresponding to the lesion. Right, Fluorescein angiogram of same area (arteriovenous phase) shows some masking caused by opaque myelin sheaths, but no abnormality of circulation on pigment epithelium.

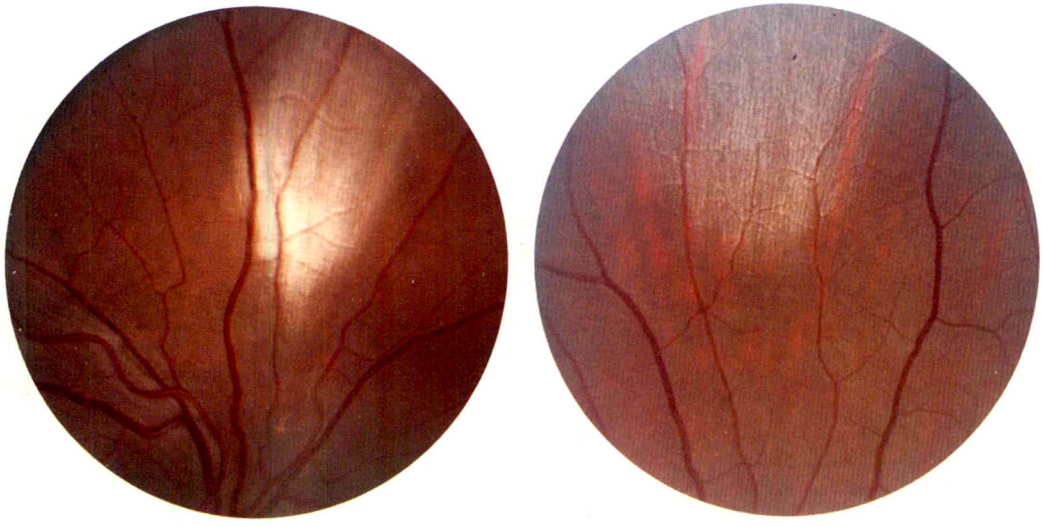


Fig. 9 (Straatsma and associates). Bilateral single patches of discontinuous myelinated nerve fibers, superior to optic nerve in a 27-year-old man (left, right eye; right, left eye).

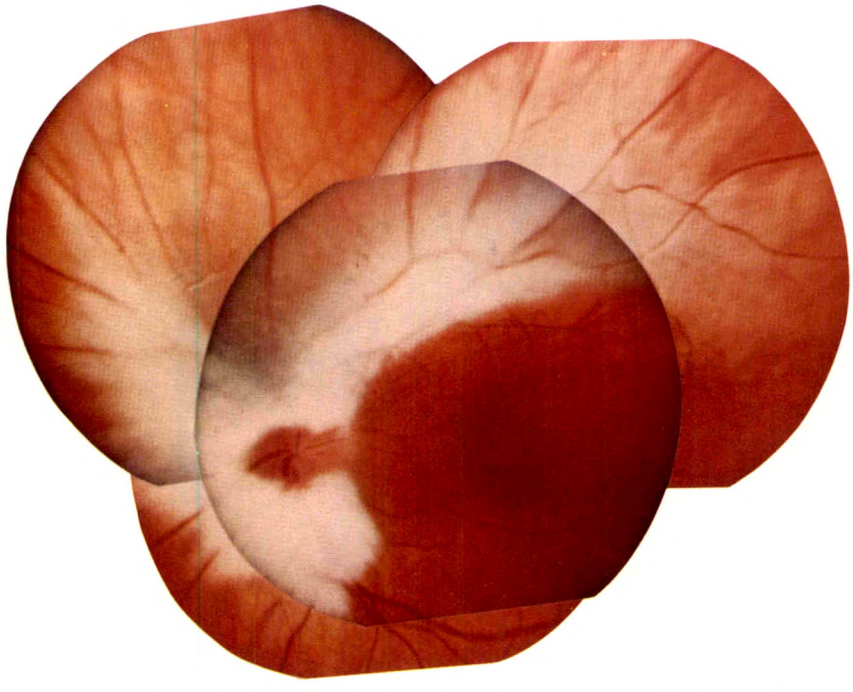


Fig. 10 (Straatsma and associates). Myelinated nerve fibers extend from full circumference of optic nerve head, arch temporal to macula, and extend in a superior direction almost to the equator. Associated findings of ipsilateral severe myopia, amblyopia, and strabismus in the left eye of an 11-year-old girl.

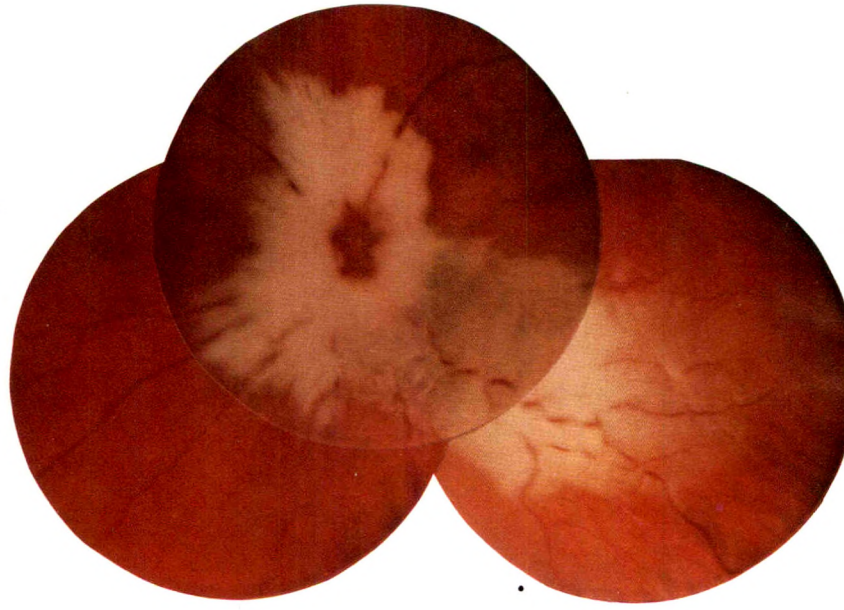


Fig. 11 (Straatsma and associates). Myelinated nerve fibers extend from full circumference of optic nerve head and reach approximately to equator in superotemporal quadrant. Associated findings of ipsilateral myopia, amblyopia, and strabismus in the right eye of a 50-year-old woman.

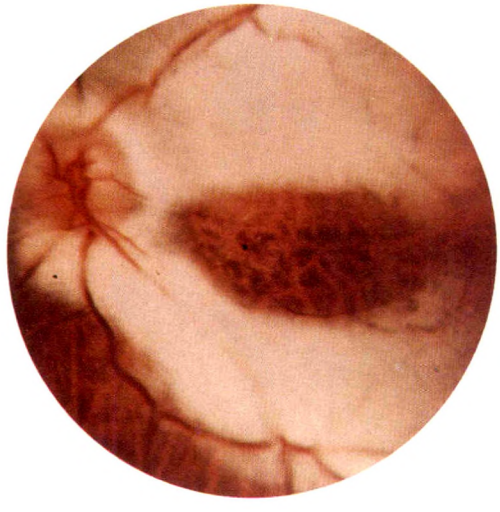


Fig. 12 (Straatsma and associates). Myelinated nerve fibers extend from entire circumference of optic nerve head and progress along course of retinal nerve fibers superior and inferior to fovea to reach horizontal raphae temporal to fovea. Associated findings of ipsilateral severe myopia, amblyopia, and strabismus in the left eye of a 7-year-old girl.

nerve fibers (Fig. 3). In general, the size, shape, and other characteristics of discontinuous lesions were especially evident with red-free light, which emphasized retinal structure and did not penetrate the choroid.

One patient, referred with leukocoria and possible retinoblastoma, had a large area of myelination. The myelinated fibers extended from the full circumference of the optic nerve head, in an arcuate direction temporal to the macula and in a superior direction almost to the equator (Fig. 10). Two other patients each had a large patch of myelinated nerve fibers (Figs. 11 and 12).

A visual field defect was usually present in association with patches of myelinated nerve fibers. This defect corresponded in general to the size and location of the myelinated nerve fiber patches; it ranged from a relative scotoma to an absolute defect corresponding to the myelinated patch. In general, however, the visual field defect was substantially less than would be expected from the extent of the myelinated nerve fibers seen ophthalmoscopically. This indicates more penetration of light to the retinal photoreceptors than expected from the clinical appearance.

Fluorescein angiography of eyes with myelinated nerve fibers demonstrated some masking of underlying structures by the opaque nerve sheaths, but there was no discernible abnormality of retinal circulation, choroidal circulation, or retinal pigment epithelium. Moreover, the myelinated areas appeared less conspicuous (Fig. 8) and the intrinsic retinal circulation was seen more completely with angiography than with white-light ophthalmoscopic examination.

Fundus photographs and complete ophthalmic examinations demonstrated that the myelinated nerve fibers were unchanged in all patients throughout the period of observation.

Discussion

Clinicopathologic correlations — The autopsy study demonstrated myelinated retinal nerve fibers in 0.98% of the cases, equal involvement of males and females, bilateral myelin patches in 7.7% of the cases, myelinated nerve fibers continuous with the optic nerve head in 33% of the eyes, and myelinated nerve fibers discontinuous with the optic nerve head in 66% of the eyes. All lesions appeared as white or gray-white striated patches that corresponded in shape to the distribution of retinal nerve fibers and demonstrated frayed borders related to the various lengths of the myelin sheaths. The sensory retina, optic nerve head, pigment epithelium, and choroid were morphologically normal by light and electron microscopy, except for myelin sheaths composed of concentric lipoprotein lamellae surrounding retinal nerve fibers, glial cells photomicrographically resembling oligodendrocytes adjacent to the myelinated sheaths, and microcystoid retinal degeneration associated with some lesions.

Clinical observations of patients with myelinated retinal nerve fibers demonstrated a comparable incidence and similar overall gross features, except for the higher percentage of males in the clinical series, bilateral myelin patches in 17% of the patients, myelinated nerve fibers continuous with the optic nerve head in 81% of the eyes, and myelinated nerve fibers discontinuous with the optic nerve head in 19% of the eyes. The higher percentage of lesions continuous with the optic nerve head in the clinical series suggests that continuous patches of myelinated nerve fibers are more apt to be observed and diagnosed correctly in clin-

ical practice. Stated another way, discontinuous patches of myelinated nerve fibers are more apt to be overlooked on clinical examination.

In patients with myelinated retinal fibers, visual field defects were substantially smaller and less dense than anticipated, and results of fluorescein angiography were normal except for obscuration related to the opaque myelin sheaths. These observations corroborate the histopathologic evidence, and indicate the general functional integrity of the retina, as well as more penetration of light to the retinal photoreceptor layer than might be expected from the clinical appearance of the myelinated nerve fibers.

Associated findings—Clinical observations suggested a syndrome characterized by ipsilateral extensive myelinated retinal nerve fibers, anisometropic myopia, amblyopia, and strabismus. This constellation of findings, present in four of the 32 patients in this series, was described in a separate report.[13] Holland and Anderson[15] reported comparable findings in two patients and other investigators have described the association of widespread myelinated retinal nerve fibers with abnormalities such as myopia, amblyopia, nystagmus, various types of strabismus, and afferent pupillary defect.[2,13,15-20] It is important to recognize this syndrome because early diagnosis makes possible correction of the unilateral refractive error and the institution of carefully monitored amblyopia therapy and appropriate strabismus management.[13]

More severe ocular and general abnormalities reported with myelinated retinal nerve fibers include coloboma, polycoria, keratoconus, oxycephalia, and other forms of dyscrania.[16,21] Although they are uncommon, these associated abnormalities must be considered.

The cause of retinal nerve fiber myelination—The cause is not known, but two significant factors are evident as a result of this clinicopathologic study. First, step-serial sections of the optic nerve head and adjacent retina in three eyes with continuous patches of myelinated retinal nerves disclosed no abnormalities of the cribiform plate and no continuous extension of optic nerve myelination through the cribiform plate into the optic nerve head and retina. Therefore, myelination of fibers in the optic nerve head and retina is separate from and not directly joined with myelination of fibers in the optic nerve. Second, light microscopy of myelinated nerve fiber patches in the retina and optic nerve head demonstrated an associated clustering of glial cells which appeared to be oligodendrocytes. Tissue autolysis interfered with precise identification of oligodendrocytes by electron microscopy. Oligodendrocytes, believed to be the cells responsible for myelination of the optic nerve and other central nervous system structures, may be found in the optic nerve head but are not normally present in the retina. Thus, myelination of nerve fibers in the retina and adjacent segments of the optic nerve head is likely to be related to the anomalous location of oligodendrocytes (or glial cells resembling oligodendrocytes) in the sensory retina. This anomalous location of oligodendrocytes in the retina is presumably responsible for the development of myelin sheaths around retinal nerve fibers.

The clinical appearance of myelinated retinal fibers, observed in three eyes of this group with sequential photographs over a 12-year period, is remarkably stable. Many reports have documented the unchanging appearance of myelinated retinal fibers in the adult eye. Some patients, however, develop a microcystoid degeneration of the nerve fiber layer in the area of the myelinated fibers. With nerve fiber destruction and optic nerve atrophy, myelination may slowly disappear over a period of one or more years.

Loss of myelination in the retina has been observed in association with optic nerve atrophy, multiple sclerosis, pituitary tumor, and embolism of the central retinal artery.[2]

Differential diagnosis—The differential diagnosis of myelinated retinal nerve fibers is seldom difficult. With extensive involvement, however, myelinated nerve fibers may produce leukocoria and superficially resemble retinoblastoma, as happened in one patient in this series who was referred with the diagnosis of retinoblastoma and in other published reports. A smaller patch of myelinated retinal nerve fibers may be confused with a cotton-wool spot, retinal artery branch occlusion, peripapillary epiretinal membrane, retinal pigment epithelium detachment, or other abnormality responsible for a localized gray or white retinal lesion. Careful examination augmented, when indicated, by fluorescein angiography provides the basis for correct diagnosis of all these conditions.

Summary

In a series of 3,968 consecutive autopsies, myelinated nerve fibers of the retina were present in 39 (0.98%) cases and bilateral in three (7.7%) affected cases; thus, 42 (0.54%) of the 7,936 eyes examined were affected.

Myelinated nerve fiber lesions appeared as white or gray-white striated patches corresponding in shape to the distribution of retinal nerve fibers and demonstrated frayed borders. Myelinated retinal nerve fibers were continuous with the optic nerve head in 14 (33%) and discontinuous with the optic nerve head in 28 (66%) affected eyes. By light microscopy and electron microscopy, myelinated retinal fibers were marked by a ganglion cell axon surrounded by concentric lipoprotein lamellae that formed the myelin sheath. Glial cells were often prominent near the myelin sheaths, but other components of the sensory retina were morphologically normal.

Clinically, 32 patients with myelinated retinal nerve fibers had comparable overall features, visual field defects less extensive than expected on the basis of ophthalmoscopic appearance, and normal findings on fluorescein angiography. Four patients had a syndrome characterized by ipsilateral extensive myelinated retinal nerve fibers, anisometropic myopia, amblyopia, and strabismus.

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OPHTHALMIC MINIATURE

As I drove back toward town I was thinking about that ancient and honorable bit of homely psychology, that myth of the ripeness of cataracts. The lens capsule can be removed as soon as it begins to get cloudy. But postoperative vision with corrective lenses is a poor resource at best, compared with normal sight. So the ripeness they speak of is the psychological ripeness of the patient, a time of diminishing vision which lasts long enough, and gets bad enough, so that the postoperative vision is, by comparison, a wonder and a delight. The patient is happy because the basis of comparison has changed.

John D. MacDonald, *The Long Lavender Look*
Greenwich, Connecticut, Fawcett, 1970

FUNCTIONAL AMBLYOPIA ASSOCIATED WITH ORGANIC OCULAR DISEASE

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Functional amblyopia has been described by von Noorden[1] as "reduced visual acuity without ophthalmoscopically detectable anomalies of the fundus." He cited estimates that subclinical organic changes account for part of the visual loss in 10% of all strabismus patients with amblyopia. He used this concept to explain plateaus in visual acuity in individuals with amblyopia after initial improvement with occlusion therapy. I undertook this study to test the possibility that functional amblyopia can be either coincidentally or causally coexistent with overt organic abnormalities.

SUBJECTS AND METHODS

This series consists of 13 patients I examined during an 18-month period. They all met the study criteria of visual loss, associated overt ocular abnormality, and improvement in visual acuity with standard amblyopia therapy. Visual acuity measurements were all performed at 6 m with line Snellen[2] letters, isolated Snellen letters, linear illiterate E, isolated illiterate E, or the Sheridan-Gardiner[3] isolated-letter-matching technique. For children too young to take these visual acuity tests, I assessed visual acuity by subjective interpretation of fixation ability. I described visual acuity as good if the ability to fixate and follow an object was present, as central if fixation was foveal as opposed to eccentric, and as maintained if that eye maintained fixation after a cover-uncover test on the fellow eye. I tested neutral density filter acuities with a Kodak Wratten No. 96 neutral density filter 2.50. Stereopsis was tested with the Titmus stereopsis test. All refractions were done with either one dose of 2% or two doses of 1% cyclopentolate. Full-time occlusion therapy refers to occlusion of the nonamblyopic eye immediately upon arising in the morning until just before retiring in the evening. Visual function in both eyes of children 4 years of age or older undergoing full-time occlusion therapy was assessed every four weeks. Children younger than 4 years of age were seen at intervals of one week per year of age. When amblyopia was overcome, the occlusion was gradually decreased in frequency before being discontinued. Family history was noncontributory for strabismus or amblyopia in all patients in this series.

CASE REPORTS

Case 1—A 5-year-old boy had a visual acuity of R.E.: 6/6 (20/20) and L.E.: 6/60 (20/200) with line Snellen letters. Visual acuity in his left eye was 6/18 (20/60) with isolated Snellen letters and 6/30 (20/100) with a neutral density filter. Refraction was +1.00 +0.25 ×180 for both eyes. Visual acuity did not improve with trial lenses. The anterior segments and pupillary responses were normal. There was a chorioretinal scar near the macula in the left eye (Fig. 1). After four months of full-time occlusion of the right eye, the visual acuity in the left eye improved to 6/7.5 (20/25) and has been stable for three years. Stereopsis was 60 seconds.

Case 2—A 26-month-old girl had a six-week history of esotropia in the left eye. Her visual acuity was good, central, and maintained in the right eye, but not maintained in the left eye. The child protested occlusion of her right eye during the examination. The esotropia in the left eye measured 20 prism diopters at distance and 60 prism diopters at near. There was a chorioretinal scar near the macula of the

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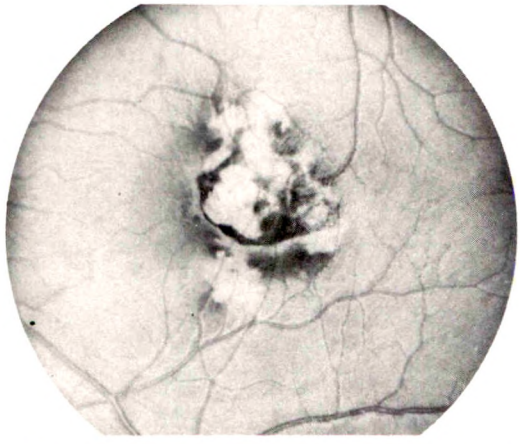


Fig. 1 (Kushner). Case 1, left eye. Chorioretinal scar involving macular area.

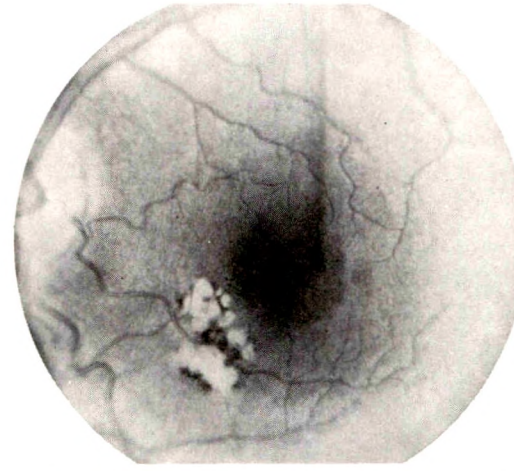


Fig. 2 (Kushner). Case 2, left eye. Perimacular chorioretinal scar.

left eye (Fig. 2). Refraction was +1.50 +0.25 × 90 in the right eye and +1.75 sphere in the left eye. I prescribed spectacles with a +2.75 bifocal addition, and instituted full-time occlusion of the right eye. After three months she alternated fixation freely so patching was discontinued. She was last examined at the age of 40 months. At that time, her visual acuity was 6/6 (20/20) in both eyes with the Sheridan-Gardiner isolated-letter-matching technique. She had orthophoria at distance and near with spectacles. Fusion was present with the Bagolini striated lenses and Worth Four-Dot at distance and near. There was no stereopsis. Without spectacles, she had a freely alternating esotropia of 15 prism diopters at distance and 35 prism diopters at near.

Case 3—A 4-year-old boy was examined because of esotropia in the left eye. He had been born prematurely (birthweight, 1,328 g) and had received oxygen for neonatal respiratory distress. At examination he was found to have esotropia of 30 prism diopters at distance and near in the left eye. Refraction was +0.50 sphere in the right eye and −5.75 +1.00 ×140 in the left eye. Best corrected visual acuity was 6/7.5 (20/25) in the right eye and 6/60 (20/200) in the left eye with linear Snellen letters. Visual acuity in the left eye improved to 6/24 (20/80) with isolated Snellen letters. The right fundus had a normal posterior pole, but there were peripheral vitreous veils and pigmentary changes. The left posterior pole showed Grade II cicatricial retinopathy of prematurity by Reese, King, and Owens' classification[4] (Fig. 3). I prescribed spectacles and began full-time occlusion for the right eye. Four months later visual acuity in his left eye was 6/9 (20/30). He underwent a 5-mm recession of the left medial rectus muscle and a 6-mm resection of the left lateral rectus muscle. Two years later visual acuity had been maintained at 6/9 (20/30) on part-time occlusion of the right eye. There was esotropia of 5 prism diopters in the left eye.

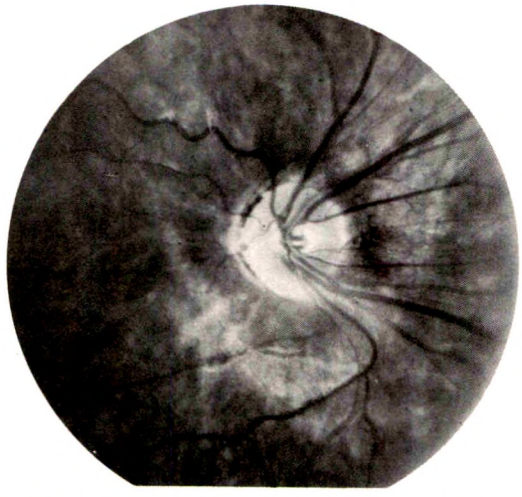


Fig. 3 (Kushner). Case 3, left eye. Posterior pole shows Grade II cicatricial retinopathy of prematurity.

Case 4—A 3-year-old boy was examined because of leukocoria in the left eye. Motility, pupils, and anterior segments were normal. Refraction was +0.75 sphere in the right eye and −12.50 +2.75 ×170 in the left eye. Best corrected visual acuity was 6/6 (20/20) in the right eye and 6/60 (20/200) in the left eye with the Sheridan-Gardiner isolated-letter-matching technique. Extensive medullated nerve fibers were visible in the left retina (Fig. 4). I prescribed spectacles and instituted full-time occlusion of the right eye. After six months the visual acuity in the left eye had improved to 6/12 (20/40) with the linear illiterate E test and has remained stable with part-time occlusion for 16 months.

Case 5—A 3⅓-year-old girl was examined because of amblyopia in her right eye. Poor compliance had

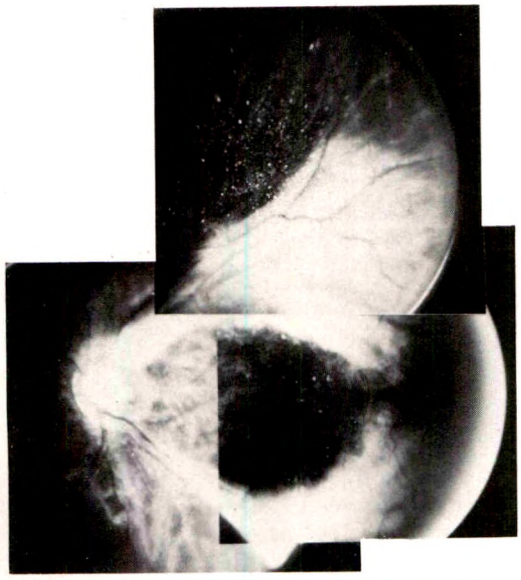


Fig. 4 (Kushner). Case 4, left eye. Posterior pole shows extensive medullated nerve fibers.

made an attempt at patching two years earlier unsuccessful. Refraction was +3.00 sphere in the right eye and +2.00 sphere in the left eye. Best corrected visual acuity was 6/60 (20/200) in the right eye and 6/6 (20/20) in the left eye with the Sheridan-Gardiner isolated-letter-matching test. There was a residual esotropia of 12 prism diopters in her right eye at distance and near with spectacles. Ophthalmoscopy showed a white condensation of the posterior hyaloid near the macula (Fig. 5). Initially, compliance with occlusion of the left eye was poor, but six months later her visual acuity was 6/9 (20/30) in both eyes with an isolated picture card test.

Case 6—I examined a 5-year-old girl after she failed a vision-screening test. Refraction was +2.50 sphere in the right eye and +2.50 +0.50 ×180 in the left eye. Best corrected visual acuity was 6/9 (20/30) in the right eye and 6/30 (20/100) in the left eye with linear Snellen letters. Visual acuity in the left eye was 6/18 (20/60) with isolated Snellen letters. There was posterior lenticonus in the left eye (Fig. 6). Sensory testing demonstrated suppression of the left eye with the Worth Four-Dot test and there was no stereopsis. I began full-time occlusion of the right eye with continuous cycloplegia of the left eye, spectacles, and a +2.50 bifocal addition. Visual acuity improved to 6/12 (20/40) after three months of full-time occlusion and has been stable for two years with part-time patching. Cycloplegia and bifocals were discontinued after one year. Stereopsis is 100 seconds.

Case 7—A 3-month-old girl was examined because of leukocoria in her left eye. There was an anterior polar lens opacity in the left eye (Fig. 7). When she was 29 months old, amblyopia was diagnosed in the left eye because she protested occlusion

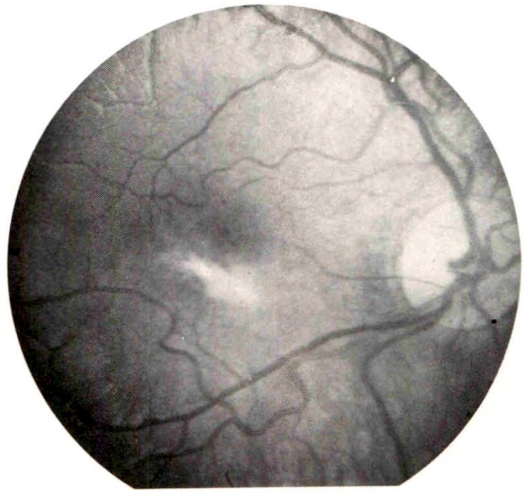


Fig. 5 (Kushner). Case 5, right eye. Posterior vitreous condensation overlying macular area.

of the right eye during an examination. Refraction was −3.00 sphere in each eye. Daily mydriasis of the left eye and full-time occlusion of the right eye were instituted. After two months the occlusion therapy was gradually decreased. When she was last examined at the age of 4½ years, her visual acuity was 6/9 (20/30) in both eyes with the linear illiterate E. She was on a maintenance program of occlusion of the right eye for two hours a day. Stereopsis was 100 seconds.

Case 8—A 4½-year-old girl had a marked persistence of the pupillary membrane in both eyes. In the right eye, it obscured a clear view of the fundus (Fig. 8). Best corrected visual acuity before dilatation of the pupils was R.E.: 6/60 (20/200) and L.E.: 6/9 (20/30) with linear Snellen letters. Visual

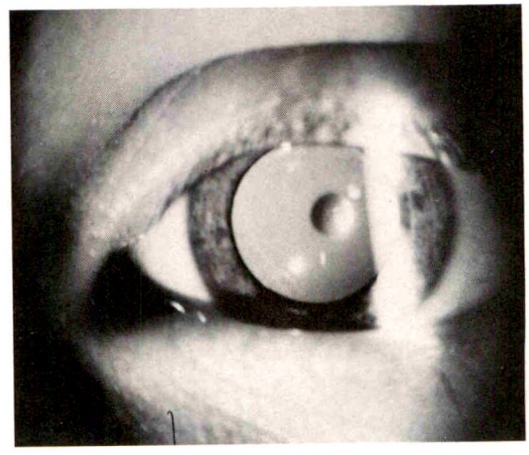


Fig. 6 (Kushner). Case 6, left eye. Red-reflex photograph demonstrates posterior lenticonus.

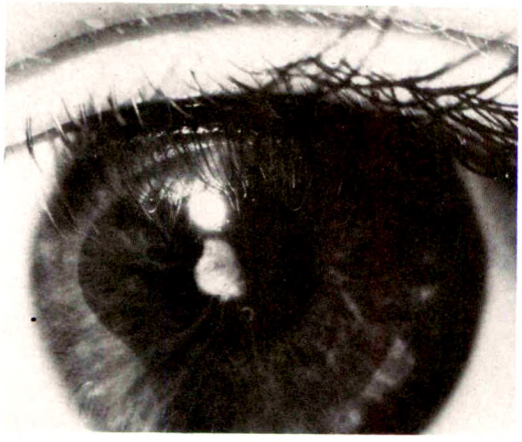


Fig. 7 (Kushner). Case 7, left eye. Anterior polar cataract.

acuity did not improve with isolated-letter testing. After mydriasis, the right fundus could be seen more clearly. Refraction was +3.50 +2.00 ×90 in the right eye and +1.25 +2.25 ×90 in the left eye. After mydriasis the visual acuity in the right eye could be corrected to 6/24 (20/80) with linear Snellen letters and 6/12 (20/40) with isolated Snellen letters. I believed the improvement in visual acuity corresponded to the increased clarity of the media after dilatation of the pupils. I prescribed spectacles with a +2.50 bifocal addition and instituted daily cycloplegic therapy. Visual acuity improved to 6/7.5 (20/25) in the right eye. When she was 6½ years old, cycloplegic therapy and bifocals were discontinued; eight months later her visual acuity was still 6/7.5 (20/25) in the right eye. Stereopsis was 100 seconds, and fusion was present with the Worth Four-Dot test.

Case 9—A 3½-year-old boy had sustained a traumatic hyphema in his right eye which cleared after bedrest and bilateral patching. He was later referred to me for evaluation of a posterior subcapsular cataract in the right eye and decreased visual acuity. When I first saw him, when he was 5 years old, his visual acuity was 6/30 (20/100) in the right eye with linear Snellen letters, and 6/15 (20/50) with isolated Snellen letters. With a neutral density filter or through a pinhole the visual acuity was 6/30 (20/100) in the right eye. Visual acuity in the left eye was 6/6 (20/20). Refraction was +0.75 +0.25 ×80 in the right eye and +1.00 sphere in the left eye. Visual acuity could not be improved with trial lenses. There was orthophoria. Full-time occlusion of the left eye was begun. After three months of full-time occlusion

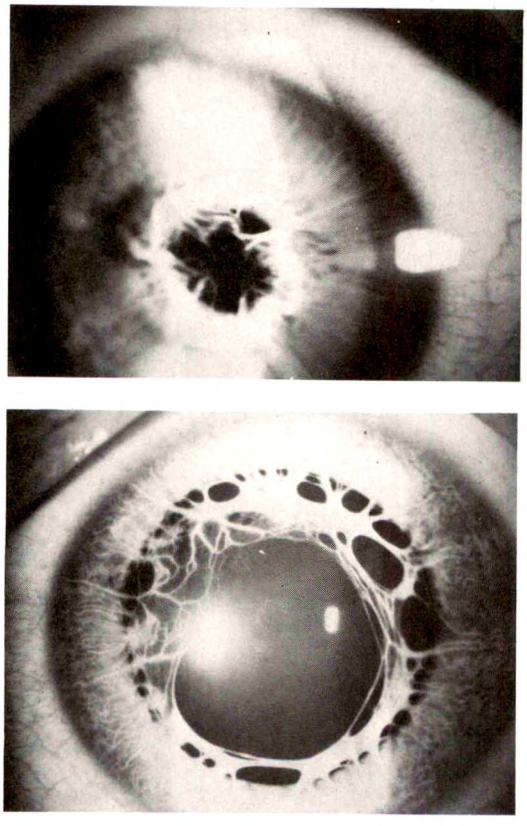


Fig. 8 (Kushner). Case 8, Marked persistence of pupillary membrane with network of fibrous tissue on anterior lens surface. Top left, Slit-lamp photograph of right eye before dilation. Red reflex is absent. Top right, Right eye after dilation. Bottom left, Left eye after dilation.

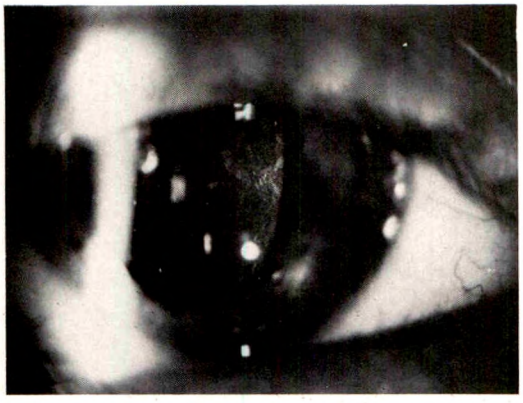


Fig. 9 (Kushner). Case 9, right eye. Posterior subcapsular cataract shown by retroillumination. Note that there is iridodialysis nasally.

the visual acuity in the right eye was 6/12 (20/40). Visual acuity has remained stable for 18 months after patching was discontinued (Fig. 9). •

Case 10—I examined a 6¼-year-old boy because of decreased visual acuity in his right eye. Refraction was −1.00 +1.25 ×80 in the right eye and +.50 +1.50 ×90 in the left eye. Best corrected visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/6 (20/20) with the linear illiterate E. Visual acuity in the right eye was 6/18 (20/60) with the isolated illiterate E and 6/30 (20/100) with a neutral density filter. A coloboma of the iris and choroid was present in the right eye (Fig. 10). Spectacles were ordered and full-time occlusion of the left eye was instituted. For the past three years he has been on a program varying between full and part-time occlusion of the left eye and has maintained a visual acuity of approximately 6/12 (20/40) in the right eye with the linear illiterate E. There is 200 seconds of stereopsis. Fusion on the Worth Four-Dot test is present at distance and near.

Case 11—I examined a 4-year-old girl because of congenital esotropia in the left eye. Family history was strongly contributory for cutaneous albinism on the maternal side. Refraction was +3.50 sphere in both eyes. Visual acuity was R.E.: 6/18 (20/60) and L.E.: 6/60 (20/200) with the linear illiterate E. Visual acuity in the left eye was 6/24 (20/80) with the isolated E. Esotropia of 35 prism diopters was present at distance and near in the left eye and there was an A pattern. There was no nystagmus. She had a very light complexion and white hair. Her peripheral iris had increased transillumination. There was a marked absence of choroidal pigmentation throughout the fundus in each eye; the macular area, however, was

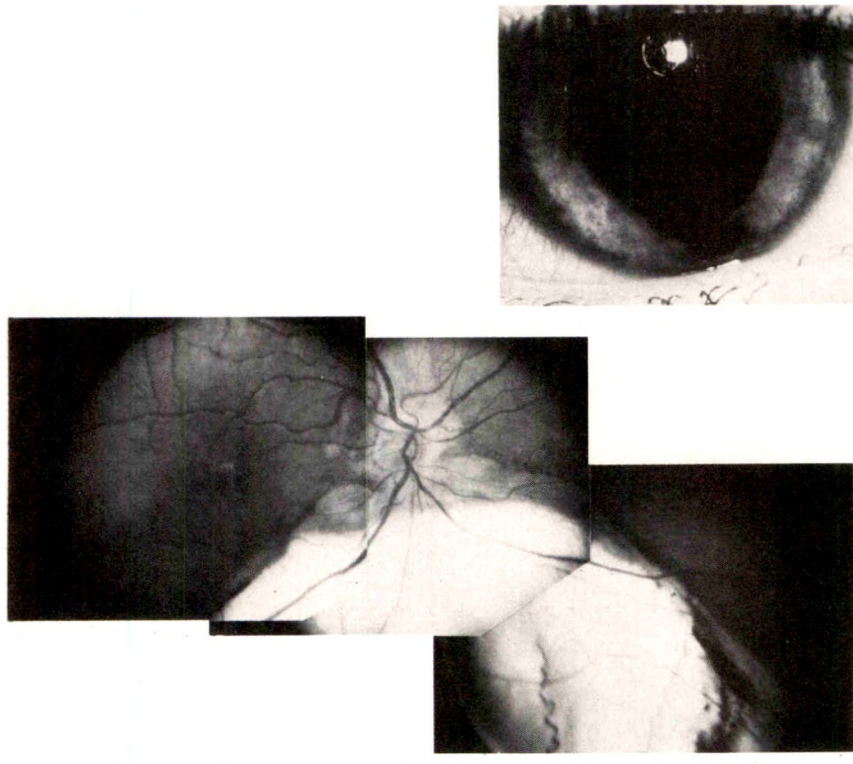


Fig. 10 (Kushner). Case 10, right eye. Extensive coloboma of choroid inferiorly right eye (at bottom) with coloboma of iris (at top).

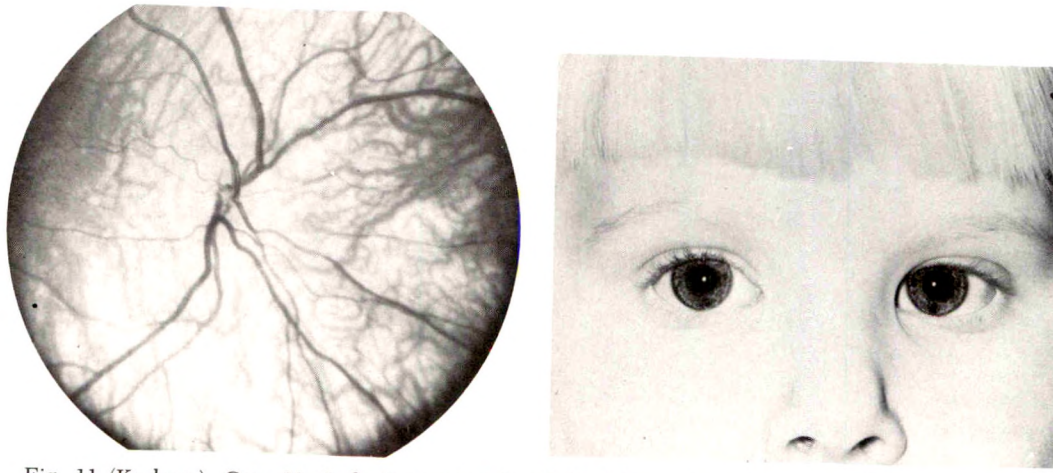


Fig. 11 (Kushner). Case 11. Left, Posterior pole of the left eye demonstrates marked prominence of choroidal vasculature (albinotic). Right, Albinotic facial appearance.

normal (Fig. 11). I prescribed spectacles and instituted full-time occlusion of the left eye. After three months the occlusion therapy was decreased, and it was discontinued after six months. When she was last examined at the age of 5 years, the child's visual acuity was 6/18 (20/60) in both eyes. There was esotropia of 5 prism diopters in the left eye while she was wearing her spectacles.

Case 12—I examined an 8-month-old boy because of esotropia of four months' duration in the right eye. At examination, I found esotropia of 35 prism diopters. I diagnosed amblyopia in the right eye on the basis of unsteady eccentric fixation in the right eye and the child's protest at occlusion of the left eye. A patent hyaloid artery containing circulating blood extended from the right optic nerve to the posterior surface of the lens, and there was a posterior subcapsular cataract nasally (Fig. 12). Refraction was $-1.50 +1.00 \times 70$ in the right eye and $+0.50$ sphere in the left eye. I prescribed full-time occlusion of the left eye and spectacles. After six weeks the occlusion therapy was decreased; it was discontinued after 4½ months. When the child was 14 months old, I performed a 5-mm recession of the right medial rectus muscle and a 7-mm resection of the right lateral rectus muscle. When he was last examined at the age of 4⅓ years, the child's visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/6 (20/20). There was esotropia of 8 prism diopters in the right eye.

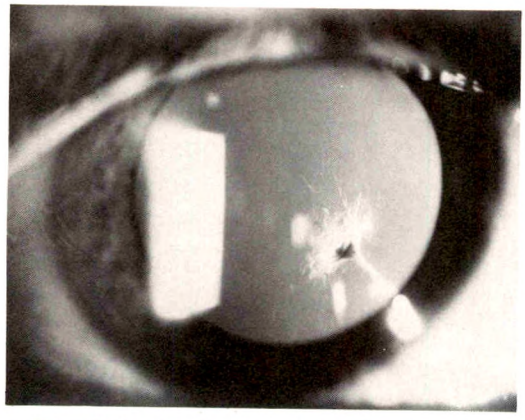


Fig. 12 (Kushner). Case 12, right eye. Patent hyaloid artery extends up to posterior cataract.

Case 13—I examined a 5-year-old girl because of esotropia in her right eye. Eighteen months previously she had undergone surgery for an adenocarcinoma of the choroid plexus. Visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/12 (20/40). Refraction was $+2.75$ sphere in the right eye and $+2.50$ sphere in the left eye. There was homonymous hemianopia in the right eye. Because of the patient's age, quantitative perimetry could not be performed and the presence or absence of macular sparing could not be determined. The pupils and optic disks were normal. There was esotropia of 25 prism diopters at distance and 45 prism diopters at near in the right eye. Spectacles with a $+2.75$ bifocal addition were ordered and full-time occlusion of the right eye was instituted. Five months later, her visual acuity had improved to 6/15 (20/50) in the right eye and patching was discontinued. Visual acuity has remained stable for eight months.

Discussion

Stuart and Burian[5] described separation difficulty (crowding phenomenon) as a universal phenomenon that increases inversely with visual acuity. Cibis, Hurtt, and Rasicovici[6] compared separation difficulty in organic and functional amblyopia. They found no significant improve-

ment in visual acuity with isolated-letter testing in patients with organic amblyopia secondary to retinal problems but significant improvement in patients with amblyopia caused by strabismus or anisometropia. Von Noorden and Burian[7] found a neutral density filter helpful in separating functional from organic amblyopia. Patients with decreased visual acuity caused by organic lesions of the retina or optic nerve had a marked decrease in visual acuity when tested with a neutral density filter, whereas patients with functional amblyopia showed a minimal decrease in visual acuity or, at times, even an improvement. In my experience with patients with organic ocular abnormalities, eyes showing separation difficulty (characterized by visual acuity at least two lines better with isolated-letter testing than with linear-letter testing) always showed improvement in visual acuity if there was an adequate clinical trial of full-time occlusion of the contralateral eye. Similarly, eyes that did not show a decrease in visual acuity when tested with a neutral density filter were always successfully treated when adequate amblyopia therapy was carried out. Both of these tests appear to be useful in determining when there is an element of functional amblyopia in patients with organic ocular abnormality.

A refractive error that decreases visual acuity slightly may result in subsequent functional amblyopia, producing a visual acuity much worse than would be expected from the optical blur alone. Mild organic causes of optical blur may similarly induce functional amblyopia. This concept was first described by Bangerter,[8] who reported functional amblyopia superimposed on organic amblyopia in patients with macular abnormalities or congenital cataracts. Organic problems may be coincidental with functional amblyopia, caused by either anisometropia or strabismus. The clinician must be cautious not to assume that an amblyopic eye cannot be rehabilitated because of the coincidental presence of organic abnormality. Unless the organic abnormality unequivocally accounts for the visual loss, further investigation is needed to determine whether there is an element of functional amblyopia.

Summary

Thirteen patients who had functional amblyopia associated either causally or coincidentally with organic ocular abnormalities still responded to conventional amblyopia therapy. The presence of separation difficulty or improvement in visual acuity with a neutral density filter helps predict the presence of functional amblyopia in patients with organic abnormalities.

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BLOOD GROUP ANTIGENS ON HUMAN CORNEAL CELLS DEMONSTRATED BY IMMUNOPEROXIDASE STAINING

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The human cornea is an antigenic tissue; when used as an allograft and transplanted from one individual to another, the graft can elicit an immune response.[1-3] The various antigens present on the cornea and potentially capable of stimulating an allograft immune reaction have not been precisely defined. It seems reasonable to predict that the cells of the cornea, being derived from the primary embryonic germ layers, will express the antigens displayed by other somatic cells derived from these same germ layers. It has been shown that corneal cells express the ubiquitous HLA (histocompatibility) antigens,[4,5] which, along with the blood group antigens, are especially important in determining organ allograft survival in man. Blood group antigens have been looked for on the cells of the cornea with varying results.[6,7] Although it is generally agreed that corneal epithelial cells express blood group antigens, no such agreement has been reached regarding the occurrence of these antigens on corneal endothelial cells. Because the endothelial cells are of primary importance in the normal physiologic functioning of the cornea and because the destruction of these cells by immunologic attack results in a loss of corneal function and visual acuity, it is important to know which antigens are present on corneal endothelial cells.

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We tested biopsy specimens of human cornea prepared to preserve blood group antigens on cell membranes for antigens A, B, and D (Rh) with the immunoperoxidase procedure.

MATERIAL AND METHODS

Tissue samples—Corneal tissue was obtained from patients undergoing penetrating keratoplasty. All patients were typed for blood groups A, B, O, and D antigens with standard blood-typing procedures.

Preparation of tissue sections and imprints for staining—Freshly obtained corneal biopsy specimens were rinsed in three changes of phosphate-buffered saline, pH 7.4. In the initial experiments, a corneal biopsy specimen was trisected into pieces of approximately equal size. One portion was quick-frozen in Cryo-Quick mounting medium for sectioning in a cryostat. A second portion was fixed in a mixture of 2.5% formalin and 3% glutaraldehyde diluted in 0.1 M phosphate buffer, pH 7.4. After a four-hour fixation period, we transferred the tissue step by step through routine histologic reagents, embedded it in paraffin, and sectioned it. We prepared imprints of both epithelial and endothelial cells by gently pressing the biopsy specimen between two clean microscope slides for approximately six hours. The dried corneal fragments were detached from the slides with the edge of a razor blade, leaving a monolayer of epithelial cells on one slide and a few scattered endothelial cells on the other slide. This procedure yielded flattened cell preparations that seemed morphologically normal. The cells were not exposed to organic solvent

fixatives which might have dissolved the cell surface antigens.

Characterization and standardization of antisera—The antisera were obtained from hyperimmunized donors, and the manufacturer indicated that the major fraction of the antibody in the reagents was of the immunoglobulin G (IgG) class. We confirmed this by double immunodiffusion analysis using monospecific antisera against the heavy chains of human IgG and IgM. Additionally, we found that the staining obtained with the blood group antisera did not occur when we precipitated out the IgG fraction with the anti-IgG antisera. There was a precipitin reaction between the blood group antisera and anti-IgG, but not between the blood group antisera and anti-IgM. The rabbit anti-human IgG and the goat anti-rabbit IgG antisera used in the immunoperoxidase staining contained antibodies directed mainly against the heavy chain determinants of the IgG class, although a minor amount of anti-light-chain antibody was also present. The occurrence of antibodies against both heavy and light chain determinants did not affect the specificity of the antigen determination experiments and might have been an advantage in that the sensitivity of the assay was increased by the presence of antibodies of both specificities.

Immunoperoxidase staining procedure—We adapted the immunoperoxidase staining procedure of Sternberger.[8] Tissue sections and endothelial cell preparations were first incubated for 30 minutes in Tris buffer, pH 7.6, containing 1% normal goat serum, and then in one of several dilutions (1:10, 1:100, or 1:1,000) of human antisera to blood groups A, B, or D antigens. The incubations in the antisera usually lasted for two hours, although we found no difference in staining intensity when the incubation period was lengthened to 24 hours. After incubation in the primary antiserum, the slides were incubated for five minutes each in three separate washes of Tris buffer. We repeated this buffer wash procedure after each subsequent incubation.

The slides were then incubated in the following sequence, with washes in Tris buffer as described between each step: 1:20 rabbit anti-human IgG antiserum diluted in Tris buffer (30 minutes); 1:20 goat anti-rabbit IgG antiserum (30 minutes); and 1:50 peroxidase/antiperoxidase (rabbit) reagent (15 minutes). Some of the initial sections stained with this technique were then incubated in 3% glutaraldehyde for 30 minutes, but once we determined that this exposure did not significantly increase the staining intensity, we dropped this part of the protocol. We then incubated the slides in freshly prepared 0.05% diaminobenzidine containing 0.01% hydrogen peroxide (ten minutes). The slides were again washed in Tris buffer after the last incubation. We routinely examined stained cells and sections before counterstaining with Diff-Quick because, in some instances, the dyes in this staining solution obscured the brown immunoperoxidase reaction product. We photographed samples stained with only the immunoperoxidase stain and samples stained with both the peroxidase stain and the Diff-Quik counterstain.

In each series, we treated control slides simultaneously with those being stained for blood group antigens. Controls included the following: (1) cells and tissue not incubated in blood group antibody, but exposed to the remainder of the reagents; (2) cells and tissues incubated in 3,3′-diaminobenzidine and hydrogen peroxide only as a test for endogenous peroxidase; (3) cells and tissue incubated in the blood group antiserum but not in the second antiserum (rabbit anti-human IgG); and (4) cells and tissues incubated in the blood group antiserum and the anti-human IgG but not

in the goat anti-rabbit IgG. These controls gave uniformly negative results.

RESULTS

We obtained corneal biopsy specimens from 12 patients and examined them for A, B, and D antigens. In every instance we were able to identify correctly the blood type of the donor on the basis of the immunoperoxidase staining, proving that the same antigens were present on red blood cells and corneal cells (Table). In the paraffin sections of human cornea stained for A antigen, the A antigen appeared as a brown deposit primarily localized on the epithelial and endothelial surfaces (Fig. 1). There was little immunoperoxidase staining in the stroma. This lack of staining may be related to the compact structure of the stroma which prevents antibodies from reaching the keratocyte membrane in concentrations sufficient to cause positive staining. We are now testing tissue-cultured stromal keratocytes for the presence or absence of blood group antigens.

Blood group antigens were found on the plasma membranes of isolated endothelial cells, restricted almost entirely to the cell surface; there was almost no staining in the cytoplasm (Fig. 2). Although only corneal cells and tissues stained with anti-A antiserum are shown here, the intensity and distribution of blood group antigens did not differ significantly when we used anti-B and anti-D antisera. We did not find nonspecific staining on control tissue sections and cell preparations.

Blood group antigens have been shown to be present in body secretions; the corneal epithelial cells and the endothelium may possibly acquire these antigens from the tears and aqueous humor by adsorption. To test this possibility, we performed short-term in vitro incubation of epithelial and endothelial cells in tissue culture medium containing 1% bovine albumin as a protein source. Epithelial and endothelial cells maintained for periods of up to four days at 37°C in culture medium remained alive and were processed for immunoperoxidase staining. These cells possessed the same blood group antigens as the patient from whom they were derived. Therefore, it seems likely that these cells were actually synthesizing and expressing the antigens and not acquiring them from their milieu. The kinetics of cell membrane turnover are such that exogenously acquired molecules would be lost, diluted to extinction, or both during the four-day incubation period.

We also attempted an alternative experiment in which corneal cells from an individual of one blood type were incubated in aqueous humor or tears from an individual of another blood type. The corneal cells did not acquire foreign blood group antigens during this incubation, further supporting our impression

TABLE

RESULTS OF BLOOD TYPING AND IMMUNOPEROXIDASE STAINING OF CORNEAL ENDOTHELIUM

No. of Patients	Somatic Blood Group	Corneal Endothelial Staining Reaction					
		Antigen A		Antigen B		Antigen D	
		Positive	Tested	Positive	Tested	Positive	Tested
6	A+	6	6	0	6	6	6
1	B+	0	1	1	1	1	1
4	O+	0	4	0	4	4	4
1	O−	0	1	0	1	0	1

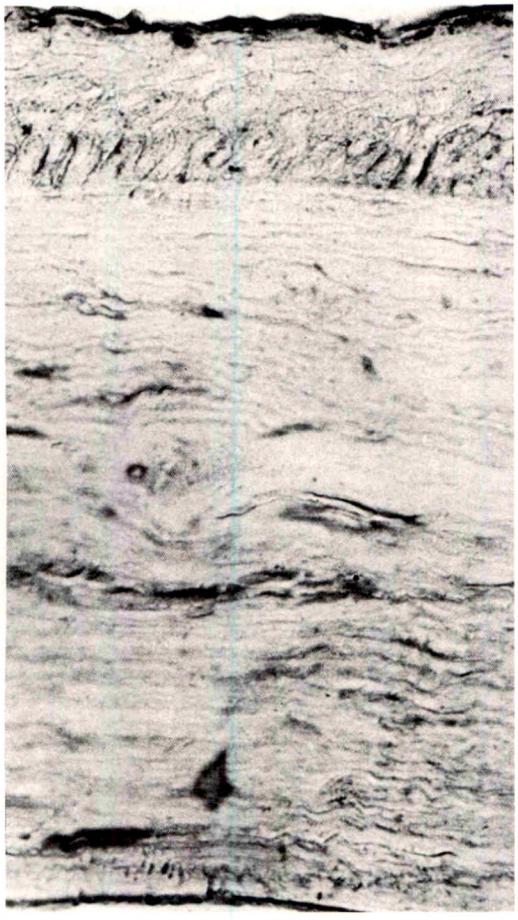


Fig. 1 (Salisbury and Gebhardt). Histologic section of the cornea from a blood type A donor, stained for specific anti-A antibody. The darkest staining can be seen as black borders on the epithelial and endothelial surfaces. At higher magnifications, it is possible to focus on the stained membranes of individual epithelial and endothelial cells (immunoperoxidase, × 160).

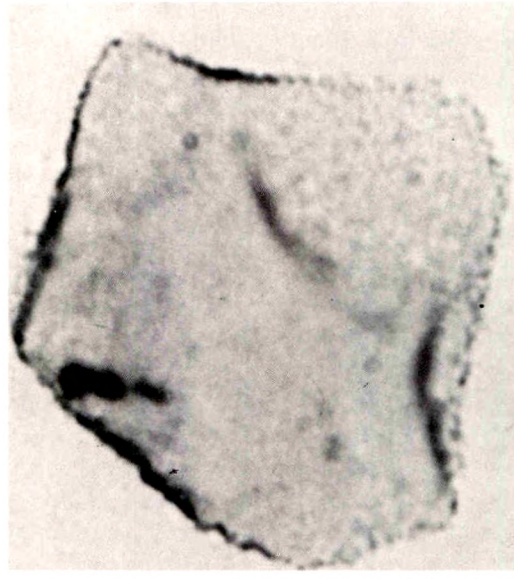


Fig. 2 (Salisbury and Gebhardt). Endothelial cell from a blood type A donor. The cell is stained only for blood group A antigen, which is localized almost entirely on the plasma membrane. Intracellular detail is not apparent because this cell was not counterstained (immunoperoxidase, × 800).

that corneal cells acquire blood group antigens endogenously and not from exogenous sources.

Discussion

The results of this study unequivocally establish that both the corneal epithelium and the endothelium possess cell membrane blood group antigens. These results are not entirely unexpected in light of previous studies in which blood group antigens were found to be widely distributed on a variety of cells in the body and particularly on stratified epithelia and the endothelial cells of blood vessels.[9]

Nelken and associates[6] sought blood group antigens in human cornea homogenates and, using an adsorption-elution protocol, were able to identify blood group antigens in these crude preparations. Obviously, they could not determine the cell layer or layers which contributed the antigens. Foster and Allansmith's[7] recent study, using fluorescent antibody and mixed agglutination techniques, indicated that the corneal epithelium but not the endothelium expressed blood group A antigen. Dua and Shidham,[10] using a similar procedure, obtained convincing evidence that both the corneal epithelium and endothelium express blood group antigenic molecules. The different results reported in these two studies may be related to the different methods used to prepare the tissue. These methods may have affected

the survival of antigen on the cell membranes. Possibly the modified red blood cell adherence procedure used by Dua and Shidham detected antigens at a lower level than the technique used by Foster and Allansmith.[7] Such a possibility is supported by the findings of this study in which the ultrasensitive immunoperoxidase procedure disclosed that both the corneal epithelium and the endothelium possess blood group antigens.

To our knowledge, no definitive data proving that donor-recipient blood group incompatibility is significant in human corneal allograft survival have been published. Results for the large group of patients studied by Allansmith and associates[11] suggest that ABO antigen mismatching does not adversely alter the fate of the donor corneal allograft. Of course, it could be argued that, although blood group antigens may not be responsible for stimulating an initial rejection response, they may be involved in strengthening or sustaining such a reaction once it has begun. Two recent, independently conducted studies, the present one and that by Dua and Shidham, confirmed the presence of blood group antigens on the human cornea, particularly the all-important endothelium, which means that we should consider blood group antigens to be potential threats to corneal allograft survival in man.

SUMMARY

The ultrasensitive immunoperoxidase staining technique demonstrated the presence of blood groups A, B, and D antigens on human corneal epithelial and endothelial cells. Nonspecific staining was not seen and there was little antigen in the corneal stroma. Blood group antigens probably do not initiate corneal allograft rejection, but they may lend antigenic support to the rejection response once it has begun.

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THE ORIGINS OF SATTLER'S VEIL

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Increased glare and halos surrounding bright lights are well-known problems associated with wearing hard contact lenses. These visual disturbances have been attributed to edema of the corneal epithelium.[1] A slit lamp shows this edema as a gray epithelial haze. Oxygen deprivation of the epithelium appears to be the cause of this disorder,[2] which is accompanied by epithelial glycogen depletion.[3] Finkelstein[4] measured the half arc of the halo seen by contact-lens users and concluded that if the halos arose from a cell layer, they could have emanated from cells 10 μ in diameter. In glaucomatous eyes, a circular diffraction grating is formed by basal epithelial cells and extracellular spaces. Miller and Benedek[5] proposed that the halos seen by contact-lens users have a similar origin. However, Smelser and associates' studies,[2,3] like more recent work,[6] disclosed that epithelial thickness does not change significantly in hypoxia. Furthermore, Dohlman[7] stated that Kuwabara found intercellular spaces of normal widths in thin sections of hypoxic epithelium. Although the proposed theoretical basis for halo formation seems sound, there is no conclusive proof.

We used an in vitro model of Sattler's veil to test the basis of halo formation.

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MATERIAL AND METHODS

We killed New Zealand White rabbits weighing between 2 and 5 kg with sodium pentobarbital and enucleated their eyes. The corneas were mounted atraumatically in an incubation chamber,[8] maintained at 35°C, and perfused with Ringer's solution.

The composition of the Ringer's solution used throughout the study was 124.7 mM sodium chloride, 25.4 mM sodium bicarbonate, 1.21 mM monobasic potassium phosphate, 7.26 mM potassium chloride, 0.61 mM magnesium sulfate, 0.65 mM calcium chloride, 0.16 mM reduced glutathione, 4.75 mM adenosine, 2.79 mM glucose, and 24 mg/liter of gentamicin. Before use, this solution was bubbled with 95% air and 5% carbon dioxide at 37°C. We obtained subatmospheric partial oxygen pressures by further bubbling with moist 95% nitrogen and 5% carbon dioxide at 37°C. We measured partial oxygen pressure with an oxygen analyzer.

In the parent Ringer's solution, the cornea maintained its normal thickness for about six hours. The endothelial surface was perfused with Ringer's solution at 6 ml/hr at a partial oxygen pressure of 35 mm Hg—the normal endothelial oxygen tension in the rabbit. The epithelial surface was perfused at 36 ml/hr with the parent Ringer's solution until stromal thickness stabilized. Subsequently, the epithelial surface was perfused with a hypoxic Ringer's solution at a partial oxygen pressure of <7 mm Hg. To ensure that the environment was hypoxic, the perfusant was transported through stainless steel tubing from glass syringes.

An automated attachment to the labora-

tory specular microscope measured both stromal and epithelial thicknesses continuously throughout the experiment with an accuracy of better than 1%.[9,10] We used the same instrument to make photometric scans through the cornea to measure the backscattering at different depths. During the photometric scans an electronic feedback loop kept the intensity of the light source constant. In companion experiments, we photographed tangential optical sections of various corneal planes with a scanning specular microscope.[11,12] All photographs were taken in the central cornea.

We set up an optical diffractometer to produce transforms of photographic images with a helium-neon laser beam as the light source.[13] The system was calibrated with square grids placed in the object plane.

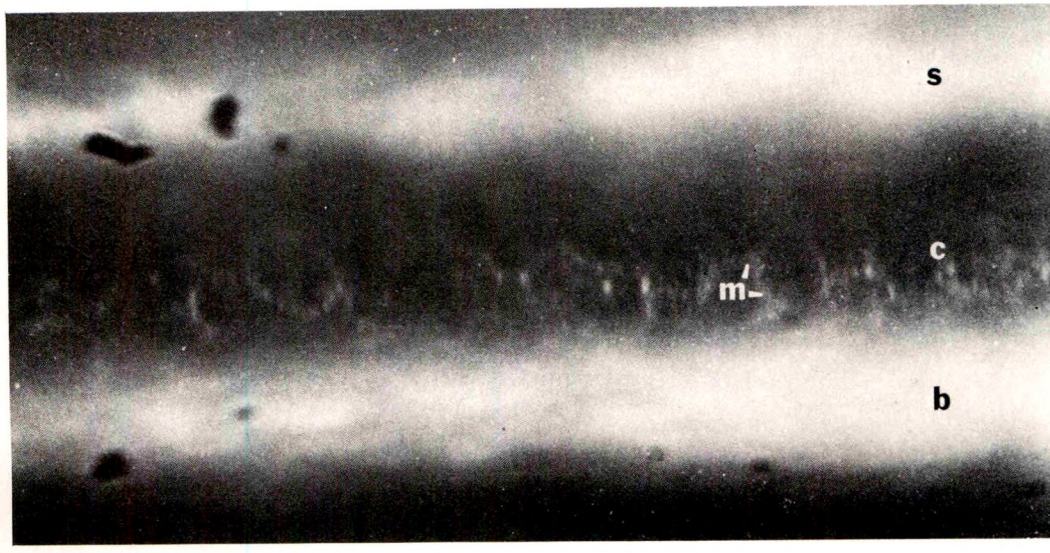
Results

When viewed with the specular microscope under control conditions, the basal and wing epithelial cells of the rabbit cornea scattered no light except at random punctate sites. Superfusion of the cornea with oxygen-deficient Ringer's solution (partial oxygen pressure, <7 mm Hg) caused the development of a polygonal mesh of scattered light that outlined the cells in the basal and intermediate epithelial regions within 30 to 60 minutes (Fig. 1). Once formed, the mesh of scattered light appeared to be constant for up to six hours with continued hypoxia. We photographed the polygonal mesh with the laboratory specular microscope (Fig. 1, top) and with the scanning specular microscope (Fig. 1, bottom). Photographic negatives obtained with the scanning specular microscope were projected at × 660 magnification and traced on acetate with India ink (Fig. 2). This was rephotographed and an optical transform, or diffraction pattern, was made with the laser diffractometer. The resulting negative showed a center maximum and a surrounding halo with a half arc of 3.1 degrees (Fig. 3). The average diameter of the epithelial cells was 12.0 μ (±0.4) by direct measurement. Knowing this and the wavelength of the laser beam, 632.8 nm, one can calculate a first-order maximum for a plane diffraction grating at 3.0 degrees (±0.1). Hypoxia produced no changes in the appearance of the endothelial mosaic. Therefore, the halo surrounding bright lights in Sattler's veil corresponds to the optical transform of the polygonal mesh of light scattered between the epithelial cells.

After the introduction of hypoxic Ringer's solution, there was a time lag of 15 to 60 minutes before the onset of stromal swelling, which coincided with the appearance of the polygonal mesh of light scattering in the epithelium. The variation in the time lag was probably caused by technical difficulties in establishing adequate hypoxia in the chamber used for the studies. Stromal edema produced by hypoxia was completely reversible as long as stromal thickness was not increased by more than 10% of its normal thickness. There were no discernible changes in the photometric scans of the cornea unless the cornea had been allowed to swell beyond 10% of its normal thickness. Beyond this point, we detected a notable increase in stromal backscatter (Fig. 4).

Occasionally, backscatter from prominent stromal elements (presumably keratocytes) was recorded during the response to hypoxia. With these features as markers of anterior and posterior lamellar thickness, measurements in three corneas showed the amounts of anterior and posterior stromal swelling to be similar.

Finally, despite the overall accuracy and objectivity of the automatic specular microscope measurements (±0.1 μ), we found no consistent changes in the thick-



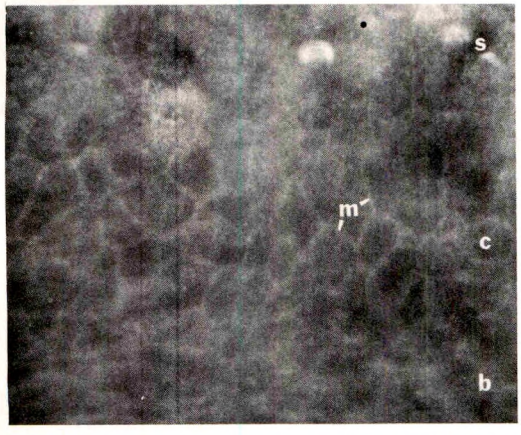


Fig. 1 (Lambert and Klyce). Patterns of light scattering in the hypoxic epithelium. Top, With the laboratory specular microscope, polygonal scattering sites (m) can be seen in the central and deeper epithelium (c). Diffuse bands of light correspond to squamous cell layer (s) and Bowman's membrane (b). Bottom, Polygonal mesh (m) of scattered light recorded with the scanning specular microscope. This tangential section scanned squamous cells (s), the central epithelium (c), and Bowman's membrane (b).

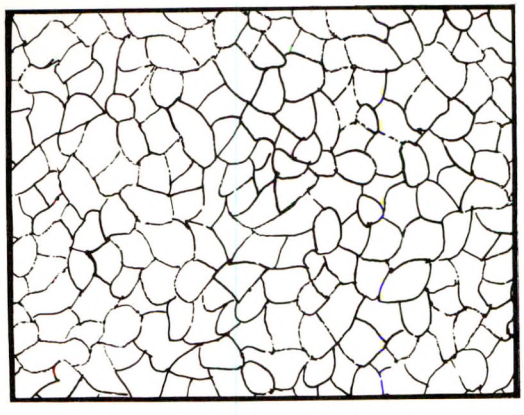


Fig. 2 (Lambert and Klyce). India ink tracing of polygonal scattering sites in epithelium from a hypoxic cornea.

ness of the epithelium in response to hypoxia with this technique.

Discussion

These results showed that light-scattering sites form between epithelial cells after hypoxia of the epithelial surface in vitro. These scattering sites presumably result from a differential refractive index between the basal cell cytoplasm and the extracellular space and should be accompanied by cell swelling and separation. Because a consistent change in epithelial thickness was not demonstrable, even with the high ac-

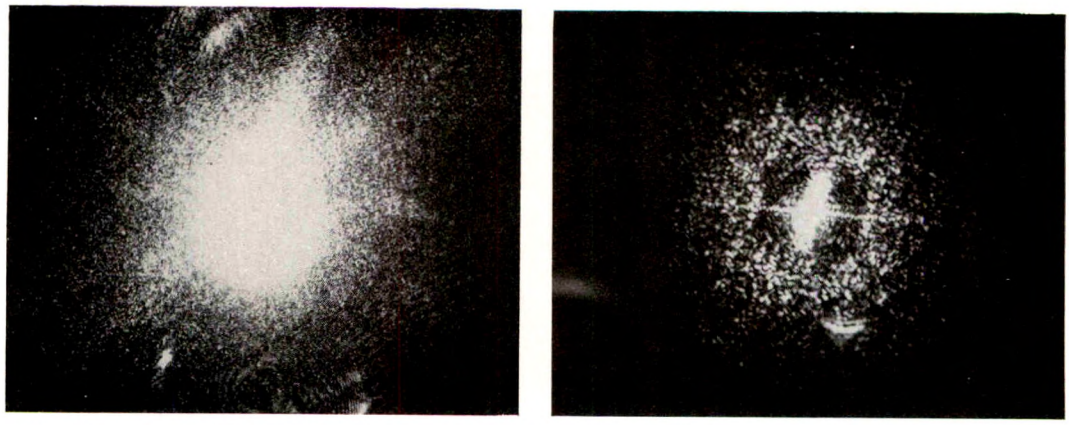
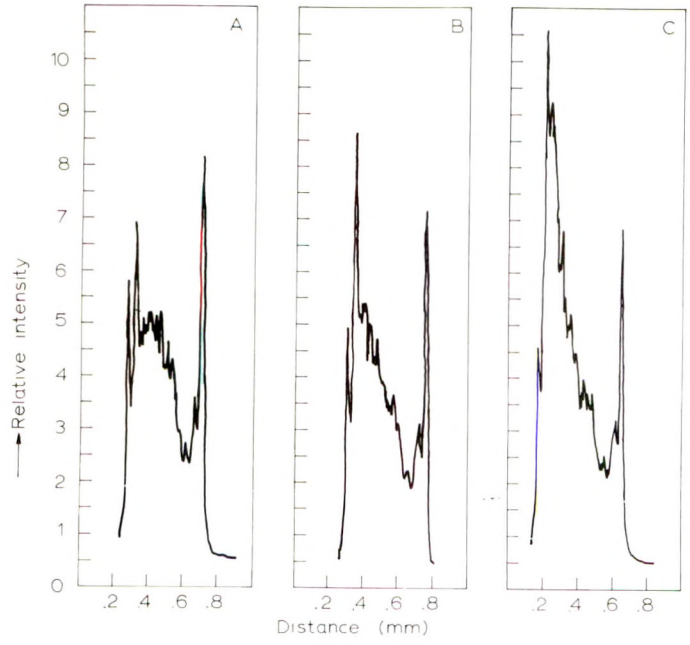


Fig. 3 (Lambert and Klyce). Optical transform of polygonal mesh of light scatter. Left, In a control cornea, there was no uniform diffraction pattern. Right, Optical transform of polygonal mesh shown in Figure 2. The arc subtended by this halo is identical to that reported by contact-lens wearers.



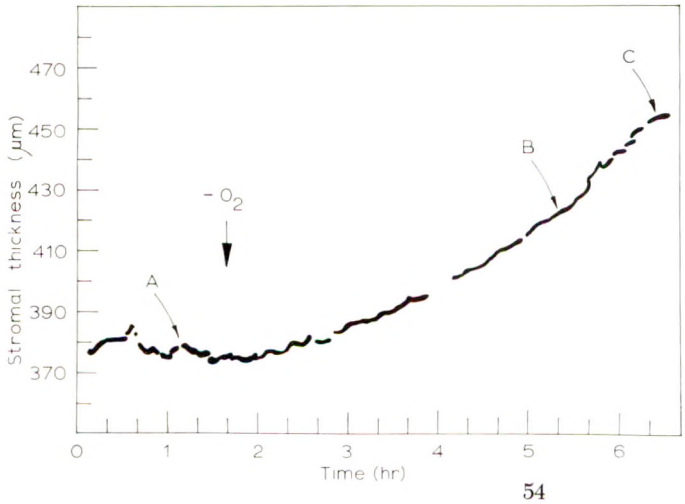


Fig. 4 (Lambert and Klyce). Stromal swelling and light scattering after hypoxia in rabbit cornea. Note the increase in stromal backscatter shown at top right. The roughly exponential characteristic of the light intensity with stromal depth is consistent with the pattern obtained from media of uniform turbidity.

curacy of the automatic specular microscope, radial swelling with a concomitant change in corneal curvature may occur. The details of morphologic alteration by hypoxia may be apparent only in fast-frozen tissue examined by electron microscopy, because conventional aldehyde fixation has not disclosed abnormalities in the extracellular space.[7] Measurements of epithelial thickness during hypoxia in frozen sections[3] and in living tissue[6] failed to demonstrate significant changes. Yet, in conventionally fixed and dehydrated tissue,[14] there is a marked correlation between epithelial thickness and oxygen deprivation. Possibly, the living epithelium contains some compensatory regulatory mechanism dependent upon metabolic energy.

The sides of the polygonal meshwork of scattered light are more or less randomly oriented. Hence, the diffraction pattern produced creates a circle, forming the diffuse halos observed around point sources of light.

The stromal edema observed in these experiments was comparable to that seen in previous work.[6] The swelling occurred throughout the stroma (Fig. 4, top right), shown by direct measurements of anterior and posterior lamellar thicknesses. The rate of lactate production by the hypoxic cornea in vitro was doubled and all excess lactate was liberated to the aqueous humor.[15] Preliminary data suggest that oxygen deprivation in the tear film leads to stromal edema through an increase in stromal osmolarity.[16] The increase in stromal backscatter that accompanies prolonged hypoxia should contribute to glare, but the hypoxic epithelium is apparently the major source of glare sensation in this disorder.[5]

Thus, hypoxia-induced changes in the refractive properties of the epithelium produce a pattern of backscattered light from the extracellular space; this is the source of the halo in Sattler's veil. Changes in epithelial volume cannot be demonstrated with the present techniques. Edema accompanying hypoxia occurs uniformly across the stroma, and, if severe, is accompanied by increased backscatter, a likely secondary source of the glare phenomenon. These findings emphasized the maintenance of an adequate supply of oxygen from the atmosphere in normal epithelial function.

Summary

The symptoms of Sattler's veil accompany prolonged wear of thick contact lenses and are thought to be the result of epithelial hypoxia. We studied light scattering in the isolated rabbit cornea after hypoxia with optical techniques. We found that hypoxia produced a polygonal mesh of light scattering, outlining cells in the intermediate and basal epithelium. An optical transform of this mesh produced a halo of the same dimensions observed in Sattler's veil. Additionally, prolonged hypoxia produced an increase in stromal light scattering that may add to increased glare sensitivity. Consistent changes in epithelial thickness were not observed, raising the possibility of radial swelling or compensatory epithelial volume regulation.

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OPHTHALMIC MINIATURE

With both eyes open and looking straight ahead, Bradley sees a hundred and ninety-five degrees on the horizontal and about seventy degrees straight down, or about fifteen and five degrees more, respectively, than what is officially considered perfection. Most surprising, however, is what he can see above him. Focused horizontally, the typical perfect eye, according to the chart, can see about forty-seven degrees upward. Bradley can see seventy degrees upward. This no doubt explains why he can stare at the floor while he is waiting for lobbed passes to arrive from above. Dr. Abrams said that he doubted whether a person who tried to expand his peripheral vision through exercises could succeed, but he was fascinated to learn that when Bradley was a young boy he tried to do just that. As he walked down the main street of Crystal City, for example, he would keep his eyes focused straight ahead and try to identify objects in the windows of stores he was passing.

William Howarth, Editor, *The John McPhee Reader*
New York, Random House, Inc., 1976

OCULAR HISTOPATHOLOGY IN MULTIPLE ENDOCRINE NEOPLASIA TYPE 2b

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The significance of thickened corneal nerves as a hallmark of multiple endocrine neoplasia type 2b has recently been stressed by Robertson, Sizemore, and Gordon.[1] This frequently familial clinical syndrome is characterized by malignant pheochromocytomas, medullary carcinoma of the thyroid gland, many mucosal neuromas, and, often, a marfanoid habitus. Early recognition of this syndrome is important because both malignancies may cause death. Ophthalmologists have a unique opportunity to diagnose this syndrome before the manifestations of the malignancies become apparent. Thickened nerves seen in a cornea with an otherwise clear stroma should be regarded as a positive indicator of multiple endocrine neoplasia type 2b.[1] In addition to thickened corneal nerves, neuromas of the eyelids and conjunctivas and keratoconjunctivitis sicca are characteristic ocular manifestations. We studied the histopathologic features of ocular involvement in multiple endocrine neoplasia type 2b.

REPORT OF A CASE

A 30-year old man was first seen at the Mayo Clinic in 1972. He was one of five affected members in a family afflicted with multiple endocrine neoplasia type 2b (he was identified as III-3 in the pedigree previously published. Obvious abnormalities found at physical examination included a marfanoid habitus, thickened everted lips, an enlarged nodular tongue with many neuromas, and irregular neuromal excrescences on the margins of the eyelids.

Enlarged nerves in the subconjunctival region caused localized elevations of the overlying tarsal and bulbar conjunctiva. On either side of the corneoscleral limbus, enlarged nerves, arranged in bundles, appeared as flat, poorly delineated neuromas. Dilated perilimbal conjunctival vessels were associated with these neuromas. Prominent corneal nerve fibers extended in a lacy pattern across the entire cornea. These nerves were invisible to the unaided eye, but a slit lamp showed that they were large and distinctly abnormal (Fig. 1).

Physical examination disclosed an enlarged thyroid gland but no other physical abnormalities. Laboratory studies failed to provide conclusive evidence of pheochromocytoma, in spite of urinary catecholamines, metanephrines, vanillylmandelic acid, and plasma catecholamines tests on several occasions before and after stimulation with both glucagon and histamine. Measurements of plasma immunoreactive calcitonin suggested medullary carcinoma of the thyroid gland and prompted thyroid surgery. The isthmus of the thyroid was removed, and a subtotal right-sided and total left-sided thyroidectomy was done. Histopathologic study confirmed the diagnosis of medullary thyroid carcinoma. Fifteen months after the thyroidectomy, plasma immunoreactive calcitonin studies indicated persistence of active medullary carcinoma. A bilateral radical neck dissection was performed for residual medullary thyroid carcinoma and metastasis of the adjacent lymph nodes. During the evening after surgery, bleeding caused compression of the trachea and, although the bleeding points were ligated, the patient suffered respiratory and cardiac arrest, failed to regain consciousness, and died 23 days later.

MATERIAL AND METHODS

The right eye and lacrimal gland were obtained at autopsy three hours after death. We excised a radial section of cornea containing a thickened corneal nerve superiorly and placed it in 2% glutaraldehyde. We cross-sectioned blocks of the corneal tissue containing the nerve at the corneoscleral limbus and at sites approximately 1.5 mm and 3.0 mm centrally from the corneoscleral limbus and prepared them for electron microscopy. We stained thick sections, approximately 1 μ thick, with azure II, and ultrathin sections with uranyl acetate and lead citrate.

We placed the rest of the globe, the lacrimal gland, and an epibulbar mass in

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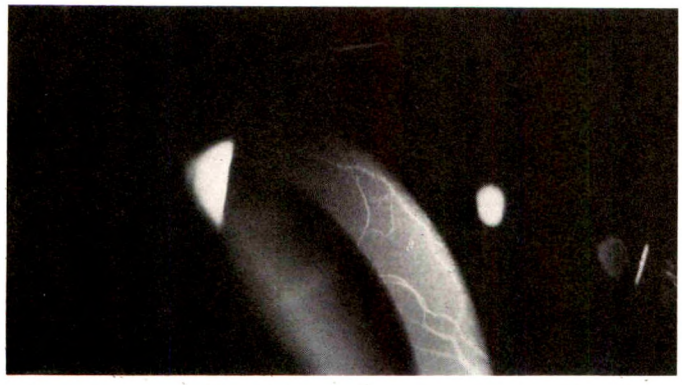


Fig. 1 (Riley and Robertson). Slit-lamp photomicrograph demonstrating thickened corneal nerves in multiple endocrine neoplasia type 2b (Reprinted with permission from Robertson, D. M., Sizemore, G. W., and Gordon, H.: Thickened corneal nerves as a manifestation of multiple endocrine neoplasia. Trans. Am Acad. Ophthalmol. Otolaryngol. 79:OP-772, 1975).

10% neutral buffered formalin, and prepared tissue blocks for light microscopy. Sections were stained with hematoxylin and eosin, PAS, Luxol fast blue, Masson trichrome, and Bodian stains.

Results

Gross pathologic findings—The globe was normal in dimensions; the lower two thirds of the cornea showed exposure keratopathy, but the upper one third of the cornea, from which we had removed the corneal section, appeared to be normal. Conjunctival neuromas were visible near the corneoscleral limbus. The intraocular structures appeared to be normal after the globe was opened.

Light microscopic findings—Abnormal findings were limited to the cornea, the conjunctiva, anterior episcleral tissues, the ciliary body, and the iris. The conjunctival epithelium was normal, but many prominent nerve bundles were present in the episcleral and superficial scleral layers from the corneoscleral limbus to near the equator. Bundles of nerves extended into the cornea at the approximate junction of the posterior two thirds and the anterior one third of the corneal stroma. With Luxol fast blue, the nerves appeared to be a mixture of myelinated and unmyelinated fibers, except in the cornea where they appeared to be unmyelinated. The central cornea showed exposure keratopathy, with thinning and loss of the epithelium, and many inflammatory cells infiltrating the stroma.

Within the ciliary body and iris, prominent nerve fibers extended from the ciliary body through the root of the iris (Fig. 2, top) and terminated in the area of the sphincter muscle, whose structure seemed disrupted by the nerve fibers. Bodian stain demonstrated many axons, while Luxol fast blue showed a mixture of myelinated and unmyelinated fibers. Masson trichrome stain disclosed no excessive connective tissue associated with the nerve bundles. We also observed myelinated and unmyelinated fibers singly or in small bundles in the anterior ciliary muscle and in the uveal meshwork, extending into the trabecular meshwork nearly to Descemet's membrane. Numerous ganglion cells were evident in some sections in the anterior ciliary body, at the root of the iris, and in the uveal meshwork, with as many as eight to ten ganglion cells present in a single section (Fig. 2, bottom). Sections of the lacrimal gland showed normal glandular tissue.

Electron microscopic findings—Examination of 1-μ sections of the corneal nerve at the corneoscleral limbus disclosed myelinated and unmyelinated fibers; two vessels were associated with the nerve bundle (Fig. 3, top). These vessels consisted of a single layer of endothelial cells surrounded by a thin basement membrane. There were no intravascular cells to identify these vessels as capillaries rather than as lymphatic vessels. The nerve bundle was contained

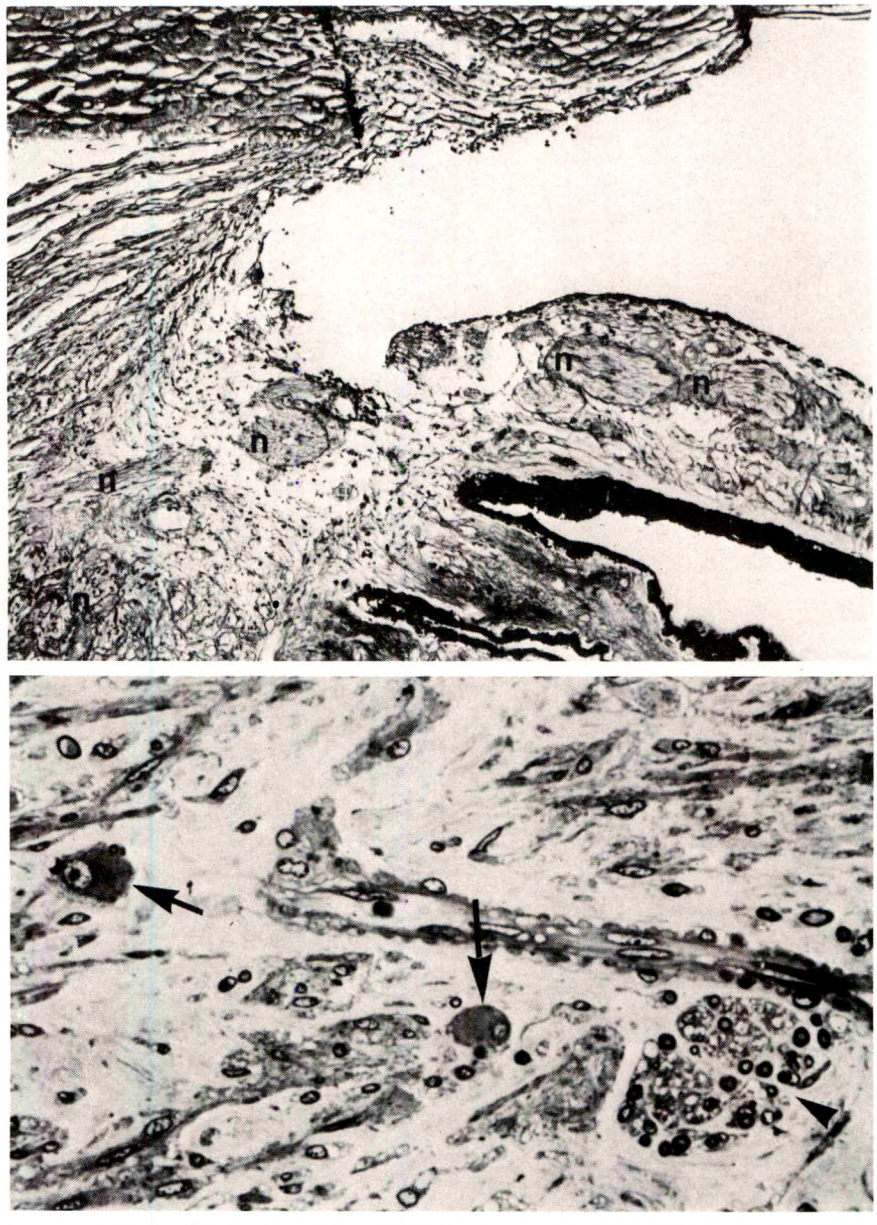


Fig. 2 (Riley and Robertson). Top, Ciliary body and root of iris. Note numerous nerve bundles (n) in both structures (hematoxylin and eosin, ×100). Bottom, Ciliary body ganglion cells (arrows), and myelinated and nonmyelinated nerve fibers (arrowhead) (hematoxylin and eosin, ×250).

within a single layer of perineural cells with associated basement membrane material. The myelinated and unmyelinated nerve fibers within the nerve bundle were encased in Schwann cell cytoplasm, and the interstices contained collagen fibers. External to this layer, the corneoscleral limbal stroma was composed of collagen and elastic fibers (Fig. 3, bottom).

The 1-μ section of the nerve bundle taken approximately 1.5 mm centrally from the corneoscleral limbus showed a cluster of endothelial cells in the central

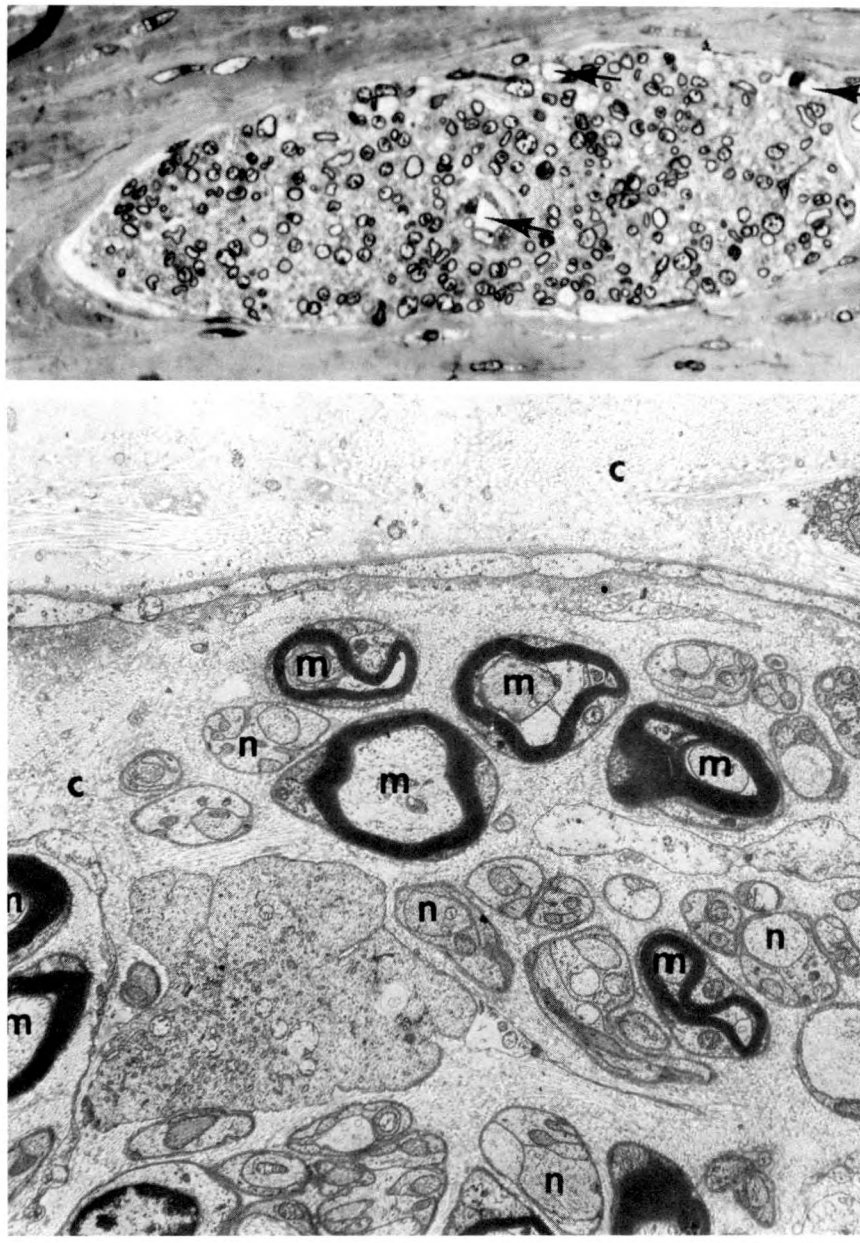


Fig. 3 (Riley and Robertson). Top, A 1-μ section from the corneoscleral limbus. Nerve bundle containing myelinated and nonmyelinated nerves. Note small vessels (arrows) (azure II, epoxy resin-embedded, ×640). Bottom, Margin of nerve bundle from the corneoscleral limbus with myelinated (m) and nonmyelinated (n) fiber surrounded by collagen (c) (×6,000).

portion of the nerve structure which appeared to be a tangential section through the apex of a capillary loop (Fig. 4, top). We did not see myelinated fibers at this level in the ultrathin sections examined, and the nerve bundle consisted entirely of unmyelinated fibers associated with Schwann cells and intervening collagen fibers (Fig. 4, bottom). The perineural cell layer was no longer

present, and corneal collagen fibers were adjacent to the basement membrane material of the Schwann cells.

Approximately 3 mm centrally from the corneoscleral limbus, the nerve bundle had no associated endothelial cells and was composed entirely of unmyelinated fibers and Schwann cells (Fig. 5, top left and top right). Inflammatory cells, probably infiltrating the cornea in response to the exposure keratopathy, were present adjacent to the nerve bundle. Individual nerve fibers in the superficial corneal stroma consisted of unmyelinated fibers surrounded by Schwann cell cytoplasm (Fig. 5, bottom left).

In general, the morphologic appearance of the myelinated and unmyelinated nerve fibers appeared to be normal, both in regard to the Schwann cell structure, whether forming a myelin sheath or mesaxon, and the axoplasm, which contained neurofilaments, microtubules, endoplasmic reticulum, mitochondria, vesicles, and dense-core vesicles. Various axonal sizes were found, ranging from diameters of approximately 0.1 to 0.2 nm to those between 0.1 and 1.4 nm.

Discussion

Corneal innervation has been studied extensively, but unfortunately few investigators have used electron microscopy. Zander and Weddell,[2] in a classic study using light and phase microscopy, found that 70 to 80 bundles of nerves enter the human cornea; approximately one half of these had 15 to 20 axons, while the remainder had fewer axons. In the rabbit, and by implication in man, they found myelinated fibers only at the corneal periphery, with most of the fibers losing their myelin within 1 mm of the corneoscleral limbus; in no instance did myelination extend more than 2 mm into the cornea. Hoyer[3] stated that most of the corneal nerves "lose their myelin sheaths 0.3 to 0.5 mm. from the corneal margin and continue as" axis cylinders enclosed by Schwann cells. Hogan, Alvarado, and Weddell[4] stated that most fibers "lose their myelin sheaths in the peripheral 2 to 3 mm. of the cornea." Whitear[5] in an electron microscopic study of corneal innervation in mice, found that the deeper peripheral nerve bundles had a perineural sheath which was not observed with nerves at other levels.

Our observations indicate that corneal nerves in multiple endocrine neoplasia type 2b generally conform to the pattern of normal corneal nerves. At the corneoscleral limbus, the nerve bundle is a mixture of myelinated and unmyelinated axons associated with Schwann cells surrounded by a perineural sheath with collagen fibers present in the interstices. An interesting association was the accompanying vascular structure. Biomicroscopic examination of some patients with multiple endocrine neoplasia type 2b has shown capillary circulation in the peripheral portion of the corneal nerves, not unlike corneal neovascularization.

Myelination and the perineural sheath, as well as the vascular structures, become absent approximately 1 to 1.5 mm centrally from the corneoscleral limbus. The nerve bundles then consist exclusively of unmyelinated axons associated with Schwann cells. We also observed this relationship for single axons in the central cornea.

In 1940, Koke and Braley[6] described a patient who had pheochromocytoma associated with plexiform neuromas of the palpebral and bulbar conjunctiva and prominent corneal nerve fibers. They believed that the prominent corneal nerves were myelinated. Other authors have subsequently referred to prominent corneal nerves as medullated fibers,[7,8] whereas some have described them as thickened or hypertrophic.[9] In the material that we examined, myelination of the corneal nerve terminated near the corneoscleral limbus. We found approximately 25 axons in the cross section of the

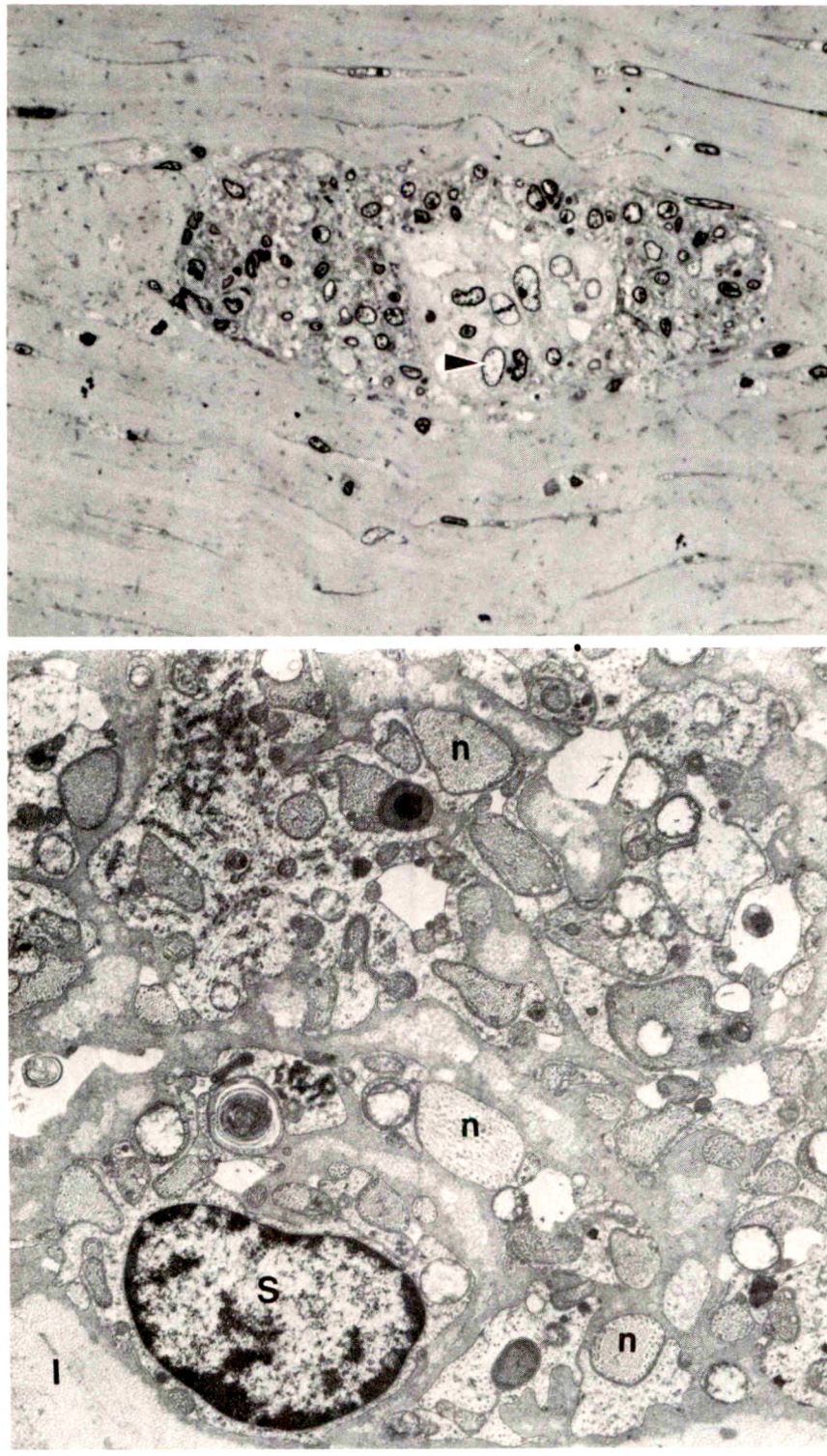


Fig. 4 (Riley and Robertson). Top, A 1-μ section 1.5 mm centrally from the corneoscleral limbus showing nerve bundle with nonmyelinated fibers and cluster of endothelial cells, one of which is identified (arrowhead) (azure II, epoxy resin-embedded, ×640). Bottom, Nerve bundle 1.5 mm from the corneoscleral limbus. Many nonmyelinated fibers (n) lie adjacent to Schwann cell (S). I, Corneal lamella (×8,000).

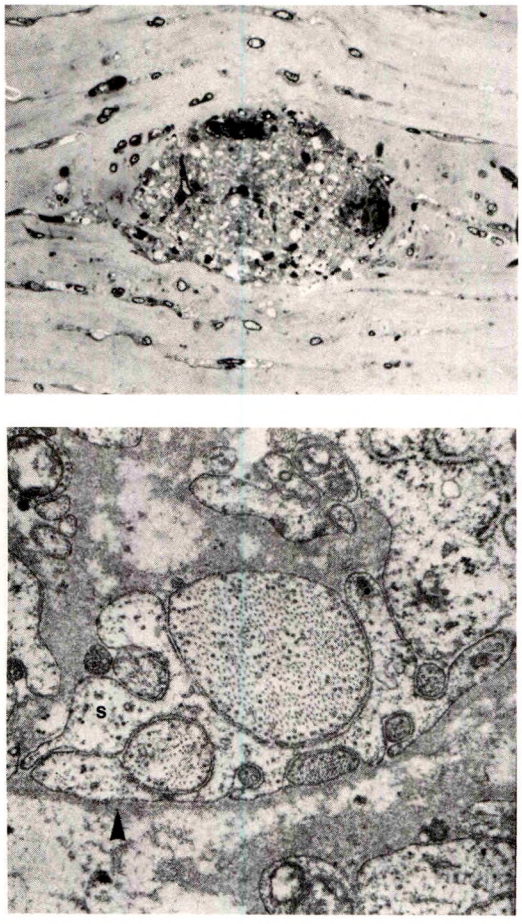


Fig. 5 (Riley and Robertson). Top left, A 1-μ section 3.0 mm centrally from the corneoscleral limbus showing nerve bundle with only nonmyelinated fibers (azure II, epoxy resin-embedded, ×640). The histologic appearance is somewhat altered by exposure keratitis. Top right, Nerve bundles 3.0 mm from the corneoscleral limbus are composed of nonmyelinated fibers (n) between corneal lamellae (I) (×12,000). Bottom left, Unmyelinated nerve fibers 3.0 mm from the corneoscleral limbus are surrounded by Schwann cell cytoplasm (s) and basement membrane material (arrowhead) (×28,000).

nerve that we studied by light microscopy, compared with 15 to 20 axons in nerve fiber bundles found in the normal eye.[2] The large size and prominence of the corneal nerves in multiple endocrine neoplasia type 2b may be caused by an increased number of axons, but careful morphometric studies comparing serial electron microscopic sections of normal and abnormal corneal nerves are needed to prove this. An increased number of Schwann cells or the increased size of individual axons (or both) may also contribute to the large size of the corneal nerves.

The presence of many ganglion cells in the ciliary body and neural abnormalities in the ciliary muscle, iris, and trabecular structures was unexpected. Normally, ganglion cells in the ciliary body are present predominantly along the inner border of the ciliary muscle, although some are located within the ciliary muscle.[10] Myelinated and unmyelinated fibers are found in the ciliary muscle,[11] whereas in the trabecular structures myelinated fibers are found only in the region of the scleral spur. There are unmyelinated fibers throughout much of the trabecular meshwork.[12]

In the sections that we studied, there were many myelinated and unmyelinated fibers, individually or in bundles, in the ciliary muscle, in the root of the iris, and in the trabecular tissues. Large nerve bundles extended through the ciliary body into the iris, appearing to terminate in association with the sphincter muscle.

Several reports of pathologic studies of patients with multiple endocrine neopla-

sia type 2b disclosed diffuse ganglioneuromatosis of the entire gastrointestinal tract, with involvement from the oral cavity to the rectum.[8,13–16] Neuromas have been found to involve the bronchi, bladder, and spinal dorsal nerve roots as well.[14] Koke and Braley[6] found ganglion cells within a conjunctival neuroma in their patient. This diffuse ganglioneuromatosis probably involves the anterior uveal tract.

We found no involvement of the lacrimal gland in our specimen. Therefore, the keratoconjunctivitis sicca frequently observed in multiple endocrine neoplasia type 2b probably represents a manifestation of an autonomic dysfunction secondary to the neuromas, as suggested by Schimke and associates.[14]

Summary

Multiple endocrine neoplasia type 2b is a syndrome, often familial, characterized by medullary carcinoma of the thyroid gland, pheochromocytoma, and many mucosal neuromas in patients who frequently have a marfanoid habitus. The ophthalmic findings are distinctive and include conjunctival and eyelid neuromas, keratoconjunctivitis sicca, and, most characteristically, greatly thickened corneal nerves.

Ocular histopathologic study of a 30-year-old man demonstrated thickened nerves in the ciliary body, iris, and corneoscleral limbal regions. In these regions, the nerves were a mixture of myelinated and unmyelinated fibers. Sections through the thickened corneal nerves, 1.5 mm centrally from the corneoscleral limbus, showed only nonmyelinated nerves associated with Schwann cells. The many axons were normal in appearance but varied in diameter; some were between 0.1 and 0.2 ng and others between 1.0 and 1.4 ng. An unexpected finding was numerous ganglion cells in the ciliary body. The subconjunctival tissue showed neuromas with a minimum of fibrous structure. Sections of the lacrimal gland showed normal glandular tissue.

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BILATERAL OPHTHALMOMYIASIS INTERNA

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Recent reviews of ophthalmomyiasis interna posterior ("fly in my eye" syndrome) have emphasized the pathognomonic appearance of linear retinal pigment epithelial disturbances throughout the fundus caused by larvae.[1,2] In the nine previously reported North American cases, no bilateral characteristic ophthalmoscopic findings have been described.[1-6] We describe a case of bilateral ophthalmomyiasis. Bilateral subretinal worm tracks suggest that maggots may bore through ocular coats or their perforating vessels, through orbital tissues, nasal cavities, and ethmoid sinuses to reach the fellow eye. Alternatively, separate periocular fly ovipositions may explain bilateral findings.

CASE REPORT

A 33-year-old man was referred for examination of a macular lesion of the left eye. The patient was an avid outdoorsman. He could not recall being struck in the eye by a fly, but he did remember being photosensitive, especially in sunlight, since childhood. Approximately one year previously, the patient had noticed some distortion in the central vision of the left eye. Occasionally his vision would clear, but gradually it worsened. On his first ophthalmologic examination he had an active parafoveal chorioretinitis. He was given corticosteroids orally for approximately one month. His vision improved somewhat until one week before his referral, when the patient again noticed metamorphopsia and poor vision of his left eye. No problems were reported with the right eye.

On ocular examination the patient's best corrected visual acuity was R.E.: 6/7.5 (20/25) and L.E.: hand motions. A central scotoma was present in the left eye. Intraocular pressures and the anterior segments of both eyes were normal. Examination of the vitreous cavities showed the absence of cells or floaters. Myelinated nerve fibers extended superiorly from the optic disks in both eyes.

Retinal examination of the right eye showed a curvilinear disturbance in the peripheral pigment epithelium anterior to the equator arching superiorly from the 9-o'clock to approximately the 3-o'clock position (Fig. 1, left). The peripheral hypopigmented track originated from a long posterior ciliary vessel temporally. At one point along the subretinal track, a single large pigment clump was seen. No organism, either alive or dead, could be found by scleral depression or gonioscopy of the right eye. Several small depigmented spots werre found outside the vascular arcades. Some of these were in line with the long migration track.

Examination of the left retina showed a similar disturbance in the retinal pigment epithelium. This track arched curvilinearly posterior to the equator from approximately the 12-o'clock position temporally to the 4-o'clock position, where it ended in a vortex vein (Fig. 1, right). There were no large pigment clumps in the course of this track. Scattered outside the vascular arcades were small hypopigmented chorioretinal scars like those in the right eye. In both fundi, the worm tracks were single and did not crisscross in the well-described random pattern noted by others.[1,2] The macula of the left eye showed a serous detachment with retinal striae and subretinal hemorrhage. A semitranslucent, larva, one-third disk diameter in length, was present just temporal and superior to the fovea (Fig. 2). Six weeks later, the parasite moved slightly closer to the fovea as the subretinal hemorrhage organized.

Fluorescein angiography of both eyes (Figs. 3 and 4) showed characteristic hyperfluorescence indicating transmission "window" defects along the damaged pigment epithelial larval migration paths. There was no abnormal dye leakage except at the left macula, where the maggot had caused a hemorrhage beneath the pigment epithelium (Figs. 5 and 6).

DISCUSSION

Parasitic larval stages of *Dipterous* flies have been found in the orbit and on the conjunctiva (ophthalmomyiasis externa), and also within the globe itself (ophthalmomyiasis interna) depending on the invasive habits of the species. Most such fly larvae have as their usual hosts sheep, deer, cattle, goats, and horses in which they penetrate intact skin, orbits, or nasal cavities. In humans, the fate of the maggot depends on its adaptation to unsuitable host tissues.[7] Some

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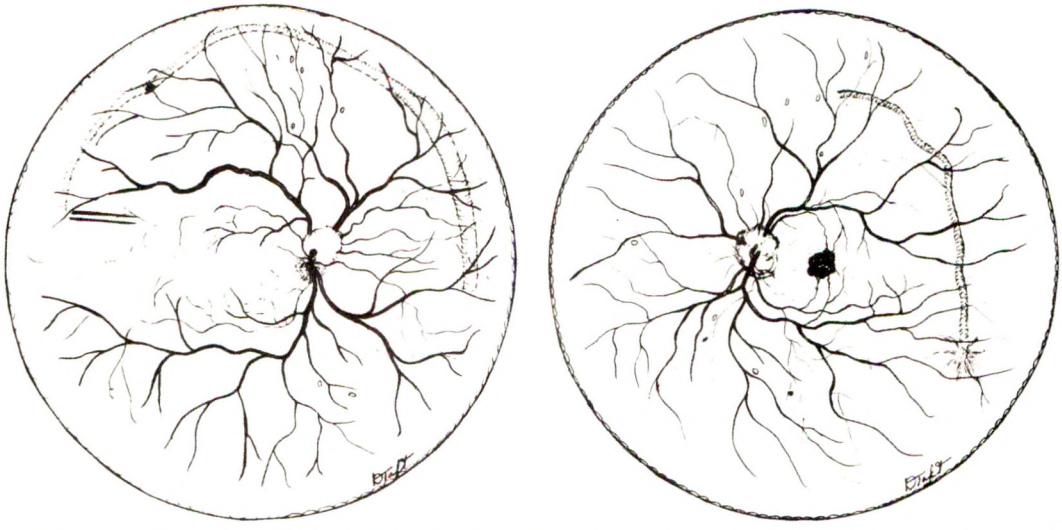


Fig. 1 (Mason). Left, Retinal drawing of right eye, showing subretinal larval track superiorly, normal macula, and scattered depigmented spots. Right, Retinal drawing of left eye, showing subretinal larval track extending to a vortex vein. Parasite is seen at macula, where it has caused subretinal hemorrhage.

larvae may survive in the human eye for years, although further maturation is doubtful.

Presumably the maggot enters and exits the human globe through the optic nerve, bloodstream, or sclera. In our case, the solitary worm tracks could be easily traced to a vortex vein ampulla in the left eye and to a long posterior ciliary vessel in the right eye. Normal ocular vascular channels may allow better entry into an eye for these parasites than would

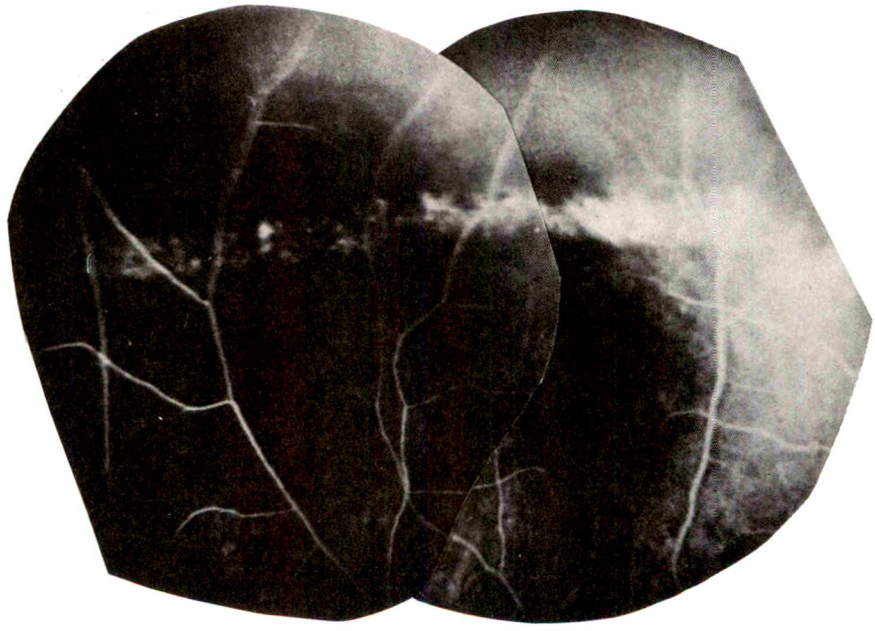


Fig. 3 (Mason). Right eye, Composite fluorescein angiogram, showing subretinal track.



Fig. 2 (Mason). Left eye, Fundus photograph of subretinal hemorrhage caused by maggot on Aug. 16, 1978. Compare to larva's position in Figure 5.

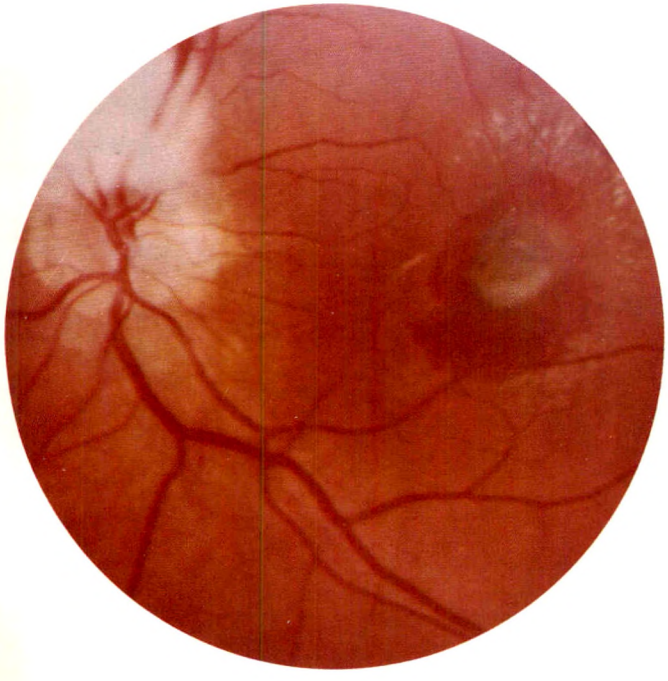


Fig. 5 (Mason). Fundus photograph of maggot at macula on Oct. 6, 1978. Note organization of subpigment epithelial hemorrhage.

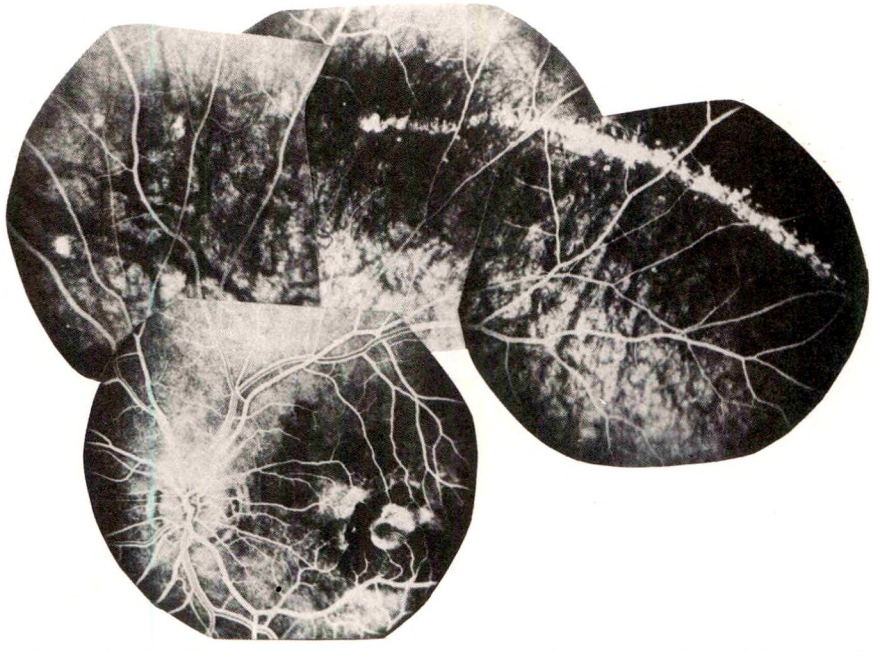


Fig. 4 (Mason). Left eye, Composite fluorescein angiogram, showing subretinal track.

intact sclera. In any case, the botfly larvae are known to possess remarkable organs for host invasion.

Not all larval fundus migrations were inscribed on the retinal pigment epithelium as tracks. For example, no marking delineated the path from the peripheral track to the larva in the left macula. Apparently a larva of small size can wander deep into the posterior choroid with hardly a trace, occasionally showing itself by surfacing through Bruch's membrane and retinal pigment epithelium. Isolated hypopigmented chorioretinal scars scattered throughout the fundi may represent either sites of excreta or points of worm excursions more internal from the choroid. Although findings such as equatorial linear pigment disturbances are seen in the histoplasmosis syndrome, photographic documentation of larval movement presented in this case supports the diagnosis of ophthalmomyiasis interna (Figs. 2 and 5).

Why larvae deposited on human conjunctiva have occasionally numbered from eight to ten organisms and yet only single worms have been reported in cases of ophthalmomyiasis interna is not understood. To our knowledge, the bilat-

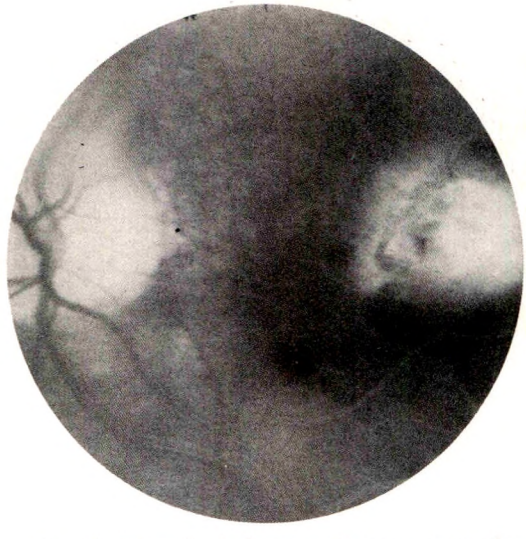


Fig. 6 (Mason). Left eye, Late-sequence fluorescein angiogram showing macular maggot.

eral worm tracks made our case unique. Either there were fly larval deposits in both eyes or a single meandering maggot bored from one orbit to the other. The first possibility is unlikely. The second possibility is extraordinary because of the variety of tissues the maggot had to penetrate before it could enter the second eye. The frontal sinus and nasal cavities can be common sites of botfly larval infestations.[8]

If the possibility of transorbital migration is confirmed, reluctance to destroy a live intraocular maggot while waiting for its spontaneous departure from the eye may eventually prove dangerous to the fellow eye. Reluctance to kill a live intraocular maggot has been based on the occasional significant inflammatory reaction that accompanied the death of the organism.[6,7] Recent reports, however, suggest that photocoagulation is effective for larval destruction without causing severe uveitis.[4,9]

SUMMARY

A 33-year-old man had bilateral ophthalmomyiasis interna. A larval maggot, presumably a botfly, caused a subretinal hemorrhage in the left macula. Diagnostic retinal pigment epithelial worm tracks were observed in both fundi, although the subretinal larva itself was observed only in the left eye. Bilateral worm tracks suggested either separate infestations in each eye or migration of a single maggot from one eye to the other.

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PERCENTAGE OF DEGRANULATED MAST CELLS IN VERNAL CONJUNCTIVITIS AND GIANT PAPILLARY CONJUNCTIVITIS ASSOCIATED WITH CONTACT-LENS WEAR

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Vernal conjunctivitis and giant papillary conjunctivitis associated with contact-lens wear have similar symptoms (itching and redness), a similar clinical appearance (giant papillae on the upper tarsal conjunctiva[1]), and a similar histologic abnormality (mast cells in the epithelium[2]). Yet, the histamine level of tears in vernal conjunctivitis is, on the average, four times greater than that in giant papillary conjunctivitis associated with contact-lens wear.[3] (Histamine levels of tears in giant papillary conjunctivitis are equal to or lower than normal.)

The main source of histamine in tissues is the mast cell.[4] The number of mast cells per cubic millimeter of conjunctiva in vernal conjunctivitis is slightly, but not significantly, greater than that in giant papillary conjunctivitis associated with contact-lens wear (by light microscopy); neither number differs markedly from normal.[2] The following question thus arises: Does the degree of mast cell degranulation (and the implied release of histamine) in the two conditions account for the observed differences in histamine levels of tears? The purpose of the present study was to determine the percentage of degranulated mast cells in the conjunctival tissue from three groups: patients with vernal conjunctivitis, patients with giant papillary conjunctivitis associated with contact-lens wear, and normal subjects.

SUBJECTS AND METHODS

Subjects—Ten patients with moderate to severe classical vernal conjunctivitis[2] were selected at random. They ranged in age from 8 to 20 years with an average age of 12 years. Ten patients with giant papillary conjunctivitis associated with contact-lens wear (judged by criteria previously published[1]) were selected at random. They ranged in age from 22 to 49 years with an average age of 32 years. Five of these patients wore soft contact lenses (average length of use, two years); five wore hard contact lenses (average length of use, 14 years). We also selected ten normal persons with no ocular inflammatory diseases. The characteristics of the normal subjects were as previously described.[5]

Tissue samples—We shaved tissue off the upper tarsal conjunctiva of each subject. We used the following anesthetics: topically applied proparacaine for six patients with vernal conjunctivitis, eight patients with giant papillary conjunctivitis, and seven normal subjects; injection-block anesthesia for one patient with vernal conjunctivitis, two patients with giant papillary conjunctivitis, and one normal subject; and a general anesthetic (for unrelated reasons) for three patients with vernal conjunctivitis and two normal subjects. This project had the approval of the institution's Human Studies Committee.

The tissues were fixed as previously

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described.[5] In brief, tissues were fixed in buffered glutaraldehyde, postfixed in osmium tetroxide, and embedded in Epon. Tissues were then sectioned at 1 μ and stained with alkaline Giemsa.

Mast cell degranulation—We counted all cells with ten or more grape-colored or purple-pink granules in the cytoplasm as mast cells. We judged the mast cells to be degranulated if they met at least one of the following criteria: (1) four or more granules were larger than most granules seen in the mast cells of normal subjects; or (2) four or more granules were purple-pink (rather than deep purple or grape-colored). We counted all mast cells in one section of tissue per subject and judged each cell as granulated or degranulated. We judged the sections without knowledge of their source.

We tested the reliability of our judgments of the sizes of mast cell granules by projecting, at an arbitrary distance, photographs of 30 mast cells the granules of which we had judged large or small. The mast cell granule was then traced, and the diameter of the granule measured on the tracing. We compared the average diameter of the small granules with that of large granules.

Statistics—We used the Mann-Whitney U test to determine the significance of differences in the numbers of degranulated mast cells among the three groups tested and in the diameters of small and large mast cell granules. The level of probability considered significant was $P \leq .05$.

Results

The percentage of degranulated mast cells in the epithelium of patients with vernal conjunctivitis was not significantly different from that in the epithelium of patients with giant papillary conjunctivitis, nor were the percentages of degranulated cells in the stroma of the two groups significantly different (Table).

We found no mast cells in the epithelium of normal subjects. Tissue from the stroma of each normal subject contained fully granulated mast cells; the number of mast cells per section of this tissue ranged from three to 22. The percentage

TABLE

PERCENTAGE OF DEGRANULATED MAST CELLS

Subjects	Epithelium	Stroma	Difference Between Epithelium and Stroma
Normal (No. = 10)			
Median	0	10%	—
Range	—	0-24%	
Vernal conjunctivitis (No. = 10)			
Median	92%	81%	$P > .05$
Range	56-100%	41-100%	
Giant papillary conjunctivitis (No. = 10)			
Median	100%	83%	$P > .05$
Range	33-100%	0-100%	
Difference between normal and vernal conjunctivitis	—	$P < .001$	—
Difference between normal and giant papillary conjunctivitis	—	$P < .001$	—
Difference between vernal and giant papillary conjunctivitis	$P > .05$	$P > .05$	—

of mast cells that were degranulated ranged from 0 to 24% (Table).

Tissue from several patients with vernal conjunctivitis had no fully granulated mast cells; tissue from others had one or more in the stroma. In vernal conjunctivitis, the number of mast cells per section ranged from two to 49 in the epithelium and from 12 to 47 in the stroma.

Two persons with giant papillary conjunctivitis had no fully granulated mast cells; the eight others had one or more. In giant papillary conjunctivitis, the number of mast cells per section ranged from one to 19 in the epithelium and from five to 26 in the stroma.

Fully granulated mast cells from normal subjects, from patients with vernal conjunctivitis, and from patients with giant papillary conjunctivitis all appeared the same (Figure). Degranulated mast cells from the three groups also appeared the same, except that those from the patients with vernal conjunctivitis were more extensively degranulated than those from the other groups.

There was a significant difference between those who wore hard contact lenses and those who wore soft contact lenses. The percentage of degranulated mast cells in the stroma of the five individuals with soft contact lenses who had giant papillary conjunctivitis was significantly greater than that in the individu-

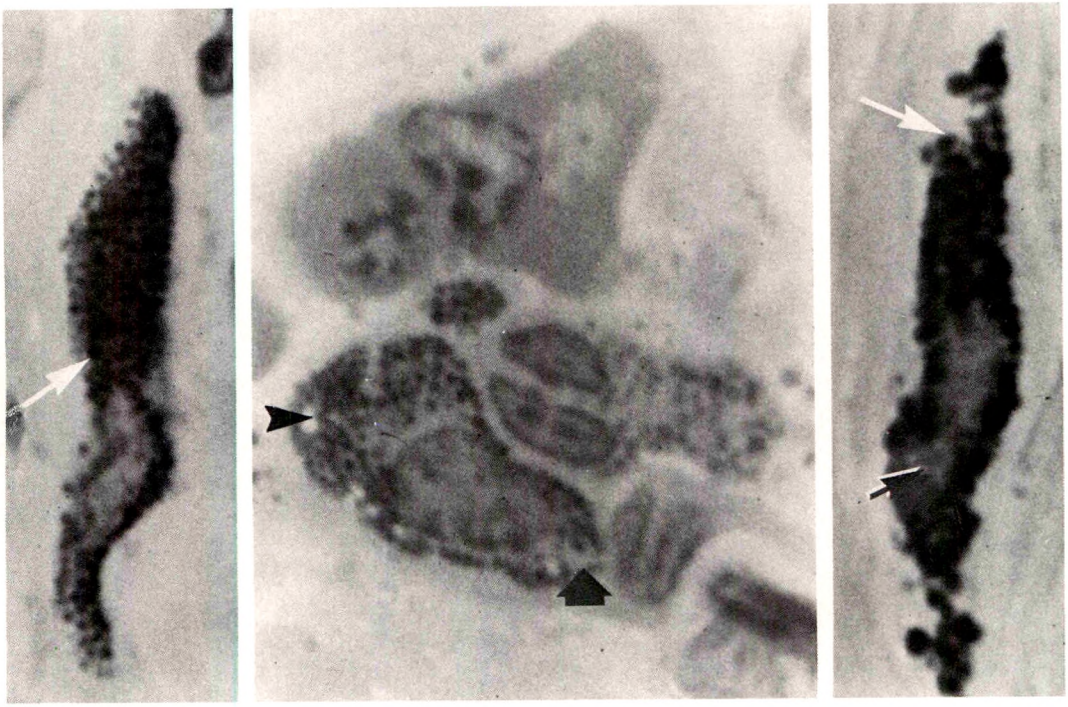


Figure (Allansmith and Baird). Mast cells of the upper tarsal conjunctiva. Left, Fully granulated mast cell. Note the many small, dark granules (white arrow). These granules stained a deep grape-purple color by alkaline Giemsa. Middle, Partially degranulated mast cell. Note the vacuole (arrowhead) and the less densely stained granule (large arrow), which was purple-pink in the section stained by alkaline Giemsa. An eosinophil is adjacent to the mast cell. Right, Degranulated mast cell. These granules are in general larger than those in the fully granulated cell shown at left. Less densely stained granules (purple-pink by Giemsa stain) are present (black arrow). Note also that deeply stained granules (grape-colored by alkaline Giemsa) are also present (white arrow).

als with hard contact lenses who had the same disease (P = .02). The percentage of epithelial degranulated mast cells in the two groups did not differ (P > .515).

In 18 of the 20 persons with mast cells in both the epithelium and the stroma, the percentage of degranulated mast cells was greater in the epithelium than in the stroma. One of the two subjects with a higher percentage of degranulated mast cells in the stroma had giant papillary conjunctivitis associated with contact-lens wear. The other had vernal conjunctivitis.

Granules considered small had an average diameter of 5 arbitrary units (± 1). Granules considered large had an average diameter of 7 units (± 1). The sizes of the two groups of granules were significantly different. Those cells judged fully granulated had only small granules. Those judged degranulated had granules of various sizes.

Discussion

The percentage of degranulated mast cells detected by light microscopy was the same for patients with vernal conjunctivitis and patients with giant papillary conjunctivitis associated with contact-lens wear; both these groups had more degranulated mast cells than did the normal subjects. Thus, the high histamine level in the tears of patients with vernal conjunctivitis was not explained by an increased percentage of degranulated mast cells detectable by light microscopy.

This finding can be explained in several ways. First, abnormal histamine pathways not present in giant papillary conjunctivitis may exist in vernal conjunctivitis. Such abnormalities might include a failure of histaminase or some other enzyme or enzymes to degrade released histamine, or a difference in control mechanisms for histamine release.[8] A second explanation is that the percentage of degranulated mast cells does differ among the three groups, but that our criteria did not demonstrate this difference. Third, some mast cells may not be observable with our method. Although light microscopy yielded similar results for the two groups of patients, many more mast cells may be present in vernal conjunctivitis, invisible by light microscopy but visible by electron microscopy. Light microscopic identification of mast cells depends on the presence of many intracellular granules, whereas electron microscopic identification depends on the presence of a few granules plus other characteristics of the cell.

Our finding that there is a greater percentage of degranulated mast cells in the stroma of patients with giant papillary conjunctivitis who wear soft contact lenses than in similar tissues of patients with the same disease who wear hard contact lenses suggests that the disease is more severe in those who wear soft contact lenses.

The greater percentage of degranulated mast cells in the epithelium than in the stroma in giant papillary conjunctivitis (P = .197) should be noted. This suggests that antigen enters from the environment and reaches the epithelial mast cells in higher concentrations than it does the stromal mast cells. An alternative explanation is that physical factors affecting mast cells promote degranulation at a higher rate in the epithelium than in the stroma.

Degranulated mast cells in tissue from all three groups appeared about the same, even though the groups are very distinct clinically. It is clear, therefore, that anaphylactic mechanisms cannot be judged by viewing a single cell. Abnormality—for example, the abnormal granulation of mast cells—can be judged by light microscopy only on a tissue basis, by calculating the percentage of degranu-

lated mast cells and comparing it with that in comparably prepared normal tissue. In the present study, the percentage of degranulated mast cells in normal tissue was clearly lower than that in tissue from patients with vernal conjunctivitis or giant papillary conjunctivitis. Our results suggest that any tissue with more than 25% of the mast cells showing degranulation, as described by the above criteria, is abnormal.

SUMMARY

Using light microscopy, we determined the percentage of granulated and degranulated mast cells in sections of tissue from ten persons with vernal conjunctivitis, ten with giant papillary conjunctivitis associated with contact-lens wear, and ten normal subjects. Tissues from both groups of patients had a significantly higher percentage of degranulated mast cells (> 80%) than did normal tissue (< 25%). The fully granulated mast cells in the three groups did not appear morphologically different, nor did the degranulated mast cells in the three groups. The percentage of degranulated mast cells in vernal conjunctivitis did not differ significantly from that in giant papillary conjunctivitis associated with contact-lens wear. The histamine level in the tears of patients with vernal conjunctivitis, which is four times higher than that of normal subjects and that of patients with giant papillary conjunctivitis associated with contact-lens wear, cannot be explained by a difference in the percentage of degranulated cells detectable by light microscopy.

ACKNOWLEDGEMENT

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SIMULATION OF MASSIVE PERIRETINAL PROLIFERATION BY AUTOTRANSPLANTATION OF RETINAL PIGMENT EPITHELIAL CELLS IN RABBITS

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In human retinal detachments, massive periretinal proliferation, the most common cause of failure, has been shown to be tissue that derives from pigment epithelial cells[1] and glial cells.[2] There was a striking similarity between the findings in humans and those in the experimental model of retinal detachment in owl monkeys.[3,4] Isolating retinal pigment epithelial cells from one eye and placing them into the vitreous cavity of the intact fellow eyes of owl monkeys demonstrated that retinal pigment epithelial cells do proliferate and exhibit metaplastic activity in the vitreous cavity.[5,6] What was still needed was the proof that pigment epithelial cells can cause vitreous and preretinal membranes. This study showed that this is indeed the case, and also that the resulting membranes are similar to those produced by fibroblast transplantation.[7]

MATERIAL AND METHODS

We used 37 pigmented rabbits, both male and female, weighing between 2.5 and 3.5 kg. After anesthetizing the rabbits with ketamine HCl (10 mg/kg of bodyweight) and injecting 1.0 ml of 2% lidocaine HCl retrobulbarly, we enucleated the left eye of each rabbit. We sutured the enucleated eye to a supporting metal ring and removed the anterior segment 4 mm posterior to the corneoscleral limbus. We also removed the lens and vitreous. The posterior segment was then filled with collagenase (1 mg/ml) and incubated at 37°C for ten minutes to loosen the retina from the retinal pigment epithelium. Using forceps, we separated the retina from the retinal pigment epithelium and cut it at the optic disk. To loosen the retinal pigment epithelium, we filled the eye cup with 0.25% trypsin and 0.02% ethylenediamine tetra-acetic acid and returned it to the incubator for ten minutes at 37°C. By applying gentle suction through a Pasteur pipette, we aspirated retinal pigment epithelium cells and collected them in a centrifuge tube containing Dulbecco's Modified Eagle Medium supplemented with 10% calf serum as a stop medium to inactivate the trypsin and ethylenediamine tetra-acetic acid. The cells were concentrated to a pellet by centrifugation of the cell suspension at 800 rpm for ten minutes.

The pellet was then resuspended in 0.15 ml of Dulbecco's phosphate-buffered saline without calcium and magnesium ions and aspirated into a tuberculin syringe through a 27½-gauge needle for injection into the recipient eye.

We slowly injected the harvested retinal pigment epithelium cell suspension (0.1 ml) into the normal fellow eye with the 27½-gauge needle at a site 5 to 6 mm posterior to the corneoscleral limbus superotemporally and directed toward the midvitreous cavity. The rabbits' pu-

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pils had been dilated previously with 2% cyclopentolate HCl and 10% phenylephrine HCl. We then withdrew the needle slowly.

The animals were examined by slit lamp and indirect ophthalmoscopy before, immediately after, and three, seven, 14, and 28 days after injection. Each time fundus photographs were taken. So that we could study pathologic changes at various stages of development, we killed some animals with an intravenous overdose of pentobarbital and enucleated their eyes three days (ten eyes), seven days (13 eyes), and 28 days (14 eyes) after autotransplantation. Two hours before enucleation, we injected 50 μc (0.05 ml) of tritiated thymidine (pH 7.3) into the midvitreous cavity. We placed the globes in cold 2% glutaraldehyde in 0.1 M cacodylate buffer. We performed a radial corneoscleral limbal incision and removed a corneal button to allow rapid access of the fixative to the eye.

In a second experiment to study the behavior of tissue-cultured retinal pigment epithelium cells in the vitreous cavity, we resuspended harvested cells in 3 ml of Dulbecco's Modified Eagle Medium with L-glucose (4,500 mg/ml), 25 mM HEPES buffer, and L-glutamine (without sodium pyruvate) supplemented with 20% fetal bovine serum with antibiotics and antimicotics. We placed the cell suspension in a 30-ml tissue culture flask and incubated it at 37°C in an atmosphere of 5% carbon dioxide in air. The medium was changed after five days and then twice weekly. When the culture was confluent, the cells were washed twice in phosphate-buffered saline and then incubated at 37° C for five minutes with 4 ml of 0.25% trypsin and 0.02% ethylenediamine tetra-acetic acid and agitated with a 5-ml pipette for another five minutes. We collected the cells in the stop medium and centrifuged them to a pellet at 800 rpm for ten minutes. The stop medium was aspirated and the cells were resuspended in 1.0 ml of phosphate-buffered saline. The cell count was made and the cell suspension of 2.5×10^6 cells/ml was prepared after another centrifugation. The cell content of the suspension was drawn into a tuberculin syringe and injected into the normal fellow eyes of the rabbits from which the cultures were derived. We used the same procedure for injection and enucleation as described previously.

After a minimum fixation time of 24 hours, we rinsed the eyes in 0.1 M cacodylate buffer and bisected them circumferentially 4 mm posterior to the corneoscleral limbus. We examined the calottes through a dissecting microscope and took gross photographs.

For histology and transmission electron microscopy, the specific structures of interest (vitreous strands, retinal puckers) were dissected from the eye, postfixed in 2% osmium tetroxide in 0.1 M cacodylate buffer, dehydrated by an ethanol series, and embedded in Epon/Araldite. We stained 1.5-μ sections with Paragon stain[8] and examined them by light microscopy. We examined 60-nm thin sections, stained with uranyl acetate and lead citrate, by transmission electron microscopy.

Specimens for scanning electron microscopy were postfixed in 2% osmium tetroxide in 0.1 M cacodylate buffer and dehydrated in a graded ethanol/freon series and dried in a carbon dioxide critical point apparatus. We then mounted the specimens on brass stubs, coated them with gold-palladium in a Hummer-Sputter device, and examined them in the scanning electron microscope.

For autoradiographic evaluation, specimens treated with tritiated thymidine were embedded in paraffin, sectioned (5 μ), and coated with nuclear track emulsion (Kodak NTB-2). The emulsion was

exposed for four days at 4°C and developed at 18°C. We counterstained the sections with hematoxylin and examined them by light microscopy. Autoradiography was done on day 7 and day 28 as previous experiments had shown these to be key days.[7]

Ten eyes served as controls. They were injected with 0.1 ml of phosphate-buffered saline, observed for 28 days, and then enucleated and fixed as described above.

RESULTS

Clinical observations—Immediately after injection of the retinal pigment epithelium suspension, a semitranslucent black-brown cloud with ill-defined borders was seen in the vitreous cavity. This cloud was always connected to the entrance wound. Within hours veils, strands, or both began to form.

On the third day after injection we examined 37 eyes. The anterior segment showed a small reaction of cell and flare. In 27 eyes, undulating arcuate sheets of gray-black cells appeared as vitreous veils, or well-defined strands occurred. They were directed toward and adherent to the medullary rays or optic disk. The vitreous was hazy. There was fluffy white and pigmented debris on the inferior retina. The area of contact between the strand and medullary ray was surrounded by slight hyperemia and tortuosity of the fine vessels. Strands were connected to the injection site in 17 eyes and were in contact with the posterior lens surface in five eyes. One eye had a traction detachment (Fig. 1). Focal deposits of pigment epithelial cells localized to one area of the medullary ray occurred in ten eyes.

Seven days after autotransplantation, we examined 27 eyes. The vitreous haze and the fluffy white and pigmented debris on the inferior retina were reduced. The anterior segment had quieted. The vitreous strands were present in 22 eyes and were thicker, more taut, and longer than on Day 3. They varied from single, more solid strands to ones with branches and thick veils. The area of contact between the strand and the medullary ray still appeared hyperemic and the tortuos-

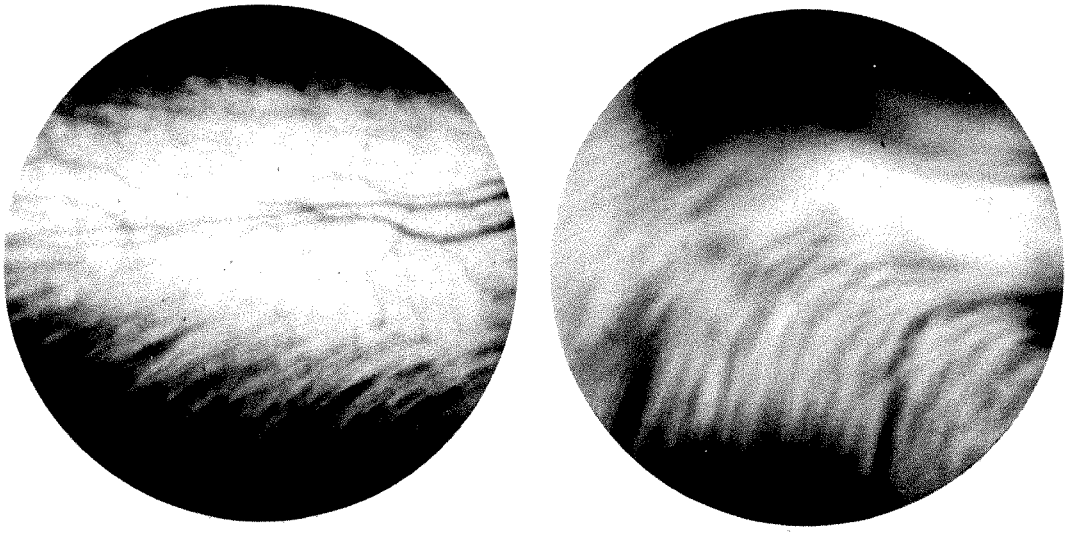


Fig. 1 (Radtke and associates). Left, Clear vitreous and medullary ray before injection of retinal pigment epithelium cells. Right, Day 3. Vitreous strand connected to the medullary ray, causing a traction retinal detachment.

ity of the fine vessels had increased. Strands were connected to the injection site in 15 eyes and were in contact with the posterior lens surface in five eyes. Ten eyes had developed traction detachments. The areas of focal deposits of pigmented cells on the medullary rays showed signs of contraction. Engorgement and tortuosity of the fine vessels with distortion and a crater-like effect of the fine fibers of the medullary ray were defined as puckers (Fig. 2).

Observations at 14 days (14 eyes) showed that white and pigmented debris on the inferior retina had decreased. The anterior segments remained quiet. There was evidence of contraction and thickening of the strands. The tortuosity of the vessels at the attachment sites had increased but the hyperemia had decreased. Strands were connected to the injection site in eight eyes, the posterior surface of the lens in four eyes, and with the retina in ten eyes. Eight eyes had traction detachments that were higher than they were on Day 7. Two eyes had developed traction detachments that were not present on Day 7. Three eyes showed pucker formation only, and two eyes showed a combination of puckers and strand attachment at the medullary rays. The puckers continued to show increased signs of contraction.

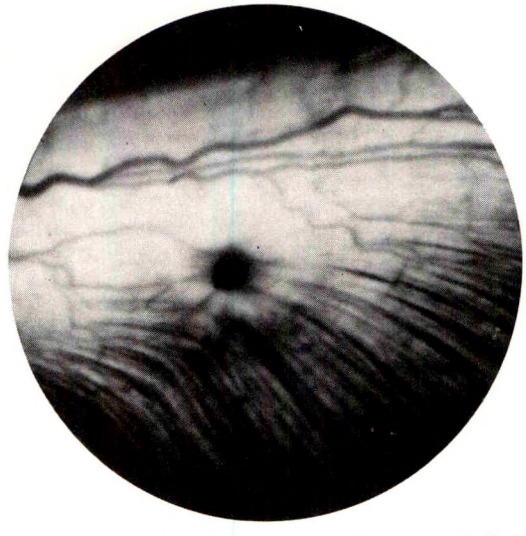


Fig. 2 (Radtke and associates). Day 7. Medullary ray with pucker.

Twenty-eight days after autotransplantation (14 eyes), the number and height of traction detachments and tortuosity of vessels at the attachment site had increased. Other than this, the findings were basically similar to those found on Day 14. Only two more traction detachments had occurred.

None of the control eyes showed pathologic changes during the four-week period.

The four eyes injected with cultured retinal pigment epithelial cells demonstrated a gray-white cloud with ill-defined edges. They showed earlier and more defined strand formation and traction detachments. Three had a traction detachment on Day 3. Neovascularization developed at the area of attachment to the medullary ray on Day 7 in one eye and the strands became pigmented on Day 14. The stabilization of activity after 14 days without much progression in the height of detachment also occurred in these animals. The vitreous and anterior chamber reaction subsided by Day 7.

Histologic studies, scanning and transmission electron microscopy, and autoradiography—The retinal pigment epithelial cells examined by light and electron microscopy immediately after harvest and before injection into the vitreous cavity were a homogeneous population of pigment epithelial cells with a few red blood cells. Examination of the callotte after retinal pigment epithelial harvest disclosed intact Bruch's membrane by serial sections with light microscopy. The harvested retinal pigment epithelial cells were intact single cells; occasionally small sheets of retinal pigment epithelial cells appeared rounded, had numerous microvilli, and contained



Fig. 3 (Radtke and associates). Freshly harvested retinal pigment epithelium cell before injection. Note abundant melanin granules and loss of cell polarity (bar = 1 μ).

abundant melanin granules. These cells exhibited loss of polarity (Fig. 3). There were free melanin granules released from damaged retinal pigment epithelial cells. Cell debris was evident.

On Day 3 the intravitreal strands showed spindle-shaped cells on the outside of the strand with their orientation parallel to its axis. They often contained non-membrane-bound melanin granules. We found suggestive myofilaments with fusiform dense bodies. There were occasional cell junctions. No collagen formation was noted. In the center of the strand the cells were less directional. There were also free melanin granules, necrotic cells, and an occasional red blood cell.

On Day 7, the strand had a more tightly packed multilayer of cells on the outer surface with an axial orientation. These cells had become thinner and more elongated (Fig. 4). Autoradiography showed high numbers of labeled cells (Fig. 5). The nuclei were spindle-shaped and there were rare convolutions. Thin bundles of myofilaments with fusiform dense bodies were frequent adjacent to the cell membrane in these spindle-shaped cells. Many cells contained only few or no melanin granules. Presumed lipofuscin granules were often present. Rough endoplasmic reticulum was dilated. There were cell-to-cell junctions (desmosomes). Collagen was interspersed between the cells. We found no basal lamina formation.

On Day 28, the elongated cells in the multilayer outer edge of the strand gave it the appearance of a tightly packed bundle. The cells were remarkably stretched. Autoradiography disclosed that the number of labeled cells was decreased compared with those on Day 7.

Occasional convoluted nuclei were seen but for the most part the nuclei retained the spindle shape. Melanin granules were sparsely scattered in each cell. Dilated, rough endoplasmic reticulum was present. We noted well-formed myofilament bundles with prominent fusiform dense bodies. Thin and elongated cellular cytoplasmic processes were embedded in a well-aligned matrix of collagen with a periodicity of 61 nm (Fig. 6). There were cell-to-cell junctions (desmosomes).



Fig. 4 (Radtke and associates). Day 7. Retinal pigment epithelium cells aligned into strand. Outer surface has a tightly packed multilayer of elongated cells and axial orientation. Note debris and pigment accumulation in the center of the strand (bar = 50 μ).

On scanning electron microscopy, a macular pucker on Day 28 showed that the surface vessels were dragged toward the center of the pucker (Fig. 7). Histologic study disclosed that the retinal surface was folded by the epiretinal fibrous membrane. The surface of the retina had a crater-like appearance at the center of the pucker, indicating a contracting force with marked distortion of the sensory retina (Fig. 8). On electron microscopy there were many cells with interlacing cytoplasmic processes containing myofilaments and fusiform dense bodies (Fig. 9).

The cultured retinal pigment epithelial cells exhibited the same characteristics as the directly harvested retinal pigment epithelial cells except for the smaller amount of pigment when examined on Day 28.

Discussion

In this experiment we were able to show that histologic and ultrastructural features of pigment epithelial cells autotransplanted into the vitreous cavity of rabbits are identical to those observed after pigment epithelial autotransplantation in owl monkey eyes.[5,6] The most pertinent similarity was the transformation of the retinal pigment epithelial cells into long spindle-shaped cells with cell junctions, oval non-membrane-bound pigment granules, and collagen formation. The new observation in this experiment was the formation of vitreous strands in contact with the retina, traction retinal detachments, and preretinal puckers after injection with retinal pigment epithelial cells. These are typical features of massive periretinal proliferation, and this model thus comes close to simulating the disease in humans.

Strand formation may be partially caused by the more solid vitreous of the rabbit compared with the rather liquid vitreous of the owl monkey. The cells simply seem to grow along the needle tract. Also, unlike owl monkey eyes, cells in rabbit eyes tend to grow towards the vascularized medullary rays and lens, forming adhesions to both and developing traction retinal detachments. We do not know why the strands grow toward the lens. Some cells settled directly on the vascularized retinal surface without maintaining connections to the vitreous.

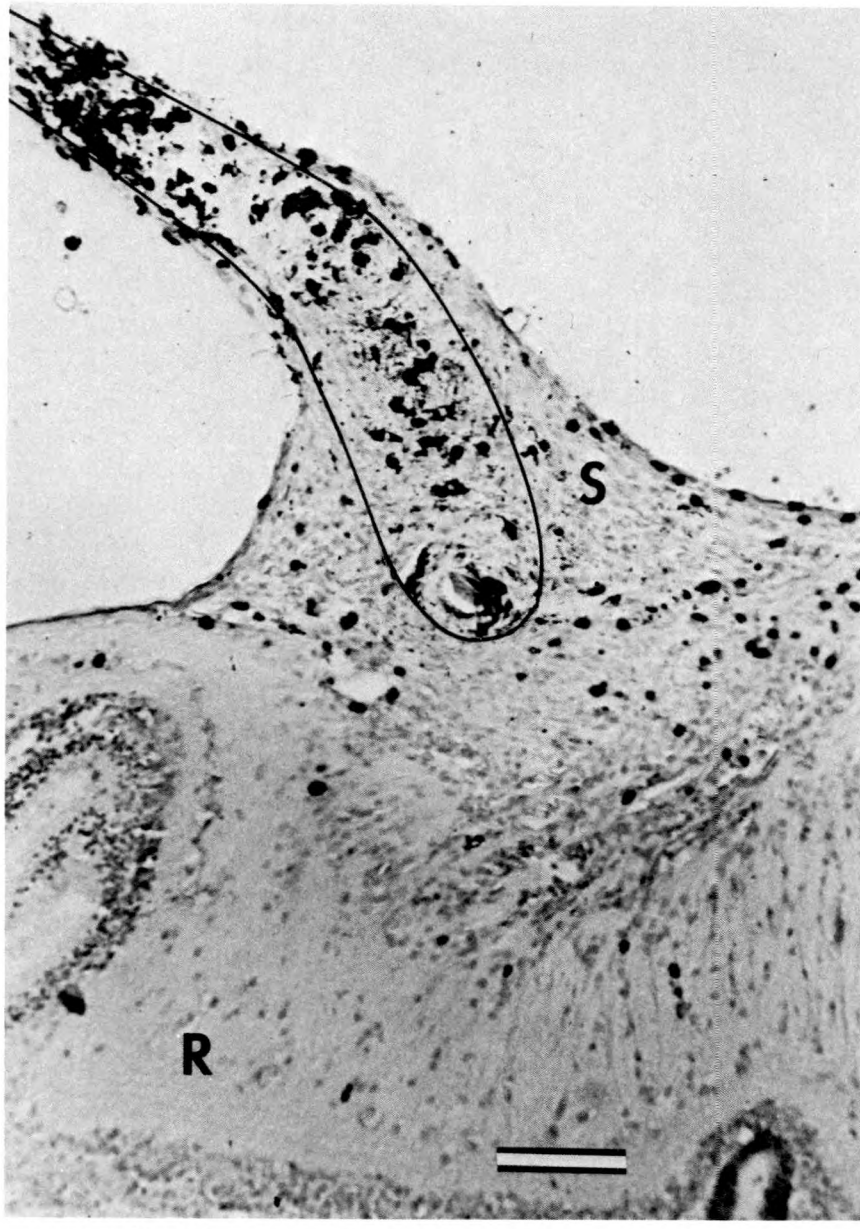


Fig. 5 (Radtke and associates). Day 7. Autoradiogram of folded retina (R) caused by traction from the strand (S). Marked labeling (black dots) after tritiated thymidine injection indicates active DNA synthesis. Outlined area contains pigmented cells in addition to labeled cells (bar = 100 μ).

These often formed contracting preretinal puckers.

We explain the development of traction detachments and puckers by the contraction of intravitreal or preretinal cells. A mechanism very similar to fibroblastic proliferation and contraction in wound repair takes place inside the eye.[9-11] The anatomic substrates most likely to account for cellular contraction are the

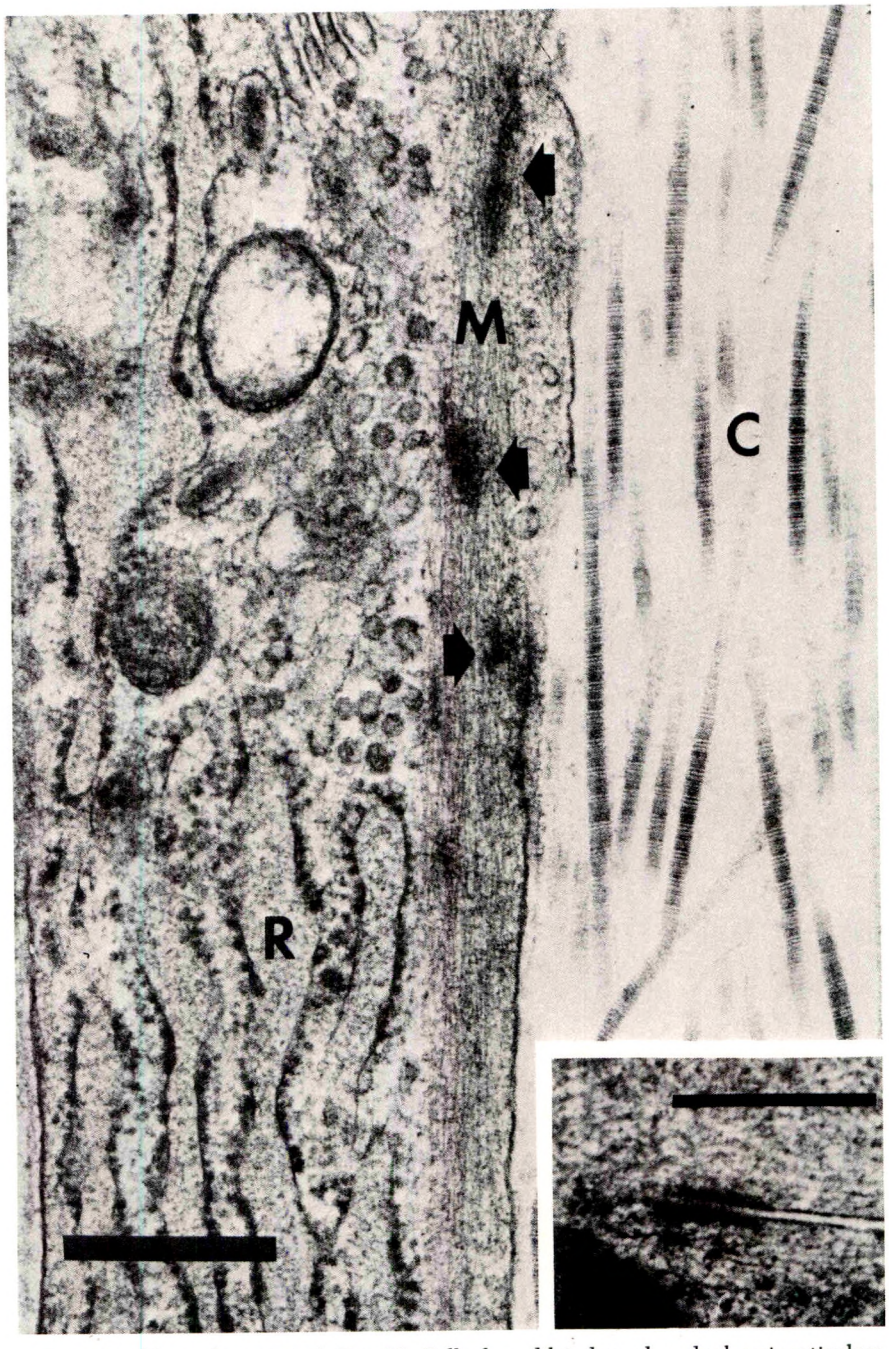


Fig. 6 (Radtke and associates). Day 28. Cells show dilated rough endoplasmic reticulum (R) and myofilaments (M) with prominent fusiform dense bodies (arrows). The surrounding collagen has a periodicity of 61 nm (bar = 0.5 μ). Inset, Desmosome from same specimen (bar = 0.5 μ).

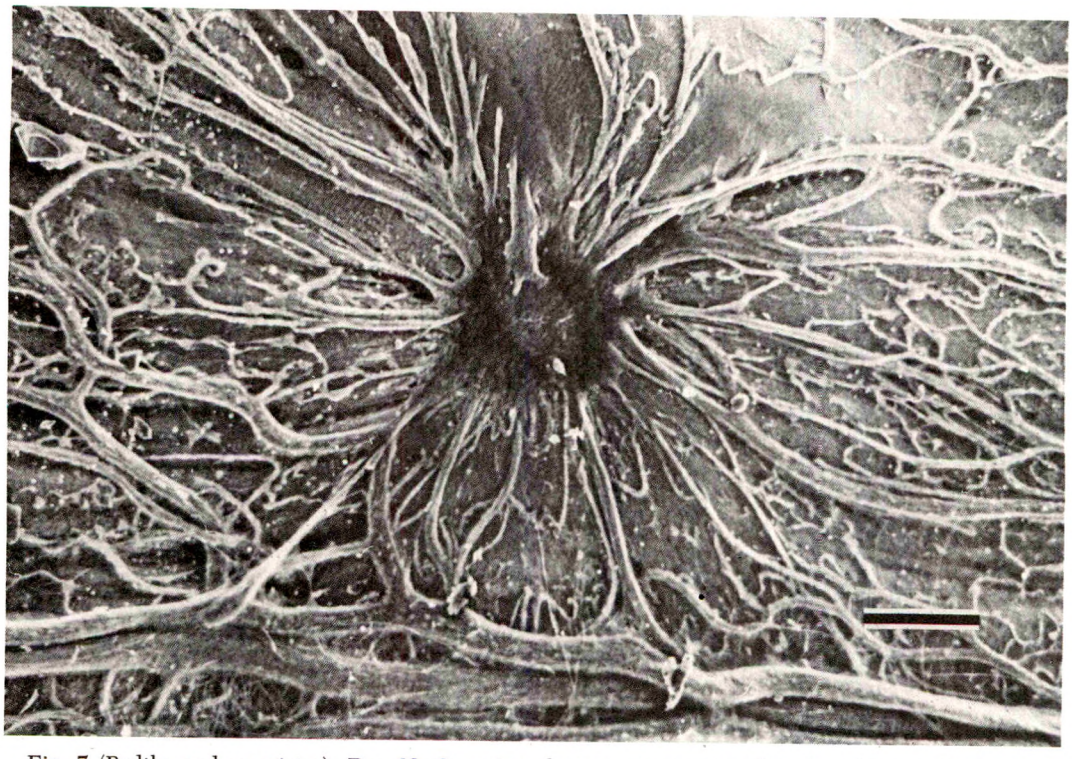


Fig. 7 (Radtke and associates). Day 28. Scanning electron microscopy of pucker shows surface vessels dragged toward the center of the pucker (bar = 100 μ).

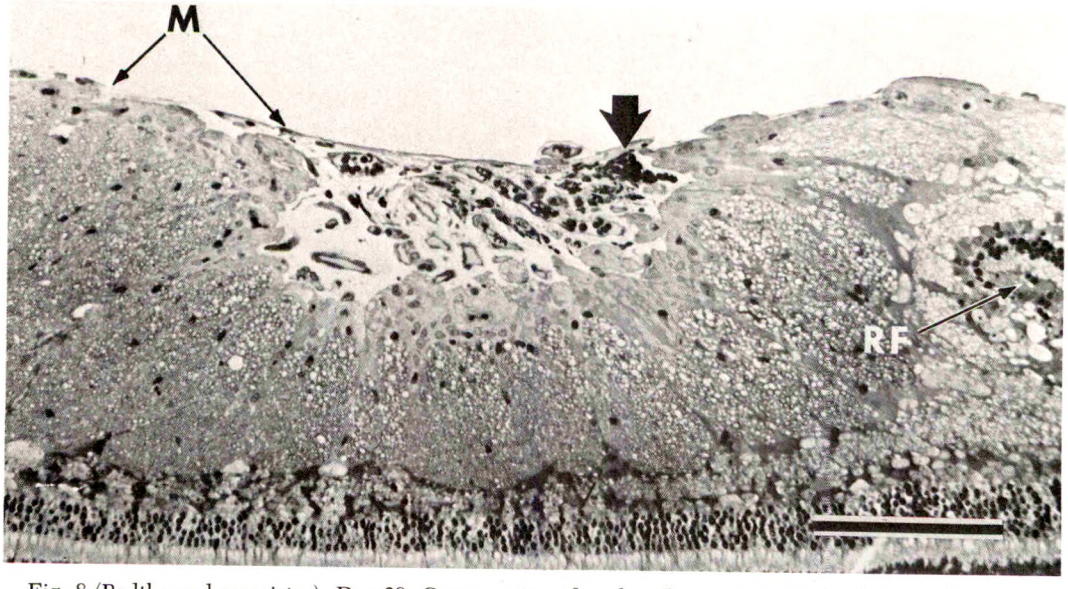


Fig. 8 (Radtke and associates). Day 28, Cross-section of pucker shows pigmented cells (arrowhead) and a membrane (M) over the surface of the retina, causing superficial layers of folded retina (RF) to be dragged toward the epicenter (bar = 100 μ).

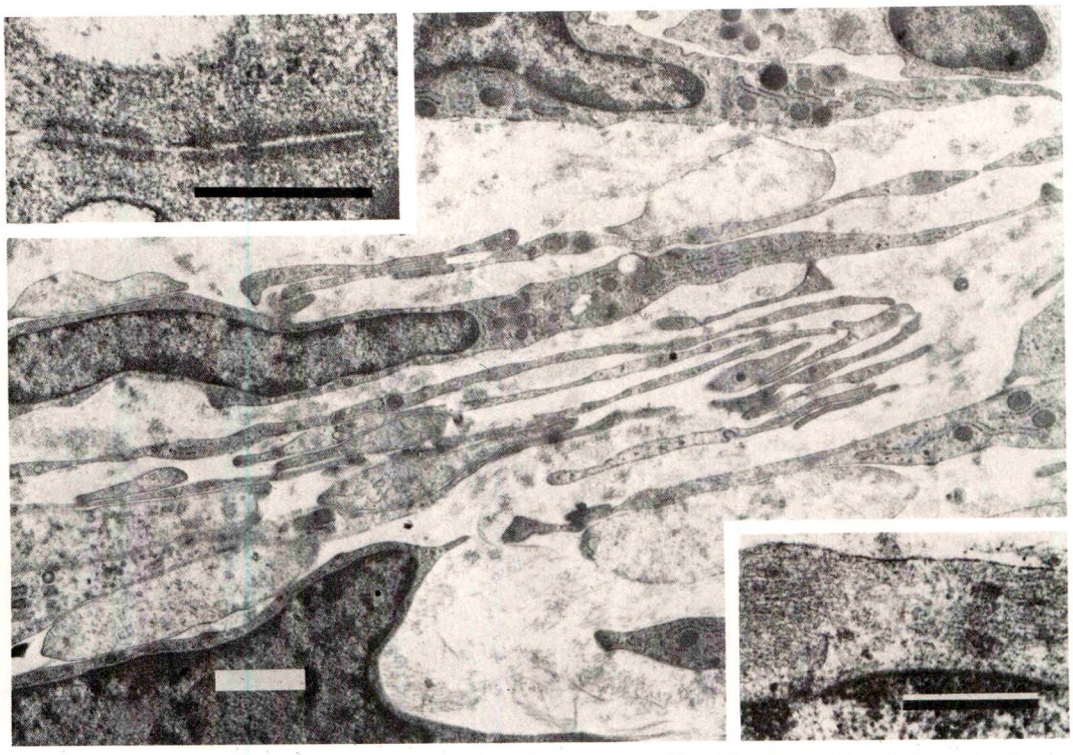


Fig. 9 (Radtke and associates). Day 28. Pucker shows many cells with adjacent interlacing cytoplasmic processes (bar = 1 μ). Upper inset, Desmosome (bar = 0.5 μ). Lower inset, Myofilaments with dense bodies from the same specimen (bar = 0.5 μ).

observed myofilaments and dense bodies.[12-14] Nuclear shape, as demonstrated by some convoluted nuclei, provides indirect evidence of contraction. Nuclear indentation and folds have been correlated with cellular contraction in smooth muscle,[15] myocardium,[16] venular endothelium,[17] and fibroblasts in Dupuytren's contracture.[18]

Increased visibility of the strands with time is caused by various factors. It appears that cells, after injection, begin to migrate to each other, as demonstrated by the transformation of the cloud into veils or strands within hours. Cell multiplication then occurs, as shown by tritiated thymidine labeling, peaking around day 7. Finally, collagen deposition accounts for the decreased transparency of the strands. Collagen formation by retinal pigment epithelial cells has been documented.[3,6,19]

We found that the pigment epithelial cells behaved like intravitreally injected fibroblasts. The retinal pigment epithelial cells also formed vitreous strands, became attached to the retina, and caused traction detachments or puckers, as has been reported after fibroblast injections into the vitreous cavity.[7] The similarity actually is so close that one can safely state that cultured pigment epithelial cells behave identically to fibroblasts provided that the same number of cells are injected into the eye. We demonstrated this in the second part of this experiment where pigment epithelial cells were first tissue-cultured to increase their

number and were then injected into the vitreous cavity. Thicker strands and more frequent and higher traction retinal detachments resulted, just as happened after injection of the same number of fibroblasts in a previous study.[7] The time available for the retinal pigment epithelial cells to metaplase into cells with fibrocyte-like characteristics during the duration of tissue culture may explain why they were more aggressive in forming strands and traction retinal detachments than were the directly harvested retinal pigment epithelial cells. The cultured retinal pigment epithelial strands had less pigment than the harvested retinal pigment epithelial cells, and this loss of pigmentation in retinal pigment epithelial cultures has been observed previously.[20-22]

We considered the possibility of contamination by cells from other sources. We thought that some cells might be introduced by perforating the sclera but the control experiment demonstrated that this did not produce strands and that the needle wound was clean. Gloor[23] has shown that bone marrow macrophages may be involved in membrane formation. We cannot completely exclude the possibility of contamination by other cells in every case. However, we examined three samples of harvested cells by light light microscopy. Except for occasional red blood cells, we could not identify other cell types. We believed that the cells involved in our strands and puckers did not originate from connective tissue or blood elements. This conclusion was supported by the results of a previous experiment[6] in which retinal pigment epithelial cell autotransplants were placed in intravitreal millipore chambers impermeable to cells so that no cells could enter or leave the chamber. Transformation of these retinal pigment epithelial cells into fibroblast-like cells was observed.

The great similarity in the clinical and anatomic findings for pigment epithelial cells and fibroblasts led us to believe that animal experiments on ways of reducing intraocular proliferation can be carried out more easily and just as effectively with tissue-cultured fibroblasts[7] rather than retinal pigment epithelial cells.

Summary

We compared the clinical pattern, the histologic findings, and the electron microscopic appearance of lesions produced by the autotransplantation into rabbit vitreous of freshly harvested and tissue-cultured (250,000) retinal pigment epithelial cells. Both harvested and cultured cells formed intravitreal strands, traction detachments, and retinal puckers, resembling the most prominent features of massive periretinal proliferation in humans. Strands produced by both types of retinal pigment epithelial cells contained cells resembling myofibroblasts which may explain the contractability of the strand. The clinical, histologic, and electron microscopic observations were similar to those found in eyes injected with tissue-cultured skin fibroblasts.

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A CLINICOPATHOLOGIC STUDY OF FOUR CASES OF PRIMARY OPEN-ANGLE GLAUCOMA COMPARED TO NORMAL EYES

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We reviewed the histologic findings concerning the normal drainage angle in both the young and the aged eye. We then compared these normal findings to the changes found in eight eyes from four patients who had clinical open-angle glaucoma. Only a few whole eyes from patients with documented open-angle glaucoma have been examined histologically. Little information, therefore, is available on structural alterations in the aqueous drainage area. Our results include the observation that major changes in six of the eight eyes occurred in the uveal portion of the trabecular meshwork.

MATERIAL AND METHODS

We reviewed the anatomy of normal eyes from age groups comparable to the age group of the patients with glaucomatous eyes. We divided the anterior segments of both the normal eyes and the glaucomatous eyes into four quadrants and cut meridional, pie-shaped sections from each quadrant. Each of these sections included peripheral cornea and iris, anterior chamber angle, and pars plicata of the ciliary body. Eyes from Patients 1 and 2 had been fixed in glutaraldehyde shortly after the patients died. Eyes from Patients 3 and 4 had been fixed in formaldehyde followed by glutaraldehyde and finally postfixed in Dalton's chrome osmium fixative. Sampling in Case 1 was limited to the two lower quadrants to avoid the regions of surgical intervention. The pie-shaped specimens were embedded in epoxy resin for cutting of sections 1.5 μ thick for light microscopy (stained with paraphenylenediamine) and thin sections (stained with uranyl acetate and lead citrate) for electron microscopy.

CASE REPORTS

Case 1—We obtained anterior segments of both eyes from a 71-year-old man with bilateral open-angle glaucoma.

Glaucoma had been diagnosed when he was 65 years old. When he was 66 years old, his intraocular pressure was as high as 32 mm Hg in both eyes. Outflow by tonography showed R.E.: C = 0.12, and L.E.: C = 0.11. Visual acuity was 6/4.5 (20/15) in both eyes. Both optic disks appeared normal and had no more than 20% cupping. Both visual fields were full.

Initial topical treatment included 2% pilocarpine, three times a day in both eyes, to which 2% epinephrine bitartrate was added to control intraocular pressure. Because this proved ineffective, demecarium bromide and then echothiophate iodide were tried. Finally, acetazolamide had to be added. Visual fields showed the development of a small superior Bjerrum scotoma in the right eye. When the patient was 70 years old, a filtering procedure was done in the right eye but failed to control the intraocular

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pressure. The patient continued to use acetazolamide and echothiophate iodide in both eyes. One year later, bilateral intracapsular cataract extractions were performed. The patient died two months postoperatively. Both eyes were obtained shortly after death. We took samples of the drainage angles from the inferior two quadrants of both eyes for study.

Case 2—We obtained both eyes of a 67-year-old man at autopsy. Intraocular pressures had been high in both eyes for eight years before his death. Untreated intraocular pressures as high as 27 and 31 mm Hg in the right eye and 24 and 26.5 mm Hg in the left eye had been recorded. Visual acuity was 6/4.5 (20/15) in both eyes. The angles were open gonioscopically. During the first year of treatment, a small arcuate field defect was detected in the right eye. During the next seven years, this field defect enlarged slightly. The central field of the left eye remained normal. Ten examinations of the visual fields were performed during this period. Treatment consisted of acetazolamide, 125 mg four times a day, and 2% pilocarpine, three times a day, in the right eye. No topical therapy was ever prescribed for the left eye.

Case 3—We obtained both eyes of a 67-year-old man with a ten-year history of open-angle glaucoma in both eyes at autopsy. He died from an intracerebral hemorrhage secondary to acute myelomonocytic leukemia.

At the patient's last examination, visual acuity was no light perception in the right eye, which had been blind for two years from glaucoma, and 6/9 (20/30) in the left eye. Intraocular pressures, measured during treatment, were R.E.: 17.3 mm Hg and L.E.: 12.2 mm Hg (Schiøtz, 5.5 g weight). Data on pretreatment pressures were not available. Gonioscopy showed wide-open angles in each eye, scattered but dense pigment clumps in the right eye, and 2+ diffuse pigment in the left eye. A cataract obscured the view of the right fundus. The left fundus appeared healthy. The visual field of the left eye was full. Medications for the right eye included 6% pilocarpine and 0.25% epinephrine, each four times a day; medications for the left eye included 1% pilocarpine four times a day and acetazolamide. The patient received intrathecally administered methotrexate for his leukemia and insulin for diabetes.

Case 4—Both eyes were removed at autopsy from a 78-year-old man who died of bronchopneumonia. He was reported to have been taking miotics for "years" and had a central field defect in his right eye only. The posterior segments were not retained or examined. Additional clinical details about the patient could not be obtained because his records could not be found.

Results

Over the years many investigators[1-8] have contributed to our understanding of the anatomy of the drainage angle; from these studies an increasingly simple and useful concept for classification of the structures in this region has evolved. The terminology and concept of the scleral roll[1] was revived, and the concept of the scleral spur as a bipartite structure closely related to the process of aging was introduced.[6,7]

The angle of the anterior chamber (the drainage angle or iridocorneoscleral angle) is formed by union of the corneoscleral coat and the uveal tract. The entrance to the drainage angle may be outlined by an arcuate line extending from the end of Descemet's membrane (anterior border ring of Schwalbe) to the abrupt peripheral termination of the anterior border layer of the iris (Fig. 1). The trabecular meshwork lies external to the arcuate line, partially fills the drainage angle, and may be divided into two main

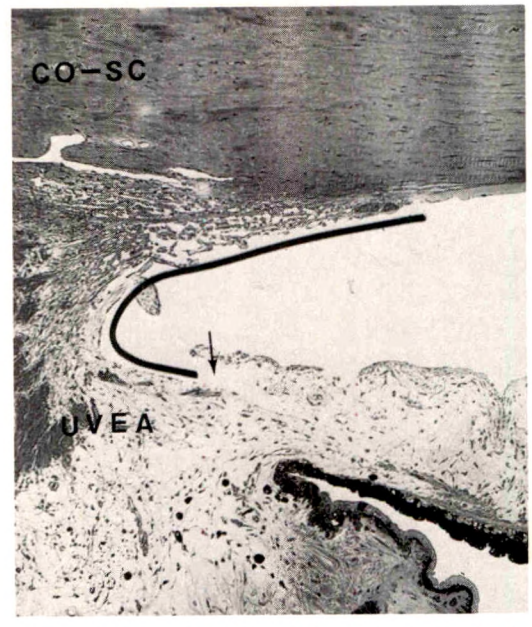


Fig. 1 (Fine, Yanoff, and Stone). Angle of the anterior chamber (of 48-year-old eye). Angle is formed by union of the corneosclera (CO-SC) with the uveal tract (UVEA). Solid line outlines the opening to the drainage angle and demarcates the angle recess. Small arrow points to a crypt in the anterior border layer of the iris near its root (paraphenylenediamine, ×55).

parts: the outer corneoscleral part that extends from cornea to sclera and the inner uveal part that lies internally to the inner scleral sulcus and is continuous with the uveal tract.[6]

In the floor of the inner scleral sulcus, against the sclera, lies the canal of Schlemm, a large vessel that is filled with aqueous humor and which closely resembles a large lymphatic channel. The vessel, or vessels, as there is frequent branching, is lined by a continuous layer of endothelial cells whose bodies protrude into the lumen. Their intercellular junctions resemble zonulae adherentes[5] and, therefore, are relatively easily separated by pressure. Similar to endothelia in other vessels, flaps and folds are present near the intercellular junctions. There are many micropinocytotic vesicles within the endothelial cells. The basement membrane of the endothelium is thin and interrupted.

Alongside and internally to Schlemm's canal, there is a tissue layer called the juxtacanalicular connective tissue,[6] which varies in extent and density according to location and age. The region was described as resembling cotton wool by Wolff,[9] called the "pore tissue" by Flocks,[10] and was labeled "endothelial meshwork" by Speakman.[11] The juxtacanalicular connective tissue contains a number of cells that lie within filamentous extracellular material and varieties of basement membrane. The adjacent, well-formed beams of the trabecular meshwork are composed of polarized endothelial cells whose apices line aqueous humor passageways and whose bases lie on a characteristic basement membrane and connective tissue matrix. The beams are arranged mostly in circumferential sheets with apertures, the transtrabecular spaces[5] (Fig. 2). The spaces lying between adjacent sheets are the intertrabecular spaces. That two distinct but related varieties of intertrabecular spaces

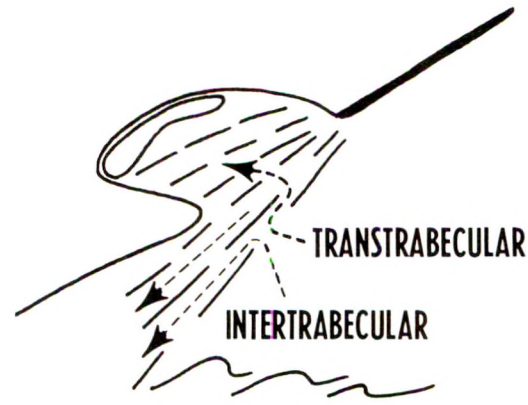


Fig. 2 (Fine, Yanoff, and Stone). Two varieties of trabecular spaces present within the trabecular meshwork as seen in meridional section.

exist was also noted by Flocks,[10] who once suggested the terms "intratrabecular" and "interlamellar" for these spaces. The transtrabecular spaces are largest in the uveal meshwork and become smaller toward the canal of Schlemm. The transtrabecular apertures appear to be randomly spaced but generally are not superimposed. The intertrabecular spaces of the uveal meshwork are large and straight and pass directly into the ciliary muscle and iris root (Figs. 1 and 2).

The corneoscleral meshwork lies within the inner scleral sulcus, extending from the cornea to the scleral roll. The scleral roll[1,5] consists primarily of bundles of collagen fibrils, arranged circumferentially around the globe, and thus differing from the more sinuous or oblique arrangements of the adjacent sclera with which it blends externally and posteriorly. Blind outpouchings from the canal of Schlemm (canaliculi or canals of Sondermann) sporadically enter the posterior corneoscleral meshwork in the vicinity of the scleral roll.

In Case 1 (increased intraocular pressures in both eyes, and a small scotoma in the right eye), severe compaction and atrophy of the ciliary muscle and its associated uveal meshwork produced an

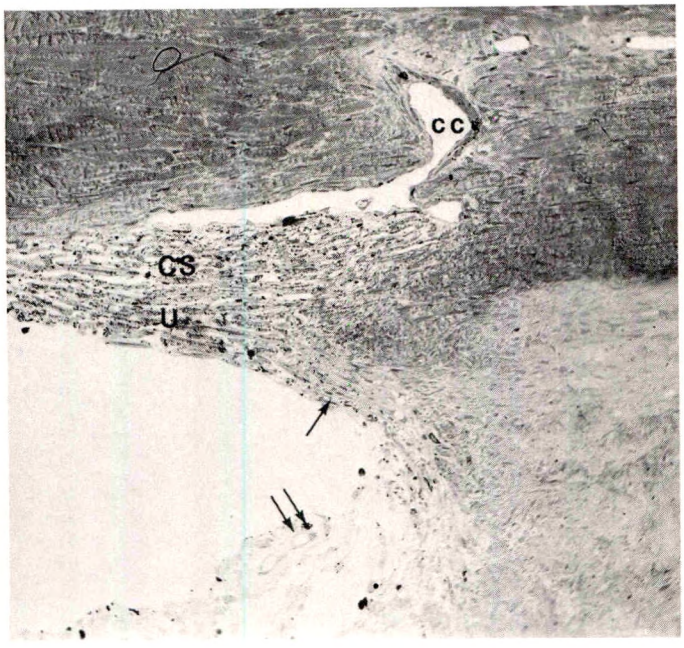


Fig. 3 (Fine, Yanoff, and Stone). Case 1. Drainage angle of right eye. Canal of Schlemm and adjacent collector channel (cc) are open. Both intertrabecular and transtrabecular spaces of the corneoscleral (CS) and uveal (U) meshwork appear to be open anteriorly. Posteriorly (single arrow) the uveal meshwork is compacted against the scleral roll, closing off the intertrabecular spaces and producing a scleral spur. Root of the iris (double arrow) is relatively normal (paraphenylenediamine, ×130).

exaggerated scleral spur upon the original scleral roll (Figs. 3 and 4), a change easily seen on examination of the intertrabecular spaces on serial frontal sectioning (Figs. 5 to 7). The canal of Schlemm, juxtacanalicular connective tissue, and anterior portions of the trabecular meshwork appeared relatively normal in most sections (Fig. 8). Some sections showed proliferations of endothelial cells or, pos-

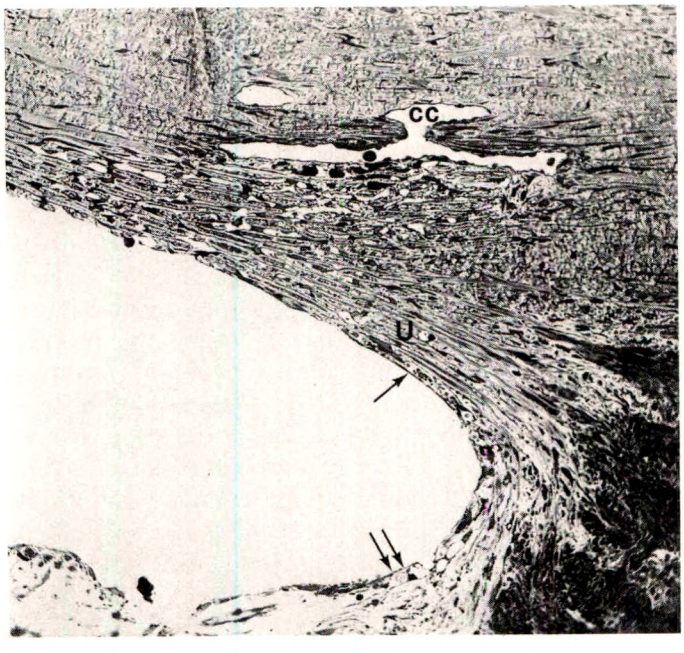


Fig. 4 (Fine, Yanoff, and Stone). Case 1. Drainage angle of left eye. Canal of Schlemm and adjacent collector channel (cc) are open. Intertrabecular and transtrabecular spaces of the corneoscleral (CS) and uveal (U) meshwork appear to be open anteriorly. Posteriorly (single arrow) the uveal meshwork is compacted against the scleral roll, closing off the intertrabecular spaces and producing a scleral spur. There is additional involvement of more anterior uveal meshwork. The root of the iris (double arrow) is relatively normal (paraphenylenediamine, ×145).

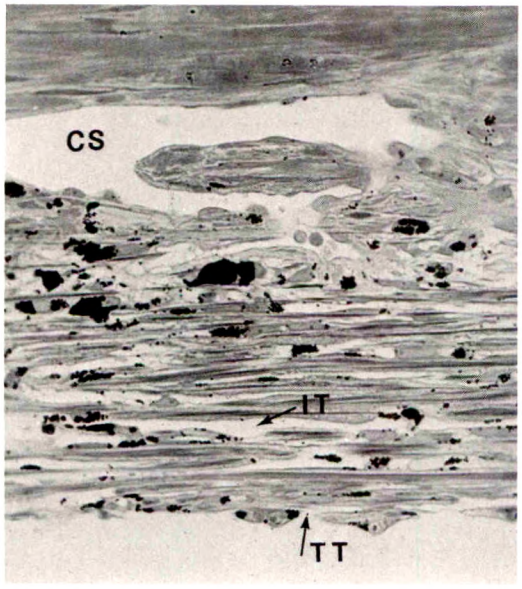


Fig. 5 (Fine, Yanoff, and Stone). Case 1. Frontal section through anterior portion of canal of Schlemm (CS) and adjacent trabecular meshwork. Both intertrabecular (IT) and transtrabecular (TT) spaces appear relatively normal (paraphenylenediamine, ×305).

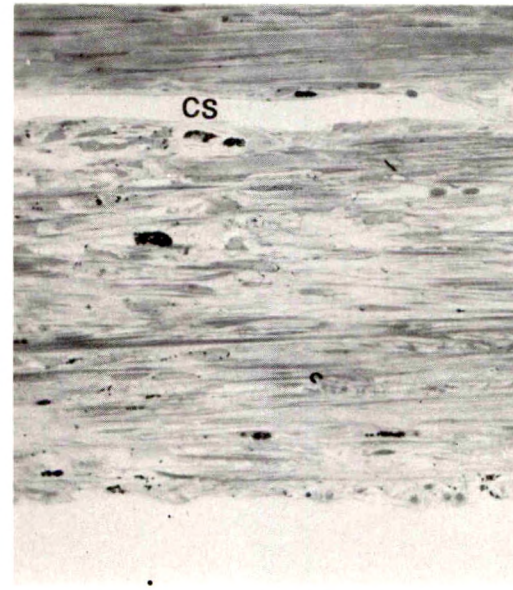


Fig. 6 (Fine, Yanoff, and Stone). Case 1. Frontal section toward posterior boundary of canal of Schlemm (CS). Note compaction of trabecular meshwork and absence of clearly defined intertrabecular and transtrabecular spaces (paraphenylenediamine, ×305).



Fig. 7 (Fine, Yanoff, and Stone). Case 1. Frontal section in region of anterior ciliary muscle shows extreme compaction and hyalinization of trabecular-muscle complex (paraphenylenediamine, ×305).

sibly, juxtacanalicular connective tissue cells, or both, into the lumen of the canal (Fig. 9). The severity of changes in the uveal portion of the drainage angle was approximately equal in the two eyes. On electron microscopy, samples of the canal of Schlemm, juxtacanalicular connective tissue (Figs. 10 and 11), and longitudinal ciliary muscle (Fig. 12) from both eyes showed changes that could not be distinguished from those of normal eyes of similar age. The most striking change was the production of large quantities of banded and other forms of basement membrane[5] in the juxtacanalicular connective tissue as well as between the cells of the ciliary muscle. There was also some proliferation of trabecular endothelial cells into the intertrabecular spaces.

In aging tissues, it is often difficult to separate clearly production of a poorly

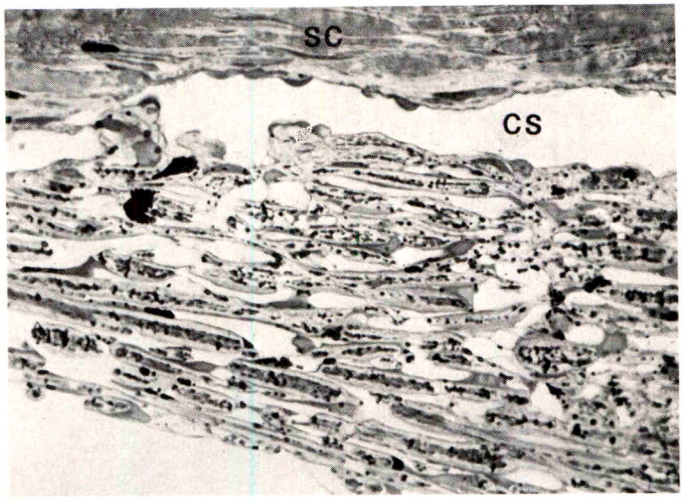


Fig. 8 (Fine, Yanoff, and Stone). Case 1. Canal of Schlemm (CS) and adjacent anterior meshwork show the relatively normal appearance common to both eyes of patient. SC, sclera (paraphenylenediamine, ×400).

formed banded basement membrane from production of elastic tissue. Indeed, there appears to be some relationship here between the two, a concept supported by the coexistence of thin basement membrane, banded basement membrane, adult collagen, and elastic tissue (both microfibrils and elastica) within the cores of normal trabecular meshwork.

In Case 2 (increased intraocular pressure in both eyes and a small scotoma in the right eye), the early changes of atrophy and compaction of the ciliary muscle and the uveal meshwork upon the scleral roll produced a scleral spur (Figs. 13 and 14). In the right eye compaction of the uveal meshwork appeared to close the posterior segment of the uveal meshwork (Fig. 13). Both eyes had mild atrophy of the iris root. Additional sections showed sporadic regions of cell proliferations within the canal of Schlemm in both eyes (Figs. 15 and 16). On electron microscopy, a sample of the canal of Schlemm, juxtacanalicular connective tissue, and longitudinal ciliary muscle from the right

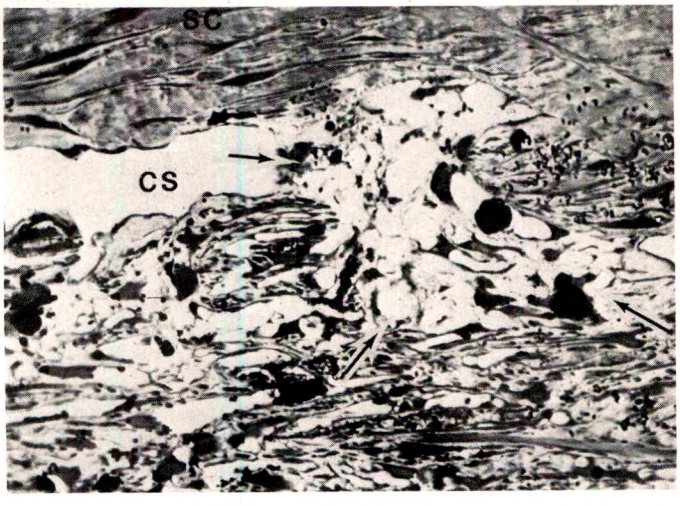


Fig. 9 (Fine, Yanoff, and Stone). Case 1. Canal of Schlemm (CS) shows region of proliferation of endothelial or juxtacanalicular connective tissue cells, or both (between arrows), into lumen, a change consistent with the age of the patient. SC, sclera (paraphenylenediamine, ×165).

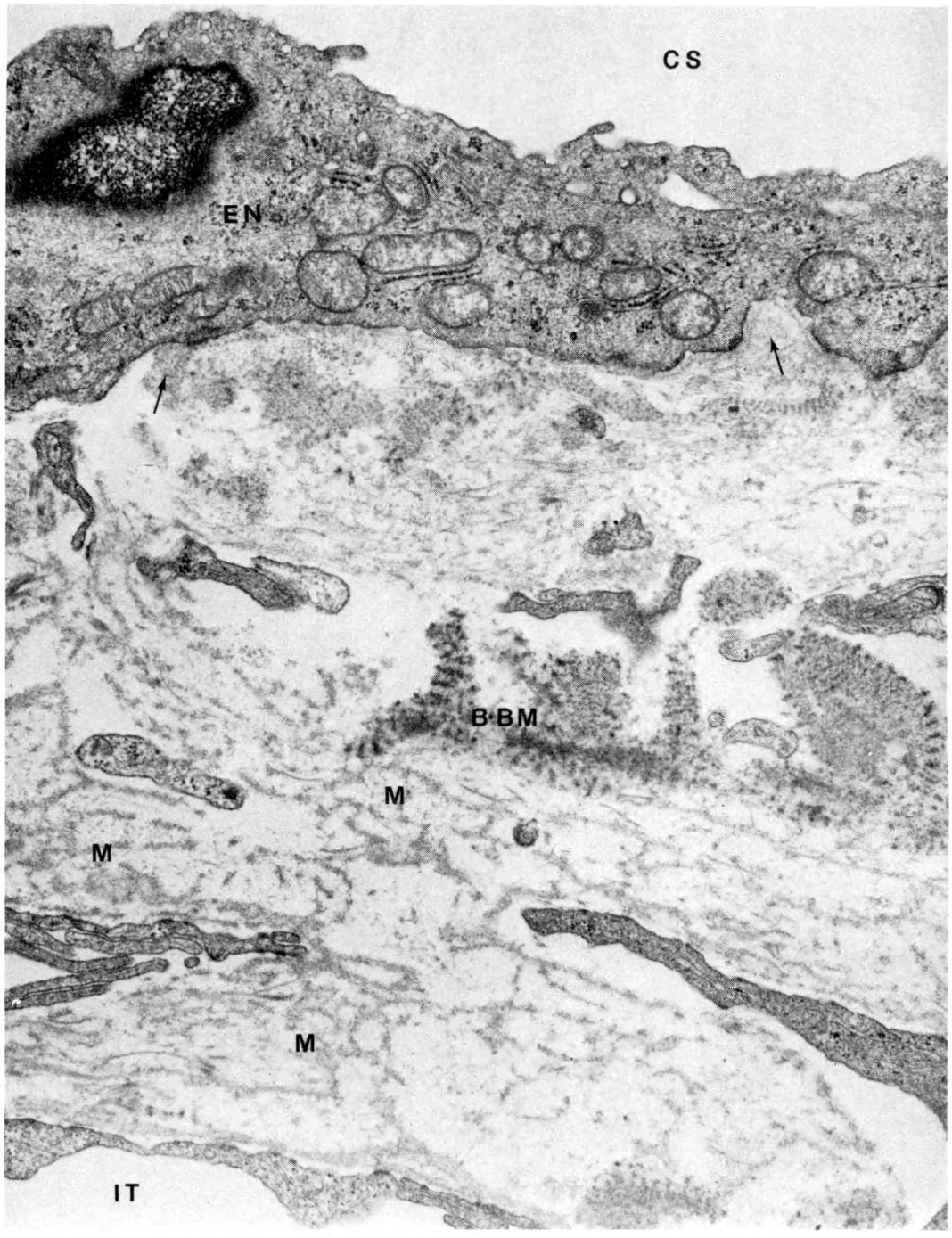


Fig. 10 (Fine, Yanoff, and Stone). Case 1. Region of juxtacanalicular connective tissue. Cells are surrounded by extracellular materials, mainly basement membranes of the multilaminar (M) or 100-nm banded (B-BM) varieties. The discontinuous thin basement membrane of the endothelial cells blends into the adjacent basement membranes in patches (arrow). The endothelial cell (EN) appears normal. IT, intertrabecular space; CS, canal of Schlemm (×17,500).

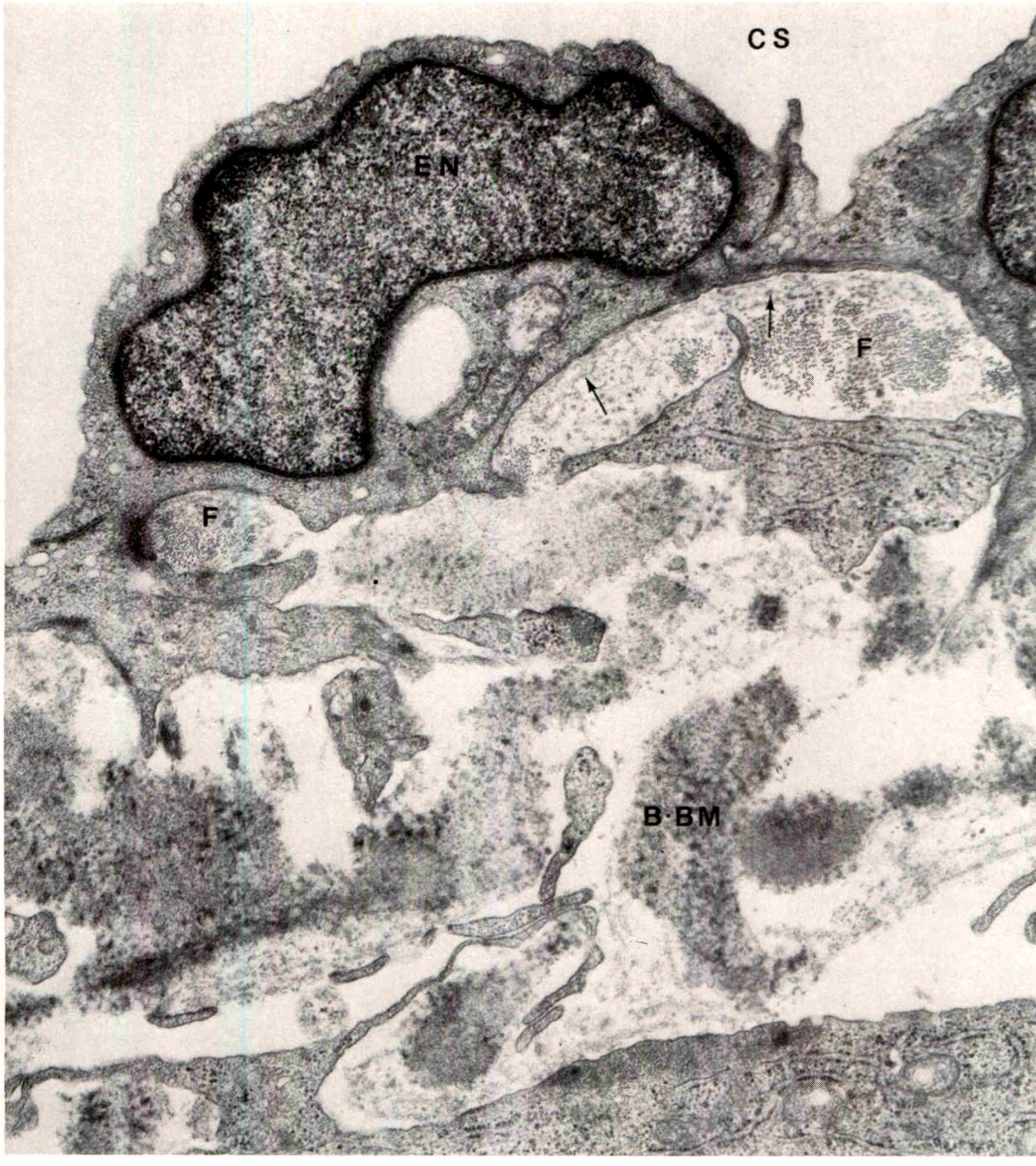


Fig. 11 (Fine, Yanoff, and Stone). Case 1. Region of juxtacanalicular connective tissue. Note patchy, thin basement membrane of endothelial cells (EN) producing a faulty, multilaminar configuration (arrows), associated delicate filaments (F), and large patches of 100-nm banded basement membrane (B-BM). A small vacuole is present in the endothelial cell cytoplasm. CS, canal of Schlemm (×17,500).

eye showed changes identical to those in Case 1.

In Case 3 (severe involvement of the right eye and mild involvement of the left eye), the optic nerve head of the right eye was severely cupped, while that of the left eye appeared normal. In the severely affected right eye, atrophy and compaction of the ciliary muscle and its uveal meshwork upon the scleral roll produced a scleral spur, almost obliterated by the severity of the ciliary muscle atrophy



Fig. 12 (Fine, Yanoff, and Stone). Case 1. Longitudinal ciliary muscle posterior to scleral spur. Intercellular spaces are filled with accumulating basement membranes of the homogeneous (H) and 100-nm banded (B-BM) varieties. A few collagen fibrils (C) are recognizable (×17,500).

(Figs. 17 and 18). The severe atrophy of the ciliary muscle produced a concavity in the face of the ciliary muscle, resembling that of the optic nerve head itself.

The canal of Schlemm varied from obliterated (Fig. 17) to open, although it was somewhat cleft-like (Fig. 18).

The left eye (Figs. 19 and 20) had

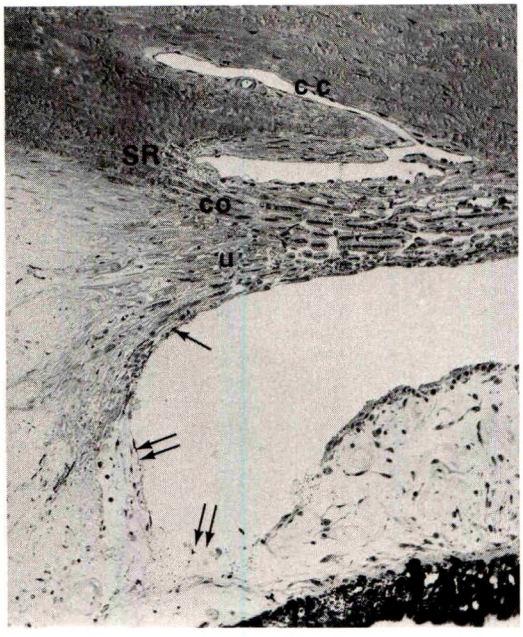


Fig. 13 (Fine, Yanoff, and Stone). Case 2. Drainage angle of right eye with small field defect. Canal of Schlemm is open, as is this particular collector channel (cc). Anterior meshwork appears open, while posteriorly the uveal (u) meshwork is markedly compacted against the scleral roll (SR), producing a scleral spur. Note additional compaction of uveal tissues along the angle recess (single arrow). Double arrows, root of iris tissue, which appears relatively normal; Co, corneoscleral meshwork (paraphenylenediamine, ×115).

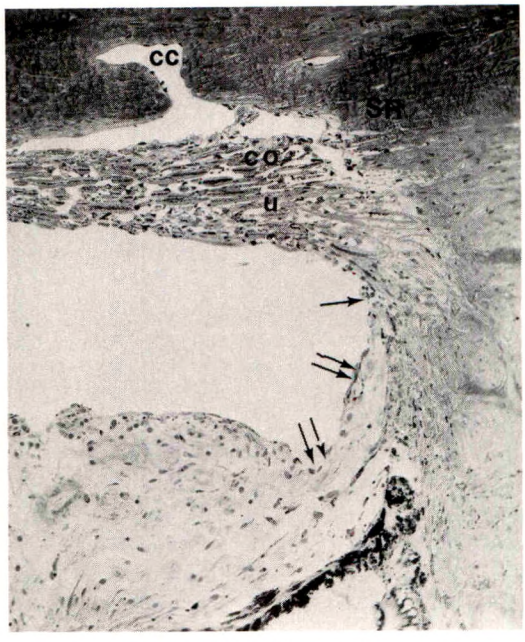


Fig. 14 (Fine, Yanoff, and Stone). Case 2. Drainage angle of left eye with increased intraocular pressure but no field defect. Canal of Schlemm is open, as is this particular collector channel (cc). The anterior meshwork appears open. Posteriorly there is only moderate compaction of the uveal (u) meshwork against the scleral roll (SR). Single arrow, region of moderate compaction of tissues, along the angle recess; double arrows, root of iris tissue, which appears relatively normal; co, corneoscleral meshwork (paraphenylenediamine, ×115).

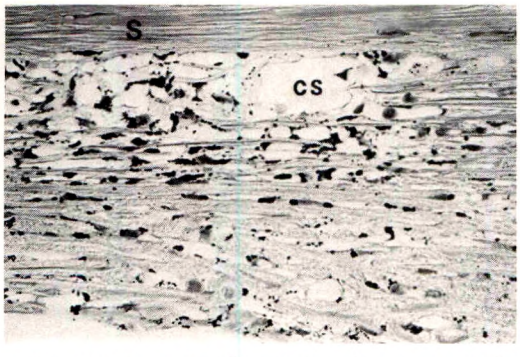


Fig. 15 (Fine, Yanoff, and Stone). Case 2. Right eye shows endothelial or juxtacanalicular connective tissue cells, or both, in the canal of Schlemm (cs) proliferating sporadically around the globe. S, sclera (paraphenylenediamine, ×250).

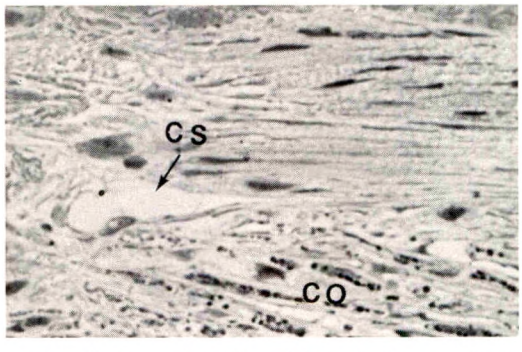


Fig. 16 (Fine, Yanoff, and Stone). Case 2. Occasional dense scarring from scleral side occluding portions of the canal of Schlemm (CS). Co, corneoscleral meshwork (paraphenylenediamine, ×575).

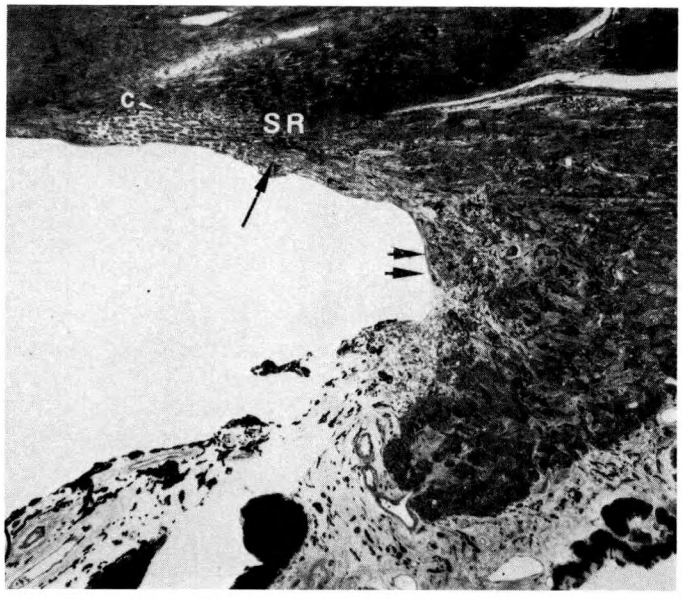


Fig. 17 (Fine, Yanoff, and Stone). Case 3. Drainage angle of the canal of Schlemm (c) and a portion of collector channel are filled with proliferated cells, but the anterior meshwork appears moderately open. Posteriorly the uveal meshwork is severely compacted (arrow) against the scleral roll (SR); also, there is severe atrophy of the tissues in the angle recess (double arrowheads). Root of the iris is severely atrophied (paraphenylenediamine, ×60).

similar atrophy of the ciliary muscle and associated uveal meshwork and a relatively unaffected optic nerve head. The posterior uveal meshwork and associated ciliary muscle region was more recognizable (that is, open) than in the right eye. Various sections showed that the canal of Schlemm was open, although cleft-like, in some areas (Fig. 19) and filled with proliferated cells in other areas (Fig. 20). The formation of a scleral spur upon the scleral roll approximated changes consistent with the age of the patient. Moderately severe atrophy of the iris root was present in both eyes. There was only mild atrophy of the temporal retinal

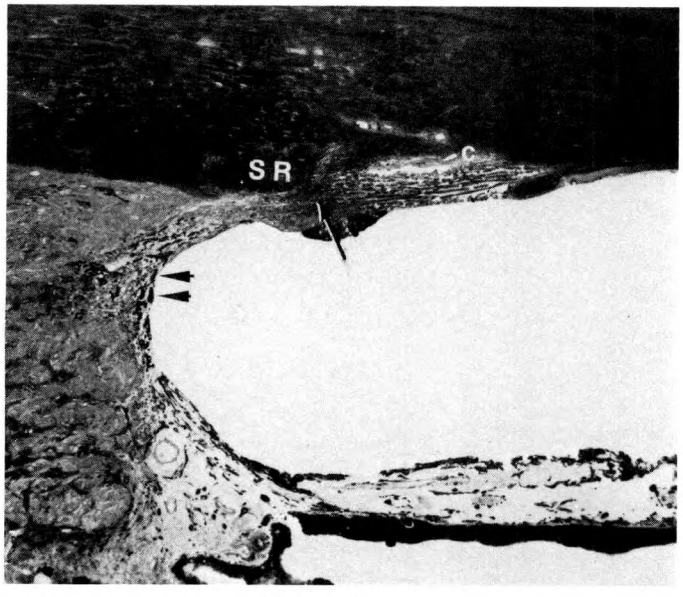


Fig. 18 (Fine, Yanoff, and Stone). Case 3. Drainage angle of right eye. The canal of Schlemm (c) is cleft-like but present. Uveal meshwork is compacted (arrow) against the scleral roll (SR) to form a prominent scleral spur. A patch of displaced pigmented neuroepithelium lies on the scleral spur. There is severe atrophy of the tissues in the angle recess (double arrowheads). Root of the iris is mildly atrophic (paraphenylenediamine, ×60).

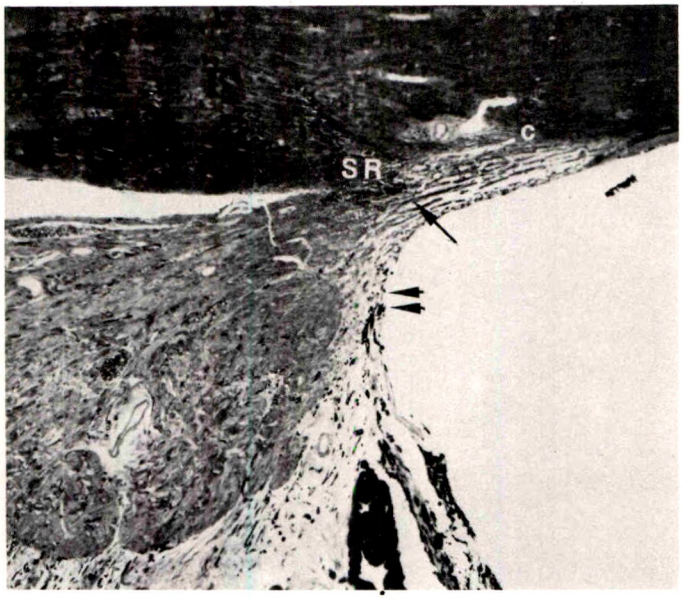


Fig. 19 (Fine, Yanoff, and Stone). Case 3. Drainage angle of left eye with increased intraocular pressure but no field defect. The canal of Schlemm (c) is cleft-like but present, and an open collector channel is present nearby. The intertrabecular spaces are open anteriorly as well as posteriorly (arrow) over the region of the scleral roll (SR). The tissues of the angle recess are mildly atrophic (double arrowheads). Root of the iris is atrophic (paraphenylenediamine, ×60).

nerve fiber layer near the optic nerve head. Many additional sections from the blocks of all meridians sampled also showed only scattered regions of closure of Schlemm's canal by proliferations or scarring, or both, most frequently in the more severely involved right eye. On electron microscopy, samples of the tissues from each eye showed large quantities of banded and other forms of basement membrane, as in Cases 1 and 2.

In Case 4 (central field defect in the right eye only), endothelial proliferation throughout much of the corneoscleral meshwork, Schlemm's canal, and associated collector channels was the most

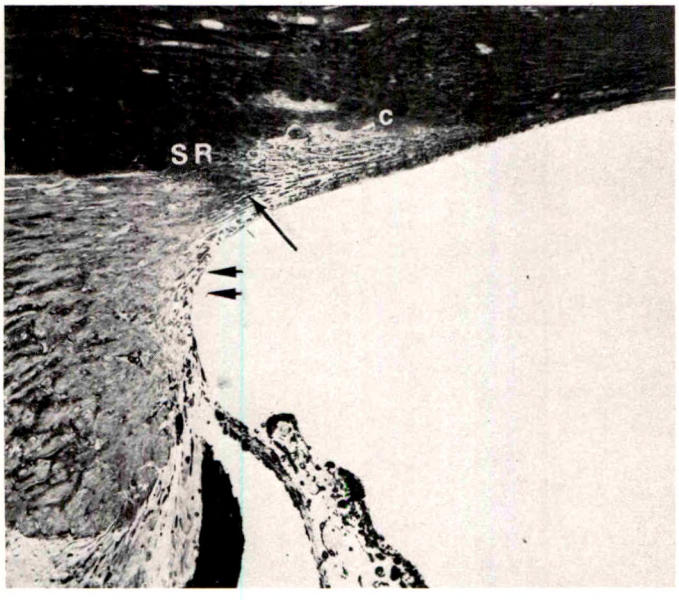


Fig. 20 (Fine, Yanoff, and Stone). Case 3. Drainage angle of left eye. The canal of Schlemm (c) is mostly filled with proliferated endothelial or juxtacanalicular cells, or both. Uveal meshwork has caused formation of a partial scleral spur (arrow) on the scleral roll (SR). There is moderate atrophy of the tissues in the angle recess (double arrowheads). The root of the iris is severely atrophic (paraphenylenediamine, ×60).

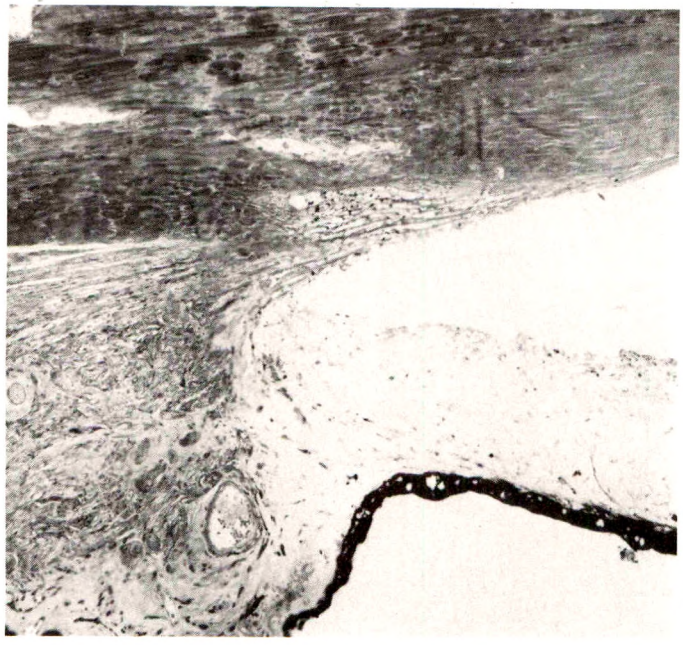


Fig. 21 (Fine, Yanoff, and Stone). Case 4. Drainage angle of right eye. Note proliferations within canal of Schlemm. Uveal meshwork is relatively open (paraphenylenediamine, ×60).

prominent finding (Figs. 21 and 22) in both eyes. The right eye (Fig. 21) also had a few areas of uveal overgrowth from the iris root onto the uveal meshwork, a change which was minimal in the left eye. In contrast to the preceding three cases, the uveal meshwork and ciliary body appeared relatively normal for the patient's age.

In none of the cases was there a continuous membrane covering the apertures of the trabecular meshwork, other

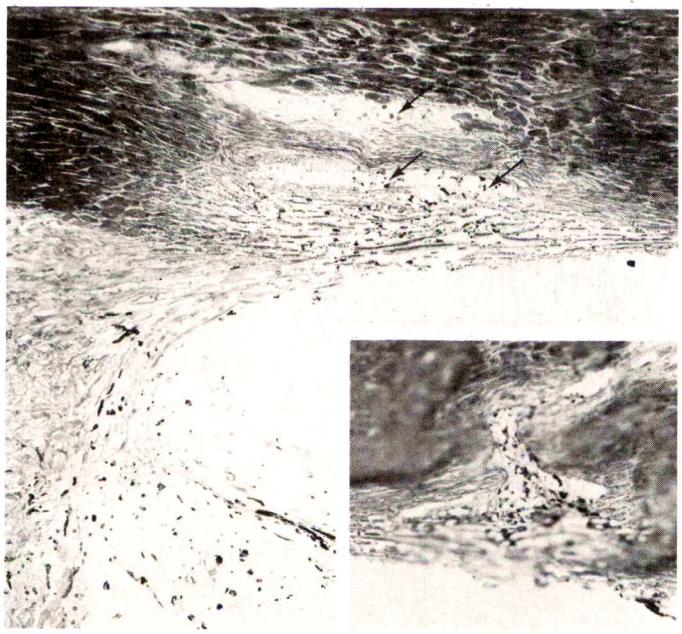


Fig. 22 (Fine, Yanoff, and Stone). Case 4. Drainage angle of left eye. Note proliferations (arrows) within canal of Schlemm and in nearby collector channel. Meshwork appears relatively open posteriorly as well as laterally. Region of scleral spur and adjacent ciliary muscle shows changes consistent with the age of patient. Inset, Continuity of proliferation within the canal of Schlemm and a collector channel (paraphenylenediamine, ×100).

than extension of some of the trabecular endothelial cells themselves.

Discussion

A histologic study of eyes with primary chronic open-angle glaucoma that tries to identify the change or changes initiating the disease process contains a number of inherent difficulties. One obvious difficulty is obtaining suitable, whole eyes from patients with adequately documented clinical histories. A second problem is that it is difficult to obtain sections truly representative of the drainage angle, a circumferential cleft that can be properly examined only by cutting true meridional sections. A third difficulty lies in the need for some form of representative sampling of the entire drainage angle (360 degrees) because of wide variations from region to region.

In our efforts to solve these problems, we tried to use only the best material available to us for study. We cut the anterior segments into small pie-shaped pieces so that all sections, whether random or serial, were as nearly meridional as possible. This differed from more conventional serial sectioning in which only a few sections through the pupil can be considered truly meridional. We cut many sections from all the small blocks available from the various meridians to enable us to assess roughly the frequency of any observation (for example, uninvolved portions of the canal of Schlemm). Finally, in order to obtain some indication of change occurring with the passage of time, we compared observations made in eyes with minimal field defects with findings in fellow eyes with no defects and in eyes with gross damage caused by long-standing glaucoma. From the data obtained in this way, we developed a theory about the pathogenesis of primary open-angle glaucoma.

A long-term increase in intraocular pressure leading to visual damage via injury to the optic nerve head—that is, glaucoma—is generally thought to be caused by an obstruction of aqueous humor outflow from the eye. The mechanism of outflow obstruction is not yet established. Many investigators, however, believe that the main site of increased resistance is in the region of the juxtacanalicular connective tissue.[12]

Aqueous humor leaves the anterior chamber primarily in the region of the drainage angle, but by several other routes as well. Part of the aqueous humor outflow occurs through the corneoscleral meshwork and the canal of Schlemm. It then enters the collector channels and the intrascleral venous plexus.[13] Part of the aqueous humor outflow also passes through the anterior uvea by several routes. After injection into the anterior chamber, coarse tracers like India ink[6] and finer tracers like ferritin[6] or fluorescein[14] penetrate the iris stroma, the iris root, the ciliary body, and the transtrabecular and intertrabecular spaces of the uveal meshwork. These tracers penetrate blood vessels of the iris and ciliary body.[6,14] The iris vessels and the inner part of the ciliary vessels leave the eye via the vortex venous system. Whether the outer part of the ciliary vessels drains into the vortex veins or into the intrascleral venous plexus and the collector channels of Schlemm's canal to reach the superficial limbal vasculature is still disputed.[15-17] A uveoscleral (that is, nonvascular) outflow pathway has also been found with radioactive albumin.[18] Such protein markers, however, may penetrate uveal vessels poorly.[19] The exact mechanism by which the anterior uvea ultimately contributes to the outflow of aqueous humor requires further experimental clarification.

Additionally, the quantity of aqueous humor leaving the human eye by uveal pathways is unknown. Experimental studies in primate eyes suggest that be-

tween 10%[19] and, perhaps, 20% to 35%[18] of total outflow may occur by uveal routes. Bill and Phillips[20] determined uveoscleral outflow in diseased human eyes by injecting them with radioactive albumin before surgical enucleation and analyzing radioactive activity after the enucleation. Quantitative conclusions about the significance of uveal outflow in normal or in glaucomatous eyes of humans cannot be made from these experiments. Perhaps 75% of a resistance to flow can be demonstrated experimentally by incision through the trabecular meshwork into the canal of Schlemm in a perfused enucleated eye.[21,22] This resistance, however, may not represent the entire, or even the most important, resistance to outflow in a normal, living human eye.

In general, a progressive increase in resistance to aqueous humor outflow from the anterior chamber develops with aging, along with a concomitant decrease in aqueous humor production.[23-25] In approximately 1% of the population, an imbalance between increased resistance to aqueous humor outflow and decreased aqueous humor production occurs, and intraocular pressure rises. In susceptible individuals, the damage to the optic nerve and visual field characteristic of chronic open-angle glaucoma develops.[12]

In order to examine the slowly progressive obstruction of aqueous humor outflow that occurs in many aging eyes, it is necessary to compare all portions of the drainage angle in both young and aged eyes. Two obvious changes occur in the drainage angle as eyes get older. First, a scleral spur forms from the scleral roll,[5,6] by collapse and accretion of uveal meshwork, together with hyalinization or atrophy, or both, of the ciliary muscle. Second, endothelial and, possibly, juxtacanalicular connective tissue cells proliferate into the lumen of the canal of Schlemm.[26-28] Additional alterations consist of increasing densification within the juxtacanalicular connective tissue itself and scarring or atrophy at the root of the iris. When such aging changes become excessive or so localized to one portion of the drainage angle as to greatly exceed normal aging changes, a suspicion of glaucomatous change can be raised on histologic grounds alone.

The four cases discussed here illustrated a possible clinical spectrum of chronic open-angle glaucoma, ranging from very early to end-stage disease.

In the first two cases, the earliest or mildest histologic changes of chronic open-angle glaucoma were localized to the uveal portion of the drainage angle. The changes consisted of compaction of the uveal meshwork and formation of a prominent scleral spur upon the original scleral roll, together with "hyalinization" or atrophy, or both, of the outermost part of the ciliary muscle and associated uveal meshwork—the uveal drainage area. The canal of Schlemm, the adjacent portions of the trabecular meshwork, and the anterior trabecular meshwork all appeared relatively normal in most sections. Occasionally, however, some sections from these early cases showed mild proliferation of canalicular endothelium into the lumen of Schlemm's canal. Such proliferations are assumed to interfere with flow and, therefore, are presumed to be obstructive. In the third case, the severely affected eye (Figs. 17 and 18) showed marked atrophy of the uveal meshwork and adjacent ciliary muscle and atrophy of the iris root. Obliteration of the canal of Schlemm was extensive but variable. The fellow, less affected eye showed less extensive involvement of the canal of Schlemm, the uveal meshwork, and the ciliary muscle. The iris root, however, was as atrophic as in the opposite eye. This case illustrated the quanti-

tative aspect to be considered when evaluating aging changes within the drainage angle that produce glaucoma.

Like glaucoma, normal aging also results in the development of a scleral spur from the scleral roll and in cellular proliferations from the inner wall of Schlemm's canal. The light microscopy findings reported here in the glaucomatous eyes, however, illustrated changes far more marked than those found in typical aged eyes. Quantitation of structural changes by electron microscopy was even more difficult, as the changes observed in such minute samplings of juxtacanalicular connective tissue (Figs. 10 and 11) and ciliary muscle (Fig. 12) did not differ significantly from those observed in normal eyes of similar age.

The first three cases of chronic open-angle glaucoma reported here indicate that significant histologic changes can occur in the uveal outflow pathways before significant histologic abnormalities in Schlemm's canal and the adjacent juxtacanalicular trabecular meshwork appear. In these cases the more severely affected eye could often be identified by the histologic abnormalities in the uveal outflow pathway. All three patients were receiving pilocarpine therapy at the time of death. Short-term experiments show that pilocarpine opens the intertrabecular spaces, especially of the uveal meshwork.[29] To our knowledge, however, there are no data on the histologic effects of prolonged administration of pilocarpine. Because of this limitation in our current understanding, it is not possible to state to what extent the observations of compaction and hyalinization of the uveal meshwork and ciliary body represent drug effects and to what extent an exaggerated aging change. The present cases emphasized the need to study the histologic effects of prolonged administration of drugs.

Case 4 illustrated a different histologic pattern. Proliferation of the cells forming the endothelial lining of Schlemm's canal was marked, suggesting functional occlusion of the canal and even of a number of collector channels. Conversely, the anterior uveal structures, including the scleral spur, appeared consistent with the age of the patient. Our observations on the canal of Schlemm accord with those previously published by Teng, Paton, and Katzin[27] and those of Flocks,[28] who took many samplings of the canal from each eye. Unfortunately, these authors were not concerned with the uveal meshwork and, therefore, we could not evaluate the meshwork retrospectively. Additionally, since the drug history in Case 4 is not completely known, the histologic findings cannot help answer the question of possible secondary drug effects.

It can also be argued that the histologic changes described here were secondary to increased intraocular pressure. If so, however, secondary changes would probably have occurred in a more diffuse and nonselective fashion. The variability of the changes found here, both in degree and in distribution, argued against their being secondary. In Case 4, in particular, there were far-advanced changes in Schlemm's canal, but the uveal meshwork was consistent with the patient's age. If the changes described here were secondary, Case 4 would have shown a histologic pattern similar to those of the other cases. Cases 1 and 2 had minimal involvement of the canal of Schlemm.

Histologic quantitation is difficult because the changes may differ in sections taken from different regions of an individual eye, whether normally aging or pathologic. Therefore, extensive sampling of properly oriented meridional sections is required for adequate assessment of the histologic findings. For example, in Case 3, although Figure 17 shows

a loss of the canal of Schlemm, many additional sections showed its presence (Fig. 18). In this context, the study of isolated surgical specimens of the trabecular meshwork and Schlemm's canal clearly is too limited an approach. The limitations are even more restrictive for the smaller tissue samples examined by electron microscopy.

There remain significant uncertainties about the mechanisms of aqueous humor outflow and the relative contributions of the corneoscleral meshwork-Schlemm's canal pathway and the uveal pathway in both the normal and the abnormal eye. Unfortunately, histologic studies cannot readily answer all our questions about the amount of aqueous humor outflow. Nevertheless, the histologic findings in our four cases may require us to revise our current ideas in regard to anatomic localization for outflow resistance in chronic open-angle glaucoma.

On the basis of our findings, the histologic abnormalities in primary open-angle glaucoma consist of exaggerated aging changes, ranging in a spectrum from excessive involvement of the uveal pathway to excessive involvement of the canal of Schlemm pathway. In one of our four cases, the findings were prominent in the area of Schlemm's canal. In the other three cases, the exaggerated changes occurred primarily within the iris root, uveal meshwork, and anterior ciliary body. Comparing fellow and involved eyes from patients with asymmetric clinical disease suggests that the uveal outflow pathway represents the earliest site of histologic abnormality in some individuals who have primary chronic open-angle glaucoma. In such patients extensive damage to the canal of Schlemm and its associated structures may cause additional changes that further occlude major portions of the circumference of the canal of Schlemm. Although the degree of functional impairment associated with these histologic findings cannot be fully determined from such morphologic studies, any theory concerning the pathophysiology of primary open-angle glaucoma should address the question of uveal outflow as well as outflow through Schlemm's canal.

Summary

Eight eyes obtained at autopsy from four patients who had chronic open-angle glaucoma were compared to eyes with normal aging changes. Three cases were characterized by early and pronounced abnormalities in the uveal portion of the drainage angle. The findings consisted of formation of an exaggerated scleral spur upon the scleral roll by accretion and compaction of the overlying uveal meshwork, hyalinization and atrophy of the adjacent ciliary muscle, and atrophy of the iris root. The fourth case showed widespread proliferation of endothelium into the lumen of Schlemm's canal. We concluded that the predominant histologic finding in eyes with open-angle glaucoma consists of an exaggeration of normal aging processes, ranging in a spectrum from excessive involvement of the uveal pathway to excessive involvement of the canal of Schlemm pathway.

Acknowledgement

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SURGICAL CORRECTION OF LENS DISLOCATION IN CHILDREN

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Surgical treatment of nontraumatic lens dislocation is controversial. Jarrett[1] and Jensen and Cross[2-5] found that visual acuity improved postoperatively in less than 60% of patients with Marfan's syndrome or homocystinuria. Several reports[1-7] record a high proportion of operative and postoperative complications. Duke-Elder[8] advised against surgery whenever possible. Other investigators reported good postoperative results and few complications.[9,10]

However, it is difficult to compare these results because of the variety of surgical techniques, causes, patients' ages, and indications for surgery. Therefore, we reviewed the procedures used in surgical treatment of lens dislocation and the results obtained here from 1968 to 1979.

SUBJECTS AND METHODS

We reviewed the records of all patients with ectopia lentis who were aged 16 years or less and who had undergone surgical lens removal by one of us (J.S.C.). We included in the study only those whose preoperative and postoperative visual acuities had been recorded and who had been observed for at least six months after surgery.

In 30 eyes, the surgeon treated lens dislocation with the irrigation and aspiration technique (Figure). Using an operating microscope, the surgeon made a tract through the inferior cornea into the anterior chamber with a Ziegler knife blade stained with methylene blue. He passed a 25-gauge irrigating cannula through the tract into the anterior chamber. He prepared a small corneoscleral limbus-based flap 180 degrees away from the dislocation and cut the equator of the lens through the corneoscleral limbus with a sweeping motion of a Ziegler knife. As the knife was withdrawn, the corneoscleral limbal incision was enlarged slightly. He placed an 18-gauge bent needle in the lens equator through the incision, injected a small amount of normal saline into the lens substance, and aspirated the lens material. Irrigation was provided as needed to maintain the anterior chamber. When most of the lens material (including the part subluxated to the far periphery) had been removed, the needle was withdrawn. He closed the corneoscleral limbal wound with one 7.0 Dexon suture and the conjunctiva with a continuous 6.0 collagen suture. Atropine 1% ointment and an ointment composed of chloramphenicol, hydrocortisone acetate, and polymixin B sulfate (Ophthocort) were applied.

In three eyes, the surgeon removed the lens with the Keeler/SITE instrument. The procedure is essentially the same as aspiration and irrigation except that the SITE probe is used instead of the 18-gauge needle and the lens capsule is removed. The wound is closed as in the irrigation and aspiration technique.

RESULTS

The mean age of patients who underwent the aspiration and irrigation procedure was 6.8 years (range, 4 to 14 years) and the average length of follow-up was 3.8 years (range, one to 12 years). Of

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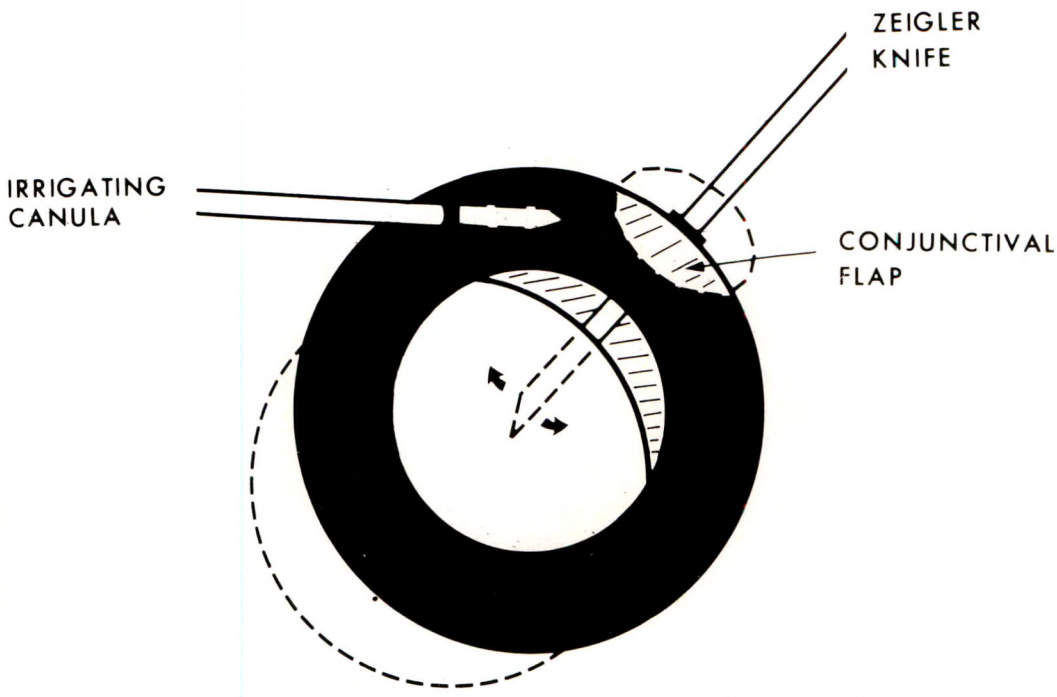


Figure (Seetner and Crawford). Surgical technique of lens removal.

these 30 eyes, 16 were in patients with Marfan's syndrome (Table 1). There were no cases of trauma or syphilis.

Poor visual acuity, usually caused by lens displacement and tilt, was the most common preoperative complication (Table 2). The best preoperative corrected visual acuity was 6/21 (20/70) (Table 3). In many of these eyes refraction was difficult because of radial astigmatism. There was severe myopia (usually lenticular) in 16 eyes, strabismus in three, and amblyopia in two.

TABLE 1

CAUSES OF DISLOCATED LENSES IN STUDY POPULATION

Diagnosis	No. of Eyes
Marfan's syndrome	16
Idiopathic dislocation	7
Homocystinuria	4
Ectopia lentis et pupillae	2
Hereditary dislocation	1

Subluxation had occurred within the pupillary area between the vitreous and iris in 29 lenses; in one it occurred in the anterior chamber and resulted in pupillary block glaucoma and corneal touch syndrome. Of the 16 eyes in patients with Marfan's syndrome, the lenses were subluxated superiorly in 14. We found no pattern of dislocation in patients with other diagnoses, possibly because their numbers were too small.

Surgery was performed to improve

TABLE 2

PREOPERATIVE COMPLICATIONS IN STUDY POPULATION

Sign	No. of Eyes
Visual acuity ≤6/21 (20/70)	30
Myopia ≥ 6 diopters	16
Strabismus	3
Amblyopia	2
Astigmatism ≥2 diopters	2
Anisometropia ≥ 2 diopters	2
Pupillary block glaucoma	1

TABLE 3

PREOPERATIVE AND POSTOPERATIVE CORRECTED VISUAL ACUITIES IN STUDY POPULATION

Visual Acuities	No. of Eyes	
	Preoperatively	Postoperatively
6/6-6/12 (20/20-20/40)	0	22
6/15-6/18 (20/50-20/60)	0	2
6/21-6/24 (20/70-20/80)	7	2
6/30-6/60 (20/100-20/200)	11	2
6/90-C.F.* (20/300-C.F.)	12	2

*C.F. = counting fingers at 2 feet.

visual acuity in 29 eyes and to relieve the pupillary block glaucoma in one. The only operative or anesthetic complication was loss of vitreous in one eye. Postoperative complications included posterior membranes requiring discission (four eyes), amblyopia (three eyes), and retinal detachment (one eye).

Postoperative corrected visual acuities were 6/18 (20/60) or better in 24 eyes (Table 3). Visual acuity had improved in 29 eyes and remained the same in one. Postoperative refractive error was recorded in 27 eyes; in 21, aphakic correction was ≥6 diopters (spherical equivalent). Astigmatism was <1.25 diopters in 26 eyes and 2 diopters in one eye. It was not unusual for a preoperative refraction error of −9 diopters to change to +9 diopters postoperatively.

In all three eyes treated with the Keeler/SITE instrument, postoperative visual acuity improved (Table 4) and no operative or postoperative complication occurred. The follow-up period ranged from 6 to 30 months.

TABLE 4

RESULTS IN PATIENTS WHO UNDERWENT SITE LENS REMOVAL

Case No.	Visual Acuities	
	Preoperative	Postoperative
1	6/60 (20/200)	6/21 (20/70)
2	6/60 (20/200)	6/12 (20/40)
3	6/18 (20/60)	6/12 (20/40)

DISCUSSION

According to many reports, the surgical removal of dislocated lenses often fails to improve visual acuity because of the frequency of operative and postoperative complications. In traumatic cases, associated ocular damage may also affect visual acuity.

In a series of 114 cases, Jarrett[1] found that visual acuity improved postoperatively in only 46 (40%), including 15 of 26 (58%) eyes in patients with Marfan's syndrome or homocystinuria. In the various series reported by Jensen and Cross,[2-5] visual acuity improved postoperatively in 27 of 47 (57%) affected eyes of patients with Marfan's syndrome. In these and other studies[6,7] operative and postoperative complications were frequent; vitreous loss occurred in 10% to 40% of cases and retinal detachment was common.

In our series, visual acuity improved in 29 of 30 eyes treated with the aspiration and irrigation technique and in all three treated with the SITE vitrectomy machine. In 24 of the 30 eyes in the first group and in two of the three eyes in the second group, postoperative visual acuities were 6/18 (20/60) or better. These results confirm those of Maumenee and Ryan[9] and Boniuk.[10]

During the irrigation and aspiration procedure, we use an 18-gauge needle,

whereas for removal of the lens in congenital cataracts we use a 21-gauge needle. The dislocated lens material seems to be more viscous than the material in congenital cataracts and requires a larger needle for removal. Entering the anterior chamber 180 degrees away from the dislocated lens makes the aspiration easier, particularly aspiration of the peripheral lens material.

One advantage of using the Keeler/SITE instrument is that the posterior capsule is removed at the time of the surgery. We set the machine at "oscillate" so there is less chance of drawing in and cutting into the iris.

Postoperative posterior lens membrane was treated with needling procedures in all four cases.

Amblyopia was present postoperatively in three eyes; all had postoperative visual acuities of 6/30 (20/100) or worse; two had had strabismus and amblyopia preoperatively. In the patient in whom amblyopia developed postoperatively, visual acuity was 6/15 (20/50) in both eyes six months postoperatively when he was 5 years old. However, subsequent development of a posterior membrane in the right eye caused exotropia and amblyopia of 6/30 (20/100) despite membrane discission and patching.

Another amblyopic patient had a preoperative refractive error of R.E.: −9.00 diopters and L.E.: −4.50 diopters with visual acuity of R.E.: counting fingers at 2 feet and L.E.: 6/90 (20/300). The right eye was exotropic and amblyopic preoperatively. Postoperative acuity was R.E.: 6/120 (20/400) and L.E.: 6/12 (20/40). The preoperative anisometropia of 4.5 diopters undoubtedly contributed to the amblyopia.

The incidence of postoperative amblyopia in patients undergoing surgical correction of ectopia lentis may be greater than the 10% seen in these 33 patients. We excluded seven eyes from this series since preoperative visual acuities had not been tested because the patients were too young (range, 3 to 5 years; average, 4 years) (unpublished data). Of these seven eyes, four were amblyopic postoperatively; the diagnoses were Marfan's syndrome (five eyes) and homocystinuria (two eyes). If we had included these eyes in our series, we would have found amblyopia in seven of 37 eyes (19%).

Myopia had been noted preoperatively in many cases. In severely affected patients the myopia often progressed dramatically. In some cases increasingly unequal myopia resulted in anisometropia and amblyopia.

In the one eye with pupillary block glaucoma, postoperative visual acuity was 6/6 (20/20) after irrigation and aspiration with concurrent intravenous mannitol infusion.

There were no anesthetic complications in our series.

SUMMARY

We treated nontraumatic ectopia lentis with the irrigation and aspiration method in 30 children and the Keeler/SITE instrument in three. Preoperative visual acuities were 6/21 (20/70) or worse in all patients; postoperatively, visual acuities improved in 29 of the 30 eyes and were 6/18 (20/60) or better in 24 (80%). Amblyopia was the major preoperative and postoperative complication. Results were also good in the three eyes in which we used the Keeler/SITE instrument. The prognosis for postoperative visual acuity in nontraumatic cases of ectopia lentis is good when either the irrigation and aspiration method or the Keeler/SITE instrument is used.

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Fifty Years Ago This Month in THE JOURNAL

Readers of the Journal and especially contributors to its columns will be interested in a recent analysis of the distribution of the Journal. This analysis was made in order to determine whether geographic factors, the location of medical centers or those famous in ophthalmology, or merely population determined this distribution, and also in order to be able to say how broad a field was reached by those using the Journal as an advertising medium.

The striking fact is that the number of subscribers varied almost directly with population. New York was first, Pennsylvania second, and Illinois third, very closely followed by California. Next came Ohio. Massachusetts was sixth, with Missouri only slightly behind. Then followed Minnesota, Texas, and Michigan, with almost equal numbers. No state was without representation.

Another point of decided interest was that twenty percent of the subscribers lived outside of the United States.

Lawrence T. Post, Subscribers and Advertising
Am. J. Ophthalmol. 14:63, 1931

NOTES, CASES AND INSTRUMENTS

THE EXFOLIATION SYNDROME IN THE APHAKIC EYE

WILLIAM C. CACCAMISE, M.D.
Rochester, New York

Sugar[1] recently reported two cases of exfoliation syndrome a number of years after cataract surgery. In 1975, Radian and Radian[2] of Romania described two patients with similar findings. I have treated a fifth patient with this rare condition.

CASE REPORT

A 65-year-old man was first examined on Dec. 9, 1966. A slit-lamp examination with the pupils dilated disclosed nuclear and posterior subcapsular cataractous changes in both eyes. There was no evidence of exfoliative material in either eye. On Jan. 5, 1967, the patient underwent an uncomplicated intracapsular cataract extraction with a peripheral iridectomy on the right eye. Three months later, his best corrected visual acuity was 6/6 (20/20). On May 1, 1967, he underwent an operation with similar results on the left eye.

From 1967 through 1977 he was re-examined approximately every three years. On each occasion there was no significant change in ocular status. On July 22, 1977, a slit-lamp examination with the pupils dilated disclosed no evidence of exfoliative material. Slit-lamp gonioscopy with the Goldmann lens showed the open angle typical of the aphakic state in both eyes. There was normal pigmentation of the posterior trabeculum. Indirect ophthalmoscopic examination of the ora serrata disclosed no fundus abnormalities. Applanation tonometry demonstrated normal intraocular pressures of 16 to 18 mm Hg in each eye.

On Sept. 5, 1980, the patient returned to the office for re-examination. He had no specific ocular or visual complaint. No change in visual acuity or refractive error was evident. However, there was a striking change in both eyes on slit-lamp examination. The pupillary margin was laden with the scaly material typical of the exfoliation syndrome. The grayish flecks involved the entire circumference of the pupillary margin. In the pupillary area, the vitreous face was densely impregnated with the same dandruff-like deposits. Slit-lamp gonioscopy disclosed no change in the angle details. There was no increase in the pigmentation of the posterior trabeculum. Results of an ophthalmoscopic examination were normal. Applanation tonometry again demonstrated normal intraocular pressures of 16 to 18 mm Hg in each eye.

DISCUSSION

The exact source of the exfoliative material evident with the slit-lamp remains conjectural. Light and electron microscopic studies have been used to support conflicting opinions. The cases reported by Sugar[1] and by Radian and Radian,[2] along with the present case, conclusively indicate that there is at least one type of exfoliation syndrome in which the exfoliative material is not derived from the lens.

However, this does not preclude the possibility of two other types of the same syndrome. In one type the lens may be solely responsible for the production of the exfoliative material. Electron microscopic studies[3] and reports[4] of regression of the exfoliative material after intracapsular cataract extraction have supported this concept. Conversely, there have been reports[5,6] of an increase in the exfoliative material after intracapsular cataract extraction in an eye already manifesting the exfoliation syndrome.

There may be a third type of exfoliation syndrome in which the lens and other intraocular structures both contribute to the production of the exfoliative material. To date there have been no studies conclusively supporting or refuting the existence of this third type.

SUMMARY

A 79-year-old man developed the classical signs of exfoliation syndrome bi-

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laterally ten to 13 years after bilateral intracapsular cataract surgery. There is, therefore, at least one type of exfoliation syndrome where the lens does not produce the exfoliative material.

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OPHTHALMIC MINIATURE

Although Teddie blazed away determinedly at the fauna of the Lower Hudson Valley (the Roosevelts had taken a summer house at Dobbs Ferry), he found, to his bewilderment, that he could not hit anything. Even more puzzling was the fact that his friends, using the same gun, seemed to be able to bag the invisible: they fired into the blue blur of the sky, or the green blur of the trees, whereupon specimens mysteriously dropped out of nowhere. The truth was slow to dawn on him.

"One day they read aloud an advertisement in huge letters on a huge distant billboard, and I then realized that something was the matter, for not only was I unable to read the sign, but I could not even see the letters. I spoke of this to my father, and soon afterwards got my first pair of spectacles, which literally opened an entirely new world to me. I had no idea how beautiful the world was until I got those spectacles . . . while much of my clumsiness and awkwardness was doubtless due to general characteristics, a good deal of it was due to the fact that I could not see, and yet was wholly ignorant that I was not seeing."

Edmund Morris, *The Rise of Theodore Roosevelt*
New York, Ballantine, 1979

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YESTERDAY'S SOLUTIONS ARE TODAY'S PROBLEMS

The much publicized report of the Graduate Medical Education National Advisory Committee (GMENAC) has again focused considerable attention on the future number of physicians and other aspects of the health-care system. Beginning in the late 1950s, it was asserted that the nation would face a substantial shortage of physicians, particularly general-care physicians. Consequently, the federal government intervened by appropriating substantial amounts of federal funds to accelerate the production of physicians. This intervention has now created the latest "problem"—a surplus of physicians, some 15% by 1990 and almost 30% by the year 2000. Given the current emphasis on containment of health-care costs, some individuals contend that a surplus of physicians would further aggravate the rising cost of health-care services. This belief has been justified on the assumption that corrective market factors of supply and demand do not apply to medical services.

Ophthalmology is one of the 18 specialties and subspecialties which are projected to be in oversupply in the next decade. For certain areas of the country, this may accurately reflect the feeling of some ophthalmologists in practice; yet, at the same time, there are other areas where patients or potential patients would strongly disagree. As ophthalmology does serve as the primary medical-care specialty for the visual system and is thus uniquely qualified to provide complete eye care (medical, surgical, and refractive services), it is important to extend the availability and accessibility of ophthalmologic services to all areas of the country. Although the Academy's preliminary data on the distribution of ophthalmologists (which will be published in the coming months in "Ophthalmology") indicate that ophthalmologists are considerably more dispersed than has been alleged, there are still areas which could

benefit from more ophthalmologic services. Among the 532 functional geographic areas which the Academy has used to depict the distribution and availability of ophthalmologists, only 32 areas (with a combined population of less than 1% of the total population of the United States) are without convenient access to ophthalmologic services. However, there are another 53 areas which could be considered "shortage areas" by GMENAC's recommended criteria for this designation.* These areas would require 932 ophthalmologists more than the current number of approximately 11,500 (excluding residents) to increase the availability of medical ocular-care services to a level just above the cut-off point for this "shortage area" designation.

The suggestion that there would be few shortage areas if ophthalmologists were more evenly distributed is not necessarily justified. Although there are a number of areas for which the ratio of ophthalmologists per 100,000 population is in excess of the average for the United States, this does not necessarily indicate that these areas are oversupplied. Rather, in many, if not most, of these areas, the demand for ophthalmologic services does require this many ophthalmologists, as shown by the volume of patient visits.

There could, indeed, be a more appropriate distribution of ophthalmologists; however, this should not be interpreted to mean that there should be an equalized distribution, because the demographic characteristics of the population being served, the residents' use of ophthalmologic services, and the productivity of the physicians can markedly affect the number needed in a given area.

It is important and appropriate to assess the future supply and requirements for physicians, and GMENAC has provided an admirable beginning. But, it must be clearly understood, lest we repeat the errors of the past, that both manpower supply and requirements are characteristics of a dynamic system and the estimation of these characteristics must be reassessed periodically with updated assumptions and data. The methodology and the modeling depend upon the accuracy of those assumptions and data and can only be validated over time. The medical profession cannot and should not ignore the GMENAC results and recommendations, but neither medicine nor government should act in haste on the basis of this report. The process of more sophisticated manpower projections has begun, and now it is our task to continue this work to insure and improve health-care delivery in this country.

BRUCE E. SPIVEY

*In volume 1 (p. 34) concerning "standards for designation of areas as adequately served or underserved," there is a recommendation that: "The minimum acceptable physician to population ratio for any area in the U.S. should be 50% of the requirements estimated by GMENAC for each type of health service in the nation as a whole." For ophthalmology, the requirements estimated by GMENAC for 1990 would result in 4.8 ophthalmologists per 100,000 population. Thus, any medically functional service area with a ratio of less than 2.4 ophthalmologists per 100,000 would be designated as an ophthalmologic "shortage" area.

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

An Ophthalmologist's Reaction to His Own Intraocular Lens Implant

Editor:

Thank you for inviting me to write of my experience with an intraocular lens implantation. The results have been so good and I am so pleased with the

procedure and the skillful care I received from my surgeon friend, Richard Kratz, and from my partners that I am happy to comply.

In 1952 an eminent ophthalmologist (later identified as Alan C. Woods), anonymously wrote a description of the vicissitudes, problems, and frustrations of aphakic vision ("The adjustment to aphakia," Am. J. Ophthalmol. 35:118, 1952). After undergoing an uncomplicated, routine, bilateral cataract extraction, he described clearly and sensitively the difficulties associated with peripheral distortion, jack-in-the-box imagery, pincushion bowing, and other problems associated with anisometropic and aniseikonic amblyopia.

Insertion of an intraocular lens is not a small variation on the standard cataract operation, but a whole new concept of respecting the corneal endothelium, estimating the endothelial density with specular microscopy, making ultrasound A-scan measurements of the globe to determine the appropriate strength of the lens, and taking keratometric readings at the operating table to govern the suturing.

Some established ocular surgeons find it bothersome to relearn the subtle aspects of extracapsular technique that have not been emphasized for many years. Until the advent of phacoemulsification, extracapsular surgery was more often unplanned than planned. Indeed, the unplanned extracapsular extraction often resulted in an eye with some of the cortex and posterior capsule retained in the upper pupillary space. We learned useful vitrectomy techniques to deal with the vitreous that sometimes appeared during the intracapsular operation.

As an early microsurgeon, I observed and appraised intraocular surgery for five years in annual visits to Groningen and Terheuzen in the Netherlands. I was so impressed with the results and with the European patients' acceptance of this idea that I came home and began intraocular lens implantations in 1974. Patients were delighted. Complications for the most part were easily resolved. Endless explanations concerning monocular aphakia were almost completely eliminated. Eyes were quiet. Visual acuity was superb the day after surgery, and the thick cataract spectacle prescriptions vanished. The main problems arose in obtaining proper intraocular lenses from Europe and coping with the controversial, experimental aspects of this new procedure, not to mention the questions raised by skeptical colleagues. When I began intraocular lens surgery, I was convinced that this was the procedure I would follow if I developed a cataract and needed surgery myself.

Through most of my professional life, I have had minimal myopic refraction with a −.50-diopter correction in each eye. In the mid-1960s, my left eye developed a cataract which was imbibing water. Visual acuity remained excellent, in the 6/9 to 6/12 (20/30 to 20/40) range, but the myopia increased substantially. During a 12-year period, a difference of 7 diopters gradually developed between the two eyes. I obtained fusional stereopsis when using the microscope by adjusting the ocular eyepiece on the left side to compensate for my increasing myopia. In the fall of 1979, the anisometropia became so marked that, even with a visual acuity of 6/12 (20/40), I encountered difficulty in identifying the posterior capsule in extracapsular lens extraction.

In the fall of 1979, I decided that a cataract extraction with an intraocular lens insertion was required on my left eye. The surgery was necessary if I were to maintain my own surgical standards. The major question was whom I would ask to do the surgery. I decided to have the operation done by my old friend, Dick Kratz, whose innovative work I had observed and admired. But, like most

concerned patients, I found myself unwilling to make a firm commitment, and conflicts of one type or another kept interfering with decisive action.

Immediately after Christmas, however, I finally decided to go ahead and I telephoned California, where I reached Tom Mazzocco, Kratz's associate. He responded with great warmth, tact, wisdom, and reassurance. We scheduled the surgery for Jan. 2, 1980. We agreed that I would gather the operative surgical data because of the holiday timing. Repeated ultrascan measurements in my own office suggested that a 15-diopter intraocular lens was indicated rather than the expected 18- to 19-diopter intraocular lens. Even though I was concerned that a 5- or 6-diopter hyperopia might occur postoperatively, I concurred with the Binkhorst formula findings. Specular microscopy estimation was good. Ocular tension was normal in both eyes. The entire ocular examination indicated no abnormality except for the lenticular myopia in the left eye. My friendly internist found no problem (except for sweating palms) that would preclude surgery.

My personal reaction surprised me. Once I decided to have the surgery, I found myself disposed to cooperate to the utmost with my surgeon. I had little desire to discuss the type of lens, the procedure, intracapsular or extracapsular, capsulotomy or not, or the type of intraocular lens to be used.

As I boarded a Los Angeles-bound airplane on New Year's Day, I felt I should not present any of my own opinions. As I contemplated the upcoming ordeal and began a little introspective soul-searching, I turned on my own brand of Transcendental Meditation to help control the gastric lepidoptera.

From my side of the desk, the admission ritual at the hospital seemed pointless and tedious. The hospital room with its familiar equipment seemed Spartan. The angular metal hospital bed conspicuously lacked a Linus blanket. I was reminded of the dictum that a physician should be hospitalized at least once a year in order to gain a more humane perspective on his established practices and procedures. It seems entirely different when you are the patient and substituting bed occupancy for bedside omniscience.

My anxiety was eased enormously by the unexpected and thoroughly welcome visit from my surgeon on the evening of New Year's Day. The night passed easily with the aid of a nostrum given by a reassuring night nurse.

In the morning my senses were dulled as I rolled down a hundred miles of corridor on my back, the ceiling lights flashing by. I was greeted in a terse but friendly professional manner and was pleased that I was not preoccupied about my oxygen supply when they started to drape my face.

As the operation progressed, I was even more grateful at not being asked to follow even simple directions. The lack of conversation and the smooth progress made my apprehensions disappear, and I began to lose track of the operating sequence that I thought I knew so well. I faintly heard in the background the tapocketa-pocketa noises of the equipment, but this was not bothersome. There was even a haziness about just when the dreaded retrobulbar injection was given. The operation ended with reassuring comments, and I spent the rest of the day counting my blessings and snoozing.

The following morning, I was ecstatic to find that I could see very well indeed and that I had no discomfort, photophobia, or prickly sensations. The two eyes worked beautifully as a team with nearly equal visual acuity.

I learned that, not surprisingly, a Kratz-modified Shearing posterior chamber lens had been used, that a capsu-

lotomy had been done at the time of surgery, that a surgical keratometer had been used, and that the lens removed was spherical because of the water content. My surgeon agreed that I could leave the hospital immediately and fly home so that my partners could monitor my recovery.

The healing process was uneventful. I had no glare, dazzle, or glitter phenomena. My partners remarked that with this type of lens I was an easy patient to refract and that my peripheral fundus was easily observed. Visual acuity was 6/6 (20/20) within one week with a minimal amount of mixed astigmatism. At the end of the second week, I felt so confident about my stereopsis that I did a penetrating keratoplasty with good results. By the end of the third week I had returned to my normal surgical schedule.

I believe that with all the exciting new developments in ophthalmology, an intraocular lens will be the rule, rather than the exception, in cataract surgery. Certainly, all the problems encountered with conventional cataract surgery weigh heavily in favor of a competent intraocular lens insertion. The management of extended-wear contact lenses, after cataract surgery, is costly in time, money, and understanding, and should not be undertaken lightly.

Without correction, I enjoy binocular vision as keen as it was 15 years ago. Will I have an intraocular lens in my other eye if a cataract develops in it? No question about it.

MALCOLM A. MCCANNEL, M.D.
Minneapolis, Minnesota

Ocular Involvement in Infectious Mononucleosis

Editor:

The finding of corneal involvement in a patient with infectious mononucleosis reported by M. Pinnolis, J. P. McCulley, and J. D. Urman ("Nummular keratitis associated with infectious mononucleosis," Am. J. Ophthalmol. 89:791, 1980) is similar to that in a patient whom we recently examined.

This 16-year old girl had a five-day history of foreign-body sensation in her right eye associated with a mild fever and pharyngitis. She had no history of ocular or systemic disease and was taking no medications. Visual acuity was 6/6 (20/20) in both eyes. Slit-lamp examination of the right cornea disclosed eight small stellate "microdendrites" involving the central and peripheral cornea in all quadrants; they were composed of swollen, opaque epithelial cells which stained with Bengal rose 1%. A moderate perilimbal conjunctival hyperemia was present, and there were follicles in the inferior fornix. An ipsilateral preauricular lymph node was palpable. A physical examination showed splenomegaly.

At the initial examination, conjunctival and tear samples for virus culture were taken with a sterile cotton-tipped applicator, inoculated directly into a plastic vial containing 2SP viral transport medium, and stored at −180°C in a liquid nitrogen cannister.

The patient was treated with topical acyclovir 3% ointment, five times daily. The corneal lesions healed within two days without any residual opacity. The follicular conjunctivitis resolved after 11 days.

Epstein-Barr virus was cultured from the conjunctival and tear samples. Serum antibody titer showed heterophil antibody to be present; no antibody to human herpesvirus type 1 or 2 was detected.

Our patient with clinical and serologic evidence of infectious mononucleosis had culture-proven Epstein-Barr virus keratoconjunctivitis, substantiating previous reports based upon serologic data.

The ocular disease rapidly resolved, but her examining physician later be-

came ill with spiking fever, myalgia, vesicular glossitis, and splenomegaly. Hematologic evaluation showed lymphopenia and a positive heterophil antibody test. Special care, including thorough hand-washing and avoidance of unnecessary contact, should be taken when examining any patient with a potentially infectious ocular disease.

KIRK R. WILHELMUS, M.D.
London, England

Occlusion of the Central Retinal Vein

Editor:

In their article, "Long-term follow-up of occlusion of the central retinal vein in young adults" (Am. J. Ophthalmol. 90:190, 1980), I. A. Priluck, D. M. Robertson, and R. W. Hollenhorst point out that the occlusion of the central retinal vein in young patients can be a visually damaging disease. I believe there are a few problems with their paper.

The authors referred to some anomalous vessels on the optic disk and stated that they were most probably "collateral shunt vessels." This is something of a misnomer. Pre-existing vascular channels, which compensate for an occlusion in the adjacent area by detouring blood flow, should be called collaterals,[1] not shunts. Shunts are arteriovenous interconnections which avoid the capillary bed and in which there is rapid flow.[2] The large vascular channels seen on the optic nerve head after central retinal vein occlusion are retinal-venous to ciliary-venous connections known as collaterals.[2] Collaterals detour blood flow from the retinal circulation, around the occlusion, into the choroidal circulation via optic disk capillaries which become dilated. These are not shunt vessels.

It has been my experience that the main cause of an acute decrease in visual acuity in central retinal vein occlusion in young patients is cystoid macular edema. The decreased visual acuity can be secondary to nonperfusion, as the authors have pointed out, but this is a much more unusual abnormality. Cystoid macular edema is readily seen by slit-lamp examination and can be confirmed by fluorescein angiography.

Cystoid macular edema can result in various changes of the pigment epithelium. When severe cystoid macular edema is present, edema fluid in the confines of the retinal tissue spills over beneath the sensory retina (between the rods and cones and pigment epithelium), thus irritating the pigment epithelium. When the presence of this fluid becomes chronic, the pigment epithelium can be irreversibly damaged, thus accounting for the "macular mottling" noted by the authors. Similarly, chronic cystoid macular edema can lead to macular hole formation, also causing an irreversible decrease in visual acuity.[3]

Additionally, although the authors dealt with the natural progression of central retinal vein occlusion in young patients, they did not mention treatment. In my experience, in most cases of central retinal vein occlusion in young patients, the cystoid macular edema can be lessened or even totally resolved with systemic corticosteroids. The condition of central retinal vein occlusion seems to be an inflammatory one,[4] and seems to be self-limiting, lasting anywhere from a few months to up to two years (perhaps more). Corticosteroids can keep the disease relatively quiescent in most cases, and can protect the macula from irreversible changes.

HOWARD SCHATZ, M.D.
San Francisco, California

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Reply

We appreciate Dr. Schatz bringing a point of semantics to our attention. The anomalous vessels we described appear to be collateral vessels which function to shunt (verb) or divert blood to an alternate route. We propose that this shunting is via anomalous optic disk capillaries that serve as collaterals.

Our findings concur with Dr. Schatz's observation that cystoid maculopathy is a major cause of decreased visual acuity in young individuals with occlusion of the central retinal vein. Our retrospective study involved a relatively small number of patients who were not managed by a definitive treatment protocol. We were unable to provide statistical conclusions about final visual acuity with respect to the use of warfarin, corticosteroids, or other agents. A controlled double-masked prospective study is needed to confirm Dr. Schatz's observation that systemic corticosteroids lessen the severity of cystoid maculopathy associated with occlusion of the central retinal vein.

IRA A. PRILUCK, M.D.
DENNIS M. ROBERTSON, M.D.
ROBERT W. HOLLENHORST, M.D.
Rochester, Minnesota

Extended-Wear Contact Lenses After Corneal Grafts

Editor:

In his article, "The effect of extended-wear aphakic hydrophilic contact lenses after penetrating keratoplasty" (Am. J. Ophthalmol. 90:331, 1980), M. A. Lemp elucidated a significant problem that should be given serious consideration.

Six aphakic keratoplasty patients whom I fitted with hydrophilic contact lenses (Hydrocurve II-55) at various intervals after keratoplasty developed superficial vascularization of the cornea and mild stromal edema. One patient spontaneously developed an endothelial graft rejection; another patient developed a small *Staphylococcus aureus* corneal ulcer and the inflammation touched off an endothelial graft rejection.

In 90 patients with simple surgical aphakia whom I fitted with extended-wear Hydrocurve II-55 and Hydrocurve II contact lenses there was less vascularization and stromal edema than in these post-keratoplasty patients.

The grafted cornea appears to respond poorly to extended-wear hydrophilic contact lenses and I strongly agree with Dr. Lemp that "alternative methods of correcting visual acuity be considered in patients with aphakia who have received corneal grafts."

JOHN J. PURCELL, JR., M.D.
St. Louis, Missouri

Reply

Editor:

I was very interested in Dr. Purcell's letter relating problems he experienced in post-keratoplasty patients fitted with extended-wear hydrophilic lenses. This confirmation of my finding further strengthens the caution which I urged for clinicians in fitting extended-wear lenses in patients with grafts.

It should be remembered that currently available extended-wear lenses with a high water content, that is, 70% to 80%, can provide minimal oxygen requirements for normal corneal epithelium. Contact lenses

with a lower water content, that is, 55% (such as those fitted by Dr. Purcell) would be expected to provide even less oxygen. These lenses do not allow for ideal oxygen conditions for the corneal epithelium but merely minimal needs in the normal cornea. The grafted cornea probably has a different metabolism with different oxygen needs. Moreover, there is relatively little exchange of tears beneath a hydrophilic contact lens. This suggests that a possible build-up of metabolic products beneath the lens may further add to corneal damage in these patients.

My thanks to Dr. Purcell for sharing his experiences with us.

MICHAEL A. LEMP, M.D.
Washington, D.C.

HLA Antigens in Diabetic Retinopathy

Editor:

We were most interested in the excellent study, "Histocompatibility antigen frequencies in diabetic retinopathy" (Am. J. Ophthalmol. 90:148, 1980), by J. Barbosa, R. C. Ramsay, W. H. Knobloch, H. L. Cantrill, H. Noreen, R. King, and E. Yunis. Their main observation was a significant positive association between HLA-B15 and proliferative diabetic retinopathy.

We reported identical findings at the Tenth Congress of the International Diabetes Federation in 1979.[1] In addition to the HLA-A, -B and -C antigens, we tested the HRL-DR determinants, which are known to be primarily associated with insulin-dependent diabetes. Both under normal conditions and in type I diabetes, HLA-DR4 is genetically linked to HLA-B15. In contrast to patients with nonproliferative retinopathy, however, patients with proliferative diabetic retinopathy had a stronger association with HLA-DR4 than with HLA-B15. The relative risk for developing proliferative retinopathy was 1.3 in HLA-B15-positive and 2.6 in HLA-DR4-positive diabetic patients. Detailed results of this study are in press.[2]

JÖRG BERTRAMS, M.D.
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MANFRED SPITZNAS, M.D.
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Reply

Editor:

We are glad that Drs. Bertrams and Spitznas found an association between proliferative retinopathy and HLA antigen, a result compatible with our findings. Schernthaner and associates[1] have also reported an association between HLA-DR4 and proliferative retinopathy. These independent confirmations make sampling bias less likely. Thus, three independent groups have now shown data suggesting a genetic contribution to severe retinopathy.

Since the relative risks mentioned by Bertrams and Spitznas are low, it would be interesting to know if stratification of their patients in a way similar to that we performed would increase the strength of the association as it did in our study. As we have mentioned previously,[2] several published reports suggest a higher frequency of HLA-B15 in patients with proliferative retinopathy, but failure to stratify patients by age at diagnosis may have led to the inability to detect an association.

JOSE BARBERA, M.D.
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Organic Nitrates in Glaucoma

Editor:

The article by A. J. S. Wizemann and V. Wizemann, entitled "Organic nitrate therapy in glaucoma" (Am. J. Ophthalmol. 90:106, 1980), is an interesting and hopeful one. The results of treatment with nitroglycerine and isosorbide dinitrate appear impressive, casting organic nitrates in a favorable light.

It is interesting to note that both glycerine and isosorbide are hyperosmotic agents with well-known modes of action. Perhaps the reason these two agents appear to be useful is not their action as organic nitrates, but their established action as hyperosmotics.

The use of isosorbide in Sequels form is a more convenient mode of therapy than the 50% solution previously suggested and may represent a suitable alternative to other hyperosmotics that are tolerated less well. Only time will tell.

ARNOLD S. PRYWES, M.D.
Bethpage, New York

Reply

Editor:

The well-established pressure-lowering effects of isosorbide and glycerol are caused by their action as hyperosmotics when used in high concentrations.[1-3] As Buckley and Shields[4] commented, the similarity between the generic names of isosorbide and isorbide dinitrate may imply that organic nitrates have hyperosmotic properties.

However, the osmolarity of the nitroglycerine solution used in our study was iso-osmolar to serum (295 ± 5 mOsm/liter). One tablet containing 40 mg of isosorbide dinitrate weighs approximately 280 mg.

Osmotic diuresis was not induced by either intravenously or orally administered organic nitrates. We conclude, therefore, that doses of organic nitrates similar to those used in angina pectoris or congestive heart failure do not act as hyperosmotics. The effect of organic nitrates on intraocular tension appears to be caused by a marked vasodilatation of retinal and choroidal vessels.[5]

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BOOK REVIEWS

The History of Wills Eye Hospital. By William Tasman. New York, Harper and Row Publishers, Inc., 1980. Hardcover, 135 pages, index, 45 black and white figures. $12.50

There is a theory of human behavior which states that one special characteristic of humans is the constant search for

connection. When we are young, these connections tend to be horizontal and we relate to people in our geographic or vocational community. As we get older, these connections spread and we realize that connections can be vertical as well as horizontal.

At this point, people begin to dig into archives to find out about their ancestors. This idea appears to hold true in ophthalmology, and more and more of us are now busy delving into the origins of our specialty. Even a superficial historical review shows that the Wills Eye Hospital appeared very early in the story of American ophthalmology.

Although a history of the Wills Eye Hospital was published in 1931, it is appropriate to have this new book published in the year the latest, the largest, and the most magnificent of the three Wills Eye Hospitals was opened. This slim volume will obviously interest primarily the alumni of the institution but others will also find a great deal to interest them. For example, I found the relationship of the Wills Eye Hospital to the arts intriguing. The celebrated Maxfield Parrish, for example, was the great-nephew of one of the first four surgeons at Wills Eye Hospital, Isaac Parrish, who is credited with performing the first cataract operation. Rebecca Gratz, the model for Rebecca in Walter Scott's "Ivanhoe" was the aunt of Isaac Hayes, another of the first four surgeons appointed. John Singer Sargent, the famous painter, was the son of FitzWilliam Sargent, a not-so-well-known eye surgeon at Wills in the 1850s.

Interesting though these items may be, the chiefs who have guided the hospital through the 150 years or so of its existence are obviously more important. These men, up to and including the current chief, Thomas Duane, were remarkable men. Duane has found time not only to build a magnificent eye center but also to edit this country's most widely consulted clinical textbook of ophthalmology.

I was surprised to learn that Wills was not always the outstanding research institute that it now is. There was practically no research done at Wills until the 1940s when, to quote the authors, "Research at Wills Eye Hospital was achieved almost single-handedly by Irving Leopold." It is fascinating to read the program of the first clinical conference of 1949 and to see Dr. Leopold's name on that program. He has probably contributed to every program since then and is the only person on that original list who is still active in research today.

The book closes with pictures of the new Wills Eye Hospital, an impressive structure as I can attest from a personal tour of the facilities. This single institution devoted to diseases of the eye is larger than many general hospitals and a brief review of the associates listed at the end of this volume will give some idea of its size. There are over 300 people on the staff and Wills has trained more than 500 residents and approximately 150 fellows. What is most noteworthy is that Wills has been able to deal in these large numbers without sacrificing quality. This is a remarkable achievement.

DAVID SHOCH

Retrolental Fibroplasia. A Modern Parable. By William A. Silverman. New York, Grune and Stratton, Inc., 1980. Hardcover, 246 pages, index, 418 black and white figures. $23.50

Dr. Silverman worked in the premature nursery at the Columbia Presbyterian Medical Center during the years when retrolental fibroplasia was common. He

has written an interesting account of this period. He corresponded with many of the pediatricians and ophthalmologists who worked actively on the problem and uses quotations from them in the form of letters, tape recordings, and telephone conversations.

Silverman has annotated the original description of retrolental fibroplasia by Drs. Chandler, Verhoeff, and Terry in Boston and the notes made by Dr. Stewart H. Clifford. He discusses the identification of this condition by Owens and Owens and the early theories that premature exposure to light, vitamin E deficiency, and adrenal cortical hormone insufficiency caused retrolental fibroplasia. Silverman describes the period of the early 1950s when excess oxygen was proven to be the principal cause of retrolental fibroplasia, and shows how this finding affected oxygen treatment practices in nurseries throughout the world.

Silverman describes the oxygen treatment practices in this country from the beginning of the century to the introduction in the 1940s of methods of delivering oxygen more efficiently to the premature infant. He cites the observation that normal premature infants have a "periodic" irregular pattern of respiration when breathing air compared with more regular respirations when breathing oxygen. This influenced pediatricians to believe that added oxygen therapy would be beneficial. The observation that premature infants could be in a state of "subcyanotic anoxia" (oxygen-lack without the sign of blue skin color), was another key factor prompting the change in oxygen practices in the late 1940s. The author describes what happened after oxygen was incriminated and points out the confusion that resulted from many pediatricians assuming that oxygen administration at concentrations of less than 40% did not harm the retina. In the 1960s reports appeared concerning excessive restriction of oxygen, because of conditions such as brain damage and the increased incidence of hyaline membrane disease; it was also in this period that the severe oxygen deprivation of the infant with idiopathic respiratory distress syndrome was proven.

A considerable portion of the remainder of the text discusses other practices in the premature nursery, including the use of nebulized mist and antibiotics. A useful appendix is included. Silverman provides a general overview of the medical community's willingness to accept observations and reports without critical evaluation, and discusses the ethics of controlled clinical trials.

This book should interest investigators interested in the history of retrolental fibroplasia and in the lessons learned from this disorder.

ARNALL PATZ

Visual Optics and Refraction. A Clinical Approach. By David D. Michaels. St. Louis, C. V. Mosby Co., 1980. Hardcover, 743 pages, bibliographies, index, 383 black and white figures. $59.50

This long book is divided into three sections: basics, technique, and management.

The first section covers visual optics and visual physiology. It is well-written, lucid, and reasonably concise. It is directed to the clinician, not overburdened with mathematics, and many chapters include a discussion of clinical applications. It is full of information that will delight the clinician and teach the neophyte.

The section on technique opens with a chapter entitled "Principles of Refrac-

tion." One wonders, however, why this section includes such subjects as dyslexia, the prevalence of visual impairment, and medicolegal considerations.

The second chapter in this section, called "Symptoms," covers the art of history-taking and the clinical features of acute visual loss with and without pain, progressive loss of visual acuity, and ocular and orbital pain. Other clinical conditions, such as diplopia, photophobia, blepharospasm, itching, dryness, vertigo, and dizziness, are also discussed.

There is an excellent chapter on visual acuity that points out the inadequacy of our clinical methods of determining visual acuity, particularly visual acuity at near.

There are chapters on basic pharmacology and the techniques of the general ocular examination, including ophthalmoscopy, electroretinography, electrooculography, and electromyography. None of these, however, have any direct relationship to refraction or to the determination of the refractive error.

The chapters on retinoscopy and subjective techniques are excellent. I particularly enjoyed the chapter on subjective testing, but I am not sure that my residents would get the same pleasure from it. The optical principles of determining strength of cylinder with the cross cylinder are adequately and clearly stated. The principles of determining axis of cylinder with the cross cylinder, a subject which very few examinees at the American Board examinations understand, is covered less well.

A lengthy chapter on vision in children contains little that is relevant to the subject of visual optics or refraction.

The third section on the management of refractive errors is complete and contains little material that is not to the point.

There is an extensive bibliography at the end of each chapter, which shows the depth of research involved. It would help the reader who wished to check out specific points by additional reading if specific references were cited in the text.

This is a well-done book full of useful information and I found little with which to disagree. Because Michaels includes so much information that is not directly related to the subject announced in the title, perhaps he should consider changing the title to reflect this.

DANIEL SNYDACKER

The Demography of Blindness Throughout the World. By Hyman Goldstein. New York, American Foundation for the Blind, Inc., 1980. Paperbound, 324 pages, references, 38 tables. $10

With his considerable experience and long-standing interest in blindness, Dr. Goldstein has produced a detailed description of the various methods by which data on blindness can be collected, and the limitations inherent in each. Differences in technique, lack of standardization, and absence of any universally accepted definition of blindness make quantitative comparisons of blindness rates between countries all but impossible. Goldstein points out that in a 1966 review the World Health Organization reported 65 different definitions of blindness in member countries.

In contrast with the section on methodology, the discussion of available data is less uniform. Goldstein presents an exhaustive 60-page description of data for the United States, primarily material published from the Model Reporting Area. The chapter that follows, however, treats data from the rest of the world,

including England and Western Europe, in 19 pages. The appendix contains a detailed description of an Egyptian blindness registration project Goldstein conducted with Professor Said in the mid-1960s.

This book should prove useful to anyone interested in statistics on blindness and their collection and interpretation.

ALFRED SOMMER

Herpetic Infections of Man. By Miro Juretić. Hanover, University Press of New England, 1980. Hardcover, 202 pages, bibliography, subject index, author index, addendum, bibliography to addendum, 15 black and white figures. $13.50

Because of the increasing incidence of herpes simplex viral diseases in the general population, physicians in almost every specialty find themselves faced with diagnosing and managing these infections with greater frequency. Dr. Juretić has put together an excellent clinical review of much of our past and present knowledge of the many diseases caused by this organism.

While his own interest is in primary infections, this book covers in detail both primary and recurrent forms of disease. The first two chapters review the epidemiology and immunology of herpetic disease. The following chapters concern specific clinical entities, including ocular disease, genital, neonatal, and disseminated infections, the immunologically compromised patient, central nervous system herpes, traumatic infections, hepatitis, and the association of this virus with carcinoma, neuropsychiatric disorders, and erythema multiforme. Each chapter discusses mechanisms of disease, clinical findings and course, diagnostic tests, prophylaxis and, if available, treatment.

The first 135 pages, which review reports published before and during the early 1970s, are particularly strong. An extensive bibliography (35 pages) and subject and author indices for this period follow. Next, there is a six-page addendum, in smaller print, that informs the reader of new developments and changes in therapy up to 1979. This includes almost all the new antiviral agents such as acyclovir, adenine arabinoside, trifluorothymidine, and phosphonoacetic acid as well as vaccines, interferon, solvents, and dye-light therapies. This is accompanied by a seven-page bibliography. It is complete, but I found at least one misreference.

The book is well-written and will inform readers about many areas of herpetic disease that may be unfamiliar to them but which are unquestionably important to our understanding and management of ocular infections. I recommend it to medical students, residents, and practicing physicians as an excellent source of information on this important clinical problem.

DEBORAH PAVAN-LANGSTON

Contact Lenses. A Handbook for Patients. By H.-W. Roth and M. Roth-Wittig. Translated by D. L. MacKeen. Hagerstown, Harper and Row, Publishers, 1980. Paperbound, 75 pages, index, 32 black and white figures. $7.95

This short book offers advice to those who wear contact lenses. It is a collection of answers to the countless questions asked by patients. It is in easy-to-read English and is well written. D. L. MacKeen is to be congratulated on his

translation of this book which was originally in German.

The book is divided into nine sections. Among the topics covered are how the eye sees, the different ways of correcting refractive errors, who can wear contact lenses, how contact lenses are fitted, how to take care of contact lenses, and possible complications of contact-lens wear. There is a strong section on special questions and problems. The book also includes a glossary of terms and tables with maintenance products for both soft and hard contact lenses.

This book will be useful in the waiting rooms of all those who fit contact lenses.

MATHEA R. ALLANSMITH

Computer Assisted Eye Examination. Background and Prospects. By Elwin Marg, Arthur G. Bennett, Maxwell M. Lang, and Robert D. Reinecke. San Francisco, San Francisco Press, Inc., 1980. Hardcover, 146 pages, author index, subject index, 2 appendices, 5 tables, 34 black and white figures, 12 black and white plates. $10

This book describes the authors' experience with computers in a clinical setting. The tone of the book is set in the introduction where Marg states, "It is the general vision practitioner rather than a contact-lens specialist or the eye surgeon who can be aided materially by computers at this time."

The first chapter reviews the optometric examination and explains how a computer can be used in some of these steps. Some floor plans for optometric and ophthalmic offices using computers are suggested. The second chapter, entitled "A Historic Review of Optometric Principles and Techniques," discusses geometric optics, the assessment of visual acuity, subjective refraction, astigmatism, and automatic refraction. The third chapter discusses optical systems for the refractive examination of the eye. A variety of trial frames and other measurement devices are illustrated. The discussion even includes astronomical telescopes and how they were modified to serve as optometers. The chapter ends by discussing several of the popular automated refractors.

The fourth chapter considers computer-assisted case histories and gives details about a system involving a Digital Equipment Corporation PDP-8 computer and a yes/no/doubtful decision tree. This system works but may not be cost effective. The fifth chapter deals with modular ocular examinations and history-taking, visual acuity and stereopsis, visual fields, refraction, and intraocular pressure measurements.

The final chapters are concerned with computer-assisted refraction, including the application of computers for subjective refraction and objective refraction with visual-evoked responses.

This book will interest those studying the history and art of refraction and those who are engaged in research involving computers in optometry.

KARL J. FRITZ

Neuro-Ophthalmology. Volume X. In Memory of Dr. Frank B. Walsh. Edited by Joel S. Glaser. St. Louis, C. V. Mosby Co., 1980. Hardcover, 242 pages, memorial to Dr. Frank B. Walsh, index, 70 black and white figures. $45

This volume is in memory of Dr. Frank B. Walsh. It leads off with four charming biographic notes concerning Walsh's interest in neuro-ophthalmology, neurology, neurosurgery, pathology, and base-

ball. There are affectionate momentos of this many-sided man with his gift for friendship, his remarkable clinical ability, and his high intelligence and wisdom that created a new specialty. He stimulated some of the best minds in medicine, and many continue his neuro-ophthalmic traditions.

Additionally, Charles Snyder provides a biographic note on Harvey Cushing and his transfer to Yale after he became emeritus professor at Harvard at 63 years of age. This gives quite a different picture than the one described in Fulton's 1946 biography.

The remainder of the book consists of reviews of various topics. These annual volumes become increasingly valuable. They provide discussions and reviews of material that would often be considered unsuitable for a medical journal inasmuch as many of them are reviews and editorializing. I congratulate Joel Glaser on bringing together such an interesting volume and preparing it so meticulously.

FRANK W. NEWELL

SYMPOSIA

Recent Advances on the Lacrimal System. Edited by Mitsuhiro Yamaguchi. Asahi, Japan, Asahi Evening News, 1980. Hardcover, preface, profiles of contributors, addendum, 146 black and white figures, 62 color figures.

JONES, I. S.: **Surgical considerations in the management of lacrimal gland tumors**

KIKKAWA, T., AND AKAGI, Y.: **Sympathetic innervation and its physiological function in rabbit lacrimal gland**

KORCHMÁROS, I., SZALAY, E., FODOR, M., AND FEDAK, L.: **Probable etiology of the non-opened lacrimal pathway**

KORCHMÁROS, I., SZALAY, E., FODOR, M., AND JABLONSZKY, E.: **Spontaneous opening rate of congenitally blocked nasolacrimal ducts**

RADNOT, M.: **Scanning electron microscopy (SEM) of the lacrimal sac**

WERB, A.: **A clinical approach to lacrimal problems**

DEL CASTILLO, J. M.: **On the gravity as one of the impelling forces of lacrimal flow**

JONES, L. T.: **Conjunctive dacryocystorhinostomy**

WEIL, B.: **The Lester Jones operation. Conjunctive dacryocystorhinostomy with permanent prosthesis**

PICÓ, G.: **Repair of the freshly lacerated canaliculus**

BEDROSSIAN, R. H.: **Silicone intubation of the lacrimal drainage system**

CALLAHAN, M. A.: **Repair of lacerated canaliculi—Quickert's silicone intubation technique with microsurgical anastomosis**

FASANELLA, R. M.: **Common canaliculus or internal common punctum problems with lacrimal sac problems**

PICÓ, G.: **Technique of dacryocystorhinostomy**

WERB, A.: **The standard dacryocystorhinostomy**

WANIA, J. H.: **A new technique for canaliculo dacryocystorhinostomy**

YAMAZAKI, M.: **Dacryocystorhinostomy—Statistical observation of over 500 cases**

ODEH-NASRALA, N.: **Dacryocystoplasty. Another approach to the obstruction of the nasolacrimal duct**

CHANDRA, D. B., AND SWARUP, D.: **Further observations on re-establishment of lacrimal drainage in unusual cases**

MIYAKE, M.: **Reconstruction of the lacrimal passage by the intubation method**

JAIN, S. C., SRIVASTAVA, S. P., AND SAINT, V. K.: **Intubation of the sac and nasolacrimal duct in chronic dacryocystitis by a specially designed polythene tube**

NAGASHIMA, K.: **Retrograde silicone intubation of the nasolacrimal duct in children**

SUTARIA, S. N.: **Silastic "nathni" in recanalisation of the lacrimal passages**

HAMARNE, N.: **Some modern methods in lacrimal system surgery**

VEIRS, E. R.: **Failures following nasolacrimal operations and procedures. Cases and corrections**

REINECKE, R. D.: **Complications of silicone lacrimal tubes for conjunctive dacryocystorhinostomy**

MILDER, B.: **Complications of lacrimal surgery**

YAMAGUCHI, M.: **A stroll in the lacrimal field—Instead of summarization**

Current Topics in Eye Research. Edited by Jose A. Zadunaisky and Hugh Davson. New York, Academic Press, Inc., Publishers, 1980. Hardcover, 336 pages, preface, index, 718 black and white figures. $35

PIATIGORSKY, J.: **Intracellular ions, protein metabolism, and cataract formation**

CHURCH, R. L.: **Cell hybrids in ocular tissues**

VARMA, S. D.: **Aldose reductase and the etiology of diabetic cataracts**

LAING, R. A.: **Specular microscopy of the cornea**

COPELAND, D. E.: **Functional vascularization of the teleost eye**

SIVAK, J. G.: **Accommodation in vertebrates. A contemporary survey**

Advances in Ophthalmology. Volume 41. Edited by E. B. Streiff. Basel, S. Karger AG Publishers, 1980. Hardcover, 216 pages, index, 11 tables, 57 black and white figures. $96

D'EPINAY, S. L.: **Kongenitales Glaukom**

SCHLEUSSNER, E., AND HEMPELMANN, G.: **Anästhesiologische Gesichtspunkte bei intraokularen Eingriffen unter Berücksichtigung der Implantation intraokularer Linsen**

DISMUSKES, K.: **Recommended standard procedures for the clinical measurement and specifications of visual acuity**

ATWELL, C.: **Workshop on the role of psychophysics and physiological optics in ophthalmic diagnosis and patient evaluation**

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Neurologica Scandinavica

BRAIN STEM AUDITORY AND VISUAL EVOKED POTENTIALS IN MULTIPLE SCLEROSIS. Kjaer, M. (Dept. Neurol., Århus Kommunehospital, Århus, Denmark). Acta Neurol. Scand. 62:14, 1980.

The diagnostic value of the checkerboard pattern-reversal visual evoked potential (VEP) and the random, low rate stimulated brain stem auditory evoked potential was compared in 99 patients with established or suspected multiple sclerosis. In normal subjects examined by both techniques no abnormal recordings were found. In 49 patients with definite multiple sclerosis an incidence of abnormality was found in 100% of VEP and in 84% of brain stem auditory evoked potential recordings. In 50 patients with probable or possible multiple sclerosis an abnormal VEP was found in 70% and an abnormal brain stem auditory evoked potential in 50%. When the two examinations were combined, the diagnostic yield increased to 100% and 80%, respectively. Twenty-two patients had only spinal symptoms; in these the VEP gave 73%, the brain stem auditory evoked potential 55% and the combination 82% abnormalities. The combination of the two techniques was found useful for demonstrating demyelinating lesions in the central nervous system, the diagnostic value being greatest when these lesions were clinically silent. (4 tables, 14 references)—Author's abstract

Annals of Neurology

BLINDNESS FOLLOWING TENTORIAL HERNIATION. Keane, J. R. (Dept. Neurol., Univ. Southern California Med. Ctr., Los Angeles, Calif.). Ann. Neurol. 8:186, 1980.

In seven patients, permanent bilateral visual loss was the chief neurological residual following relief of tentorial herniation. The causes of herniation were unilateral subdural hematoma in three patients and bilateral subdural hematomas, bilateral subdural empyemas, traumatic intracerebral hematoma, and postoperative infarction in one patient each. Three patients, including two with occipital infarction documented by computed tomographic scan, developed optic atrophy, indicating that both pregeniculate visual pathway damage and posterior cerebral artery compression occurred. Increasing availability of computed tomographic scanning should help eliminate this rare cause of blindness by facilitating earlier diagnosis of supratentorial masses. (5 figures, 2 tables, 20 references)—Author's abstract

Archives of Internal Medicine

BILATERAL ENDOGENOUS ESCHERICHIA COLI ENDOPHTHALMITIS. Cohen, P., Kirshner, J., and Whiting, G. (Dept. Med., State Univ. New York, Syracuse, N.Y.). Arch. Intern. Med. 140:1088, 1980.

Exogenous endophthalmitis following trauma or surgery is unfortunately not uncommon but endogenous endophthalmitis is rarely reported. The patient described here was a 62-year-old man who had a lower urinary tract infection from which *Escherichia coli* was isolated. He received rather small doses of antibiotics for several days and on the 17th hospital day reported loss of vision in one eye. Two days later he reported reduced vision in the second eye. A clinical diagnosis of endophthalmitis was made and an anterior chamber tap performed which was negative. In spite of intensive antibiotic therapy, given systemically, topically and subconjunctivally, vision was lost in both eyes. The patient had severe pain in one eye because of an increase in intraocular pressure and this eye was enucleated. Vitreous aspirate cultures yielded *E. coli* with the identical sensitivity pattern to the *E. coli* recovered from the urine. The second eye remains totally blind one year after the episode. (9 references)—David Shoch

British Medical Journal

RELEVANCE OF COLOR VISION AND DIABETIC RETINOPATHY TO SELF-MONITORING OF BLOOD GLUCOSE. Graham, K., Kesson, C. M., Kennedy, H. B., and Ireland, J. T. (Depts. Med., and Ophthalmol., Southern General Hosp., Glasgow). Br. Med. J. 281:971, 1980.

A study was performed to determine the effect of color vision defects and diabetic retinopathy on diabetic patients' ability to use a visual method of measuring their own blood glucose concentrations. Forty-eight diabetics whose color vision and retinal status was assessed by an ophthalmologist carried out 311 blood glucose estimations using oxidase-peroxidase test strips which were then compared with laboratory values. There was a trend towards poor performance with advancing age but neither color vision nor diabetic retinopathy had a significant effect on patients' ability to use this visual method of estimating blood glucose concentrations. The vast majority of diabetics who will benefit from being able to monitor their own blood glucose control should have no difficulty in using a visual method of testing, even if they do have defects of color vision. (2 tables, 13 references)—Authors' abstract

Bulletin of the World Health Organization

A THREE-YEAR FOLLOW-UP OF OCULAR ONCHOCERCIASIS IN AN AREA OF VECTOR CONTROL. Thylefors, B., and Tønjum, A. M. (Onchocerciasis Control Program in Volta River Basin Area, Upper Volta). Bull. WHO 58:107, 1980.

A study was done on representative samples of inhabitants of five villages in the Volta River Basin in 1975. A vector controlled program was then put in place and the same villages were re-examined three years later. The total incidence of ocular onchocerciasis was about 8.6% and this showed little change over the three years. However, there was significantly less infection among children aged five to 14 years compared with 1975 and a disappearance of ocular signs of 11.7% of the sample examined. There was a particularly low incidence of sclerosing keratitis as compared with areas of uncontrolled transmission and this may be associated with the finding of a significantly decreased microfilarial load in the cornea even though the number of living microfilariae in the anterior chamber of the eye was apparently unchanged. In the test areas the incidence of blindness due to onchocerciasis was low and confined to individuals who already presented severe eye manifestations of the disease before the beginning of the vector control campaign. (7 tables, 11 references)—David Shoch

Canadian Medical Association Journal

EYE INJURIES IN RACQUET SPORTS: A CONTINUING PROBLEM. Easterbrook, M. (1849 Yonge St., Toronto, Ont.). Can. Med. Assoc. J. 123:268, 1980.

Over an 18-month period 67 eye injuries were reported in Canada as a result of playing squash and 18 from racquetball. This type of injury does not occur only in new players since the average time that these people had engaged in racquet sports was five years. Of importance is the fact that there were eight injuries in squash and ten in racquetball in players wearing an open-type of eye guard. In two of these cases the cause of the injury was the racquet but in 16 of them it was the ball; the ball went between the upper and lower rims of the eye guard striking the eye directly. Therefore, the author recommends that a better eye guard would be a thick plastic lens with the appropriate prescription for those who need one. (4 figures, 1 reference)—David Shoch

Circulation

OCULAR PNEUMOPLETHYSMOGRAPHY IN THE EVALUATION OF CAROTID STENOSIS. Baker, J. D., Barker, W. F., and Machleder, H. I. (Dept. Surg., UCLA Medical School, Los Angeles, Calif.). Circulation 62:1, 1980.

Ocular pneumoplethysmography is a noninvasive technique for detecting carotid ste-

nosis by measurement of ophthalmic artery pressure. The results of the test were studied in 227 patients who underwent biplane angiography. The overall accuracy was 93% in the correct identification of hemodynamically significant stenosis (defined as a 60% or greater decrease in luminal diameter). The good result of this single test makes it the authors choice of noninvasive methods for both screening and follow-up study of carotid occlusive disease. (1 figure, 1 table, 5 references)—Authors' abstract

Diabetes

A DETAILED STUDY OF RISK FACTORS FOR RETINOPATHY AND NEPHROPATHY IN DIABETES. West, K. M., Erdreich, L. J., and Stober, J. A. (Dept. Biostatistics and Epidemiol., Univ. Oklahoma Health Sciences Ctr., Oklahoma City, Okla.). Diabetes 29:501, 1980.

A large population of Indians in 27 contiguous Oklahoma counties was examined and an attempt made to identify and recruit Indians with clinically diagnosed diabetes who were between 35 and 54 years of age. In total there were 973 subjects and more than 100 variables were measured. The most powerful risk factor for microangiopathy was duration of diabetes, but frequency of both retinopathy and nephropathy was impressively related to the level of plasma glucose at the time of examination. This latter relationship was independent of the effects of other variables. Risk of retinopathy was also related to factors that were probably associated with higher previous plasma glucose levels—need for insulin therapy, history of ketonuria, present level of plasma triglycerides, leanness, and younger age of onset of diabetes. In subjects with a systolic blood pressure of less than 170 mm Hg who did not have proteinuria, no significant relationship was found between blood pressure and frequency of retinopathy, but when the systolic pressure was greater than 170 mm Hg, rates of retinopathy were excessive, even in those subjects without proteinuria. Several other factors had no significant relationship to retinopathy or nephropathy: these included age, level of education, smoking, and plasma cholesterol. Rates of retinopathy and of mild proteinuria were similar in men and women. After adjustment for duration of diabetes, the risk of retinopathy was no higher in the 102 subjects with two diabetic parents than in the 254 who had no family history of diabetes. (8 tables, 14 references)—David Shoch

Investigative Ophthalmology and Visual Science

RETINAL LIGHT EXPOSURE FROM OPHTHALMOSCOPES, SLIT LAMPS, AND OVERHEAD SURGICAL LAMPS. Calkins, J. L. and Hochheimer, B. F. (Wilmer Ophthalmological Inst., Baltimore, Md.). Invest. Ophthalmol. Visual Sci. 19:1009, 1980.

The projected beam radiance of several common ophthalmologic instruments was measured, and potential hazard to the patient from light exposure was analyzed with reference to safety standards for coherent light. The indirect ophthalmoscopes tested appear to be "safe" under moderate voltage settings, provided exposure is reasonably brief. Slit-lamp biomicroscopy of the fundus, however, merits caution. It produces a three-times-higher retinal irradiance than the indirect ophthalmoscope. Overhead surgical lamps produce a retinal irradiance about one-third that of the indirect ophthalmoscope (for clear media and dilated pupil). This could be dangerous, since an operation may take long enough to exceed the maximal permissible exposure by several orders of magnitude. Major design changes are indicated for surgical illuminators to extend the "safe time" to the 40 to 60 min. range. (4 figures, 3 tables, 18 references)—Authors' abstract

Journal of Cardiovascular Pharmacology

[^{3}H] DIGOXIN IN THE OPTIC TRACT IN DIGOXIN INTOXICATION. Binnion, P. F., and Frazer, G. (Cardiol. Res. Lab., Pennsylvania Hosp., Dept. Biomed. Sci., Drexel Univ., Philadelphia, Pa.). J. Cardiovasc. Pharmacol. 2:699, 1980.

Normal and chronically hypokalemic dogs were infused with [^{3}H] digoxin until ventricular tachycardia occurred, at which point the concentration of digoxin was measured in all tissues involved in vision. The highest concentration was found in the choroid-retina of the eye, and this was considered the most likely site for the various visual changes seen in digitalis intoxication in man. Chronic hypokalemia did not influence the concentration or distribution of digoxin in the optic tract. It is speculated that the increased digoxin level in the extracranial part of the optic nerve is due to a weakness in the blood—retina barrier where the optic nerve fibers pass through the retina. One eye was left *in situ* for three days after death to study post-mortem changes in digoxin distribution. Vitreous humor analysis is being used to study the cause of death in man, but the authors found an increase in the vitreous humor digoxin level after death due to loss from its primary binding site in the choroid-retina. A similar effect would be expected with any drug bound to the retina and would have to be taken into account when considering the cause of death in forensic pathology. (3 tables, 35 references)—Authors' abstract

Journal of the National Medical Association

SNAKEBITE AND SNAKE VENOM OPHTHALMIA. Ajao, O. G., and Hawtin, J. G. (Dept. Surg., Univ. College Hospital, Ibadan, Nigeria). J. Natl. Med. Assoc. 72:961, 1980.

Poisonous snakes are classified into three types: the elapids (neurotoxic), sea snakes (myotoxic), and the vipers (vasculotoxic). Two members of the elapid group are found in West Africa and these are known as the spitting cobras. The spitting cobra can eject venom for up to five meters toward the eye of its foe. The immediate effect is severe pain in the eyes and tearing. Blindness can occur if the eyes are neglected. Treatment of snake venom ophthalmia includes immediate irrigation of the eyes followed by topical application of atropine and antibiotics. The efficacy of local antivenom administration has not been proved. (3 figures, 18 references)—David Shoch

Journal of Neurology, Neurosurgery and Psychiatry

A COMPARISON OF FLIGHT OF COLOURS WITH VISUALLY EVOKED RESPONSES IN PATIENTS WITH MULTIPLE SCLEROSIS. Swart, S., and Millac, P. (Dept. Neurol., Leicester Royal Infirmary, Leicester, England). J. Neurol., Neurosurg., Psychiatry 43:550, 1980.

The flight of colors is a series of after images which results when a bright light is directed at the retina and then withdrawn. These take the form of circles and haloes of light which fade, change and reappear in sequence over a period of a few minutes. Normally these will persist for about two and a half minutes but it has been reported that they do not last this long in patients with optic neuritis or multiple sclerosis. The authors studied 36 eyes and did both a flight of colors test and a visual evoked response. They found a good correlation between the increased latency and a diminution in the time of persistence of the flight of colors. The two tests appear to be comparable in their ability to detect disturbance in the visual system. In doing the test it is necessary to dark adapt the patients for about five minutes and then shine a bright light at the eye from about an inch away for ten seconds. (1 figure, 5 references)—David Shoch

Journal of the Royal Society of Medicine

VISUAL ACUITY, ITS DEVELOPMENT AND AMBLYOPIA. Ikeda, H. (Vision Res. Unit, Rayne Inst., St. Thomas's Hosp., London). J. Royal Soc. Med. 73:546, 1980.

Foveal visual acuity is apparently not only quantitatively different, that is, due to the packing of more cones in the area, but also qualitatively different. There are special retinal ganglion cells which may be divided into two major physiologic classes: one of these, the X cell is suitable for fine spatial discrimination (visual acuity) and is also called a sustained or tonic cell. The second is the Y cell which is also called a transient cell or

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phasic cell which are suitable for the detection of rapid and coarse movement and the initiation of the fixation reflex. These cells can be studied by the use of slow-moving gratings. The cells are challenged with progressively finer gratings until the cell no longer gives a rhythmic response to individual lines of the grating. Thus, the visual acuity of the cell can be defined as the finest grating which the cell responds by firing to individual lines. As a result of these studies the author concludes that high visual acuities is provided by sustained-X cells in the area centralis of the retina. This high foveal visual acuity developes postnatally and amblyopia is an arrest of development of the sustained-X cell function. Amblyopia therefore, is caused by a habitually blurred image at the visual axis during the critical period of development. The author further suggests that within the first two years of life occlusion therapy should be used with caution to prevent amblyopia developing in the good eye. (12 figures, 24 references)—David Shoch

Laboratory Investigation

MEASLES RETINOPATHY: A HAMSTER MODEL OF ACUTE AND CHRONIC LESIONS. Parhad, I. M., Johnson, K. P., Wolinsky, J. S., and Swoveland, P. (Dept. Neurol., Univ. California, San Francisco, Calif.). Lab. Invest. 43:52, 1980.

Measles and subacute sclerosing panencephalitis strains of virus inoculated intracerebrally in suckling and weanling hamsters spread immediately to the eye via the blood. Virus replicates in the brain and eye in parallel, and in higher titers than it does in other organs. Acute retinal lesions consist of focal syncytia, inclusion-bearing cells, and necrosis. Immunofluorescence studies show measles-specific antigen in the affected cells. Ultrastructurally, measles nucleocapsids and virions are seen in the retinal cells. The necrosis is followed by a differential repair of the retinal layers that results in retinal dysplasia. Chronically, the retinal dysplasia progresses to atrophy and gliosis of the retina. The chronic lesions are histologically similar to those seen in human subacute sclerosing panencephalitis and measles retinopathies. (18 figures, 1 table, 39 references)—Authors' abstract

Lancet

BLOOD ALCOHOL AND EYE MOVEMENTS. Bittencourt, P., Wade, P., Richens, A., Smith, T., Lloyd, D., and Toseland, P. (Inst. Neurol., National Hospital, London). Lancet 2:981, 1980.

Alcohol has been reported to affect eye movements but the degree of the effect in relation to blood concentration of alcohol has not been examined. The authors allowed 12 healthy volunteers (six male and six female) to drink alcohol as whiskey or gin until they considered themselves unfit to drive. At this point blood levels of alcohol were measured and a standardized electro-oculographic procedure used to record smooth pursuit eye movements on magnetic tape. There was a direct linear relationship between the decrease in smooth pursuit velocity and the alcohol concentration. This provides reliable evidence that at a blood alcohol level of 80 mg/100 ml (the legal limit in the United Kingdom for driving purposes) smooth pursuit eye movement, that is the capacity of the eyes to track objects moving slowly across the visual field, is impaired by about 25%. (1 figure, 7 references)—David Shoch

Mayo Clinic Proceedings

PSEUDOTUMOR CEREBRI. Rush, J. A. (Dept. Ophthalmol., Univ. South Florida, Tampa, Fl.). Mayo Clin. Proc. 55:541, 1980.

The visual status of 63 patients with pseudotumor cerebri, seen between 1961 and 1978, was retrospectively analyzed. The median interval between first examination and response to a questionnaire was six and one-fourth years. Seven patients (11%) had 6/9 (20/30) or worse visual acuity at last ocular examination. Visual loss was not statistically related to (1) the presence of visual obscurations, (2) the duration of symptoms, (3) the degree of papilledema, or (4) the number of recurrences. Surgical intervention may have

prevented further deterioration of vision in several patients, but therapeutic efficacy could not be determined in this study. No patient with visual loss had substantial improvement, regardless of type of treatment. (6 tables, 13 references)—Author's abstract

HEALTH CARE EXPENDITURES: THE APPROACHING CRISIS. Herrell, J. H. (Sec. Financial Analysis and Planning, Mayo Clinic, Rochester, Minn.). Mayo Clin. Proc. 55:705, 1980.

Over the past 30 years the total annual expenditures in the United States for medical care and other forms of health-related activities increased 1,500% and has grown from 4½% of the gross national product to 9%. The major expansion has been in hospital care which has gone from 33% of the health care dollar to 40%. What is surprising is that the percentage of the health care dollar for physician services has gone down 5% from 23% to 18% and the outlay for drugs, eyeglasses and appliances has decreased from 18% to 10%. It appears obvious that if there is going to be some attempt to contain health costs this must be in the area of reducing hospital expenditures and this should give impetus to the development of more and more outpatient procedures. (5 figures, 4 tables, 6 references)—David Shoch

Ophthalmology

THE HISTOLOGY OF HUMAN GLAUCOMA CUPPING AND OPTIC NERVE DAMAGE: CLINICOPATHOLOGIC CORRELATION IN 21 EYES. Quigley, H. A., and Green, W. R. (Glaucoma Ser. and Eye Pathol., Wilmer Institute, Johns Hopkins Univ., Baltimore, Md.). Ophthalmol. 86:1803, 1979.

The optic nerve head and optic nerves of 21 eyes obtained from glaucoma patients were examined by light and electron microscopy. In all of these cases clinical information was available and in several cases it was possible to correlate the degree and distribution of optic nerve damage with the clinical appearance of the optic disc and visual field studies. There was no selective loss of astrocytes of the optic nerve head in early glaucoma cupping. Acquired increases in optic disc cup size prior to detectable visual field loss probably represents loss of ganglion cell axonal fibers which is not yet significant enough to produce field defects. At the level of the retrobulbar optic nerve, the ganglion cell axonal fibers of the superior and inferior quadrants seem to be lost earlier than the fibers of the nasal and temporal nerve periphery. Since the superior and inferior poles of the optic nerve may contain the fibers of arcuate area ganglion cells, these data confirm the presumption from visual field testing that arcuate area ganglion cell fibers are selectively more susceptible to damage in chronic glaucoma. (22 figures, 1 table, 26 references)—David Shoch

PERIPHERAL NASAL FIELD DEFECTS IN GLAUCOMA. Werner, E. B., and Beraskow, J. (Dept. Ophthalmol., McGill Univ., Montreal, Canada). Ophthalmol. 86:1875, 1979.

One hundred fifty-one eyes of 101 consecutive patients with chronic open-angle and low tension glaucoma showed typical visual field changes. Sixty of the eyes had a nasal step, either alone or in combination with other defects. In 17 of these eyes, it was possible to demonstrate an isolated scotoma in the nasal periphery. It was concluded that the peripheral nasal step is a nerve fiber bundle defect that in its earliest phase produces a scotoma. In this sense, it behaves similar to the more central defects, rather than simply as a depression of isopters nasally. (5 figures, 1 table, 14 references)—Authors' abstract

THE NATURAL COURSE OF CENTRAL RETINAL VEIN OCCLUSION. Zegarra, H., Gutman, F. A., and Conforto, J. (Cleveland Clin. Foundation, Cleveland, Ohio). Ophthalmol. 86:1931, 1979.

Complete central retinal vein occlusion usually results in a hemorrhagic retinopathy which is severely progressive and results in a permanent decrease in vision. Venous stasis retinopathy which is probably due to a partial retinal vein occlusion is a less severe form of the disease, is benign and usually self-limiting with minimal visual impairment. The occlu-

sion here occurs posteriorly in the optic nerve, allowing collaterals to develop, while in hemorrhagic retinopathy the occlusion is probably at the level of the lamina cribrosa or anterior to this where there are few collaterals and therefore a more florid hemorrhagic retinopathy develops. The authors studied 25 patients with these two entities for a period of one to eight years. A systemic vascular disease was commonly associated with retinal venous occlusive disease. Although generally the progress in venous stasis retinopathy is good, two of ten patients with venous stasis retinopathy developed hemorrhagic retinopathy with a poor visual outcome. (2 figures, 5 tables, 17 references)—David Shoch

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FRANK W. NEWELL, M.D.
Editor-in-Chief
AMERICAN JOURNAL OF OPHTHALMOLOGY

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

INTERNATIONAL SYMPOSIUM: HERPESVIRUS: CLINICAL, PHARMACOLOGICAL, AND BASIC ASPECTS

An international symposium, "Herpesvirus: Clinical, Pharmacological, and Basic Aspects," will be held in Tokushima City, Japan, July 27–30, 1981. For further information, write Dr. H. Shiota, Dept. of Ophthalmology, Tokushima University, Kuramoto-cho, Tokushima City 770, Japan.

FIGHT FOR SIGHT, INC.: CLOSING DATE FOR 1981–1982 AWARD PERIOD

Fight for Sight, Inc., announced March 1, 1981, as the closing date for applications to be reviewed for its 1981–1982 award period. For further information, write Secretary, Fight for Sight, Inc., 139 East 57th St., New York, NY 10022.

SOCIETY OF AIR FORCE CLINICAL SURGEONS: POSTGRADUATE SEMINAR

The Society of Air Force Clinical Surgeons is sponsoring a surgical postgraduate seminar April 12–15, 1981, in San Antonio, Texas. For further information, write Lt. Colonel Donald E. Novicki, Chairman, Dept. of Urology, Wilford Hall, USAF Medical Center/SGHSU, Lackland AFB, TX 78236.

LENOX HILL HOSPITAL: COURSE ON EXTERNAL DISEASES OF THE EYE

Lenox Hill Hospital will hold a course on external diseases of the eye April 9 and 10, 1981, in New York City. The fee is $200 for physicians and $75 for residents. For further information, write Ms. Betty C. Agree, Assistant Director, Dept. of Public Relations, Lenox Hill Hospital, 100 E. 77th St., New York, NY 10021; telephone (212) 794-4500.

NEW YORK MEDICAL COLLEGE: SEMI-ANNUAL OCULOPLASTIC SURGICAL DISSECTION COURSE

The Semi-Annual Oculoplastic Surgical Dissection Course, sponsored by the New York Medical College, will be held in Tarrytown, New York, May 13–16, 1981. The fee is $650. For further information, write Ms. P. Tamkin, c/o Dr. P. Guibor, 630 Park Ave., New York, NY 10021.

OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA CONGRESS

A meeting of the Ophthalmological Society of South Africa will be held June 2–5, 1981, in Johannesburg, South Africa. The registration fee is $175. For further information, write Secretary, 7th Floor, Tower Hill, Kotze St., Johannesburg, South Africa.

UNIVERSITY OF CHICAGO: ANNUAL ALUMNI DAY

The Department of Ophthalmology of the University of Chicago will hold its Annual Alumni Day on March 4, 1981. The guest speaker is Irving H. Leopold. The staff and residents of Chicago area departments of ophthalmology, University of Chicago alumni, and other physicians are invited to attend. There will be a full-day scientific program, buffet luncheon, and reception following the afternoon session. Registration will be at 9 A.M. with the scientific program scheduled to begin at 9:30 in Room P-117, Albert Merritt Billings Hospital, 950 E. 59th St., Chicago. For further information, write Mrs. Karin Cassel, Eye Research Laboratories, University of Chicago, 939 E. 57th St., Chicago, IL 60637; telephone (312) 947-6064.

MERCY-BAPTIST EYE FOUNDATION: TWELFTH EYE SEMINAR

Mercy-Baptist Eye Foundation will sponsor the Twelfth Eye Seminar on refractive surgery, May 17, 1981, in St. Louis, Missouri. Registration fee is $100. For further information, write Eye Seminars, 621 S. New Ballas Rd., Suite 343, St. Louis, MO 63141.

AMERICAN SOCIETY OF OPHTHALMIC PLASTIC AND RECONSTRUCTIVE SURGERY: NEW OFFICERS

The 11th Annual Scientific Symposium of the American Society of Ophthalmic Plastic and Reconstructive Surgery was held in Chicago Nov. 7 and 8, 1980. The new officers are: President John L. Wobig became the chairman of the advisory board. Allen M. Putterman is president for 1981. Other officers elected included: Jan Berlin, president-elect; Fay E. Millett, vice president; Richard P. Carroll, executive secretary; Joseph Flanagan, secretary; Eugene O. Wiggs, treasurer; and Clinton D. McCord, program chairman.

FIGHT FOR SIGHT, INC.; POSTDOCTORAL FELLOWSHIPS AND DEPARTMENTAL AWARDS

Fight for Sight, Inc., postdoctoral fellowships and departmental awards approved for funding and currently in progress total 22 awards, in the amount of $167,929. A listing of postdoctoral fellowship and departmental awards follows:

FIGHT FOR SIGHT POSTDOCTORAL RESEARCH FELLOWSHIPS 1980–1981

INVESTIGATOR AND INSTITUTION	DIRECT SUPERVISOR	PROJECT TITLE	AMOUNT AWARDED
Carlos G. Arce, M.D. University of Florida Gainesville, Florida	Frank M. Polack, M.D.	Epithelium-stromal interaction during corneal wound healing	$ 8,000
Richard Berkowitz, M.D. Louisiana State University New Orleans, Louisiana	Stephen D. Klyce, Ph.D.	Corneal endothelial structure and function	5,000
Eileen Birch, Ph.D. Massachusetts Institute of Technology Cambridge, Massachusetts	Richard Held, Ph.D.	Development and maintenance of normal binocular vision	10,000
Douglas B. Bowling, Ph.D. Yale University New Haven, Connecticut	Charles Michael, Ph.D.	Central projections of single, physiologically identified ganglion cells from the retinas of cats and monkeys	10,000
Terry A. Cox, M.D. University of Iowa Iowa City, Iowa	H. Stanley Thompson, M.D.	Pupillary and visual evoked response indicators of optic nerve disease	8,500
John J. Donnelly, Ph.D.* Scheie Eye Institute University of Pennsylvania Philadelphia, Pennsylvania	John H. Rockey, M.D.	Immunopathology of helminthic infections of the eye: *Onchocerca cervicalis* infection of the eye as a model for human ocular onchocerciasis	7,500
Geoffrey Gold, Ph.D.* University of California San Francisco, California	Juan Korenbrot, Ph.D.	The detection of light evoked calcium fluxes from intact rods	2,475 (supplemental support)
Barbara M. Goldman, Ph.D. The Rockefeller University New York, New York	Gunter Blobel, M.D.	Molecular analysis of opsin biosynthesis and integration into membranes	7,500
Robert B. Guss, M.D. Stanford University Stanford, California	Peter R. Egbert, M.D.	Endotoxin induced uveitis and its relationship to human diseases	8,334
Anthony S. Hajek, Ph.D.* University of Miami Miami, Florida	Paul Palmberg, M.D.	Human trabecular endothelial cell culture	8,500

*Continuation Award

Investigator and Institution	Direct Supervisor	Project Title	Amount Awarded
Michael E. Hettinger, M.D. Eye Research Institute of Retina Foundation Boston, Massachusetts	Deborah Pavan-Langston, M.D.	The study of new antiviral drugs in ocular and latent herpetic infections	$ 7,500
Ingrid Holmgren, M.D. Harvard University Cambridge, Massachusetts	John E. Dowling, Ph. D.	Investigations of ultrastructure and pharmacological aspects of retinal neurons	10,000
Marguerite B. McDonald, M.D. Louisiana State University New Orleans, Louisiana	Bryan M. Gebhardt, Ph.D.	Cornea cell killing and protection by antibody	10,000
Grant D. Nicol, Ph.D. University of California Berkeley, California	Frank S. Werblin, Ph.D.	Role of cyclic nucleotides in photoreceptor function	10,000
Thomas C. Prager, Ph.D. University of Texas Medical School Houston, Texas	Charles A. Garcia, M.D.	The effect of strict diabetic control by continuous subcutaneous insulin infusion pump. Evaluation by vitreous fluorophotometry	6,000 (supplemental support)
Marjorie D. Shaw, Ph.D. New York University Medical Center New York, New York	Robert Baker, Ph.D.	Superior colliculus connections to the oculomotor nucleus	1,620 (supplemental support)
Ryutaro Tamaki, M.D. University of Michigan Ann Arbor, Michigan	Mathew Alpern, M.D.	Individual differences in the color vision mechanisms of the human fovea in normal and abnormal retinas	9,500
George C. Thorne, M.D. University of California San Francisco, California	G. Richard O'Connor, M.D.	Reactivity of patients' lymphocytes following the implantation of a corneal allograft	10,000
R. David Tomlinson, Ph.D. The Johns Hopkins University Baltimore, Maryland	David A. Robinson, Ph.D.	Site and function of the TVP cell in oculomotor control	10,000

Fight For Sight Departmental Awards 1980–1981

Institution	Chief of Department	Amount Awarded
Ochsner Clinic New Orleans, Louisiana	Thom Zimmerman, M.D., Ph.D.	$5,000
Tulane University New Orleans, Louisiana	Delmar R. Caldwell, M.D.	5,000
University of North Carolina* Chapel Hill, North Carolina	David Eifrig, M.D.	7,500

*Continuation Award

INSTRUCTIONS TO AUTHORS

For the preparation of manuscripts for THE AMERICAN JOURNAL OF OPHTHALMOLOGY

THE AMERICAN JOURNAL OF OPHTHALMOLOGY publishes original and timely articles dealing with clinical and basic ophthalmology. Every typescript submitted is evaluated by two or more scientific referees who recommend that the paper be (1) accepted as submitted, (2) returned for revision, or (3) rejected. Acceptance is determined by such factors as the originality, significance, and validity of the contribution; the suitability of the subject matter for subscribers of THE JOURNAL; and the editorial care with which the manuscript has been prepared. Manuscripts that require extensive editorial correction or mechanical preparation will be returned for that purpose.

All papers are accepted subject to editorial corrections that conform to JOURNAL requirements.

Authors will be advised promptly of receipt of their papers. Thereafter they will be advised within 30 days of acceptance, rejection, or need for revision.

MECHANICAL PREPARATION OF MANUSCRIPT The manuscript should be prepared in the style used by THE JOURNAL. Use black ribbon, use block not cursive type. Provide a 1 1/2-inch margin on all four sides of each typescript page; use 8 1/2 × 11-inch heavy white bond paper. Paragraphs should be indented at least one-half inch. Do not type anything in all capitals; do not underline. Except for standard measures such as mm, Hg, cm, and ml used with numeric quantities, do not use abbreviations anywhere in the text. In the upper right-hand corner identify each page with a number, the first author's name, and an abbreviated title (maximum 40 characters and spaces.)

Single spacing should not be used anywhere in the manuscript. The entire manuscript—including title page and footnotes, text, acknowledgments, references, tables, and legends—should be double spaced. Vertical lines and underlining should not be used anywhere in the text, references, tables, or legends. See the section on tables for the use of horizontal lines.

Submit an original and at least one duplicate copy of both the typescript and the figures. Xerographic duplicates of the typescript are preferred to carbon copies.

The manuscript should be identified and arranged in the following order:

1. Title page
2. Text and summary
3. Acknowledgments
4. References
5. Tables
6. Legends for figures

Each major section should begin on a separate sheet.

TITLE PAGE: The title page should be numbered page 1. It should contain the title, abbreviated heading (no more than 40 characters and spaces), each author's name with a single academic degree, and the city and state where the work was carried out. The departmental affiliation of each author and the institution where the study was performed should be credited in a footnote. Organizational grants and funds should include the names of the authors who are the principal investigators. The name and mailing address of the author to whom correspondence and requests for reprints should be directed must be provided. Each page after the title page must be numbered sequentially, and must include the senior author's name and the abbreviated title in the upper right-hand corner.

ORGANIZATION OF CONTENT: Manuscripts should be organized and prepared in the style used by THE JOURNAL. A brief introductory statement of the problem should be presented. The material and methods should then be precisely and clearly described so that the reader may replicate the study. Explanation of the results of the study should be given, followed by a discussion. The discussion is an elucidation of the results and thus it must relate directly to the topic of the paper.

SUMMARY: Each paper must have a summary that specifically synthesizes the content of the paper in no more than 150 words. The summary must be written so that it can be read independently of the text.

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Published acknowledgment requires an exceptional and conspicuous contribution different from that which occurs with the preparation of every paper printed in THE JOURNAL. Thus, acknowledgments will be published rarely.

REFERENCES: The author is responsible for complete and accurate references, including the proper capitalization and accent marks used in foreign-language publications. References must be numbered consecutively, according to their appearance in the text. Extensive bibliographic reviews are not acceptable. Personal communications should be kept to a minimum and they must be incorporated into the text without a reference number. Reference to studies that have been accepted but have not yet been published should indicate where they will be published. Reference to studies still in progress should be described as such in the text without a reference number. Primary, not secondary, sources must be cited; references derived from encyclopedic reviews are seldom acceptable. References to textbooks must contain inclusive pages. References should be cited in the text as follows: Allen and Smith[1] and Jones[2] described. . .

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1. Fuxe, K., and Jonsson, G.: The histochemical fluorescence method for the demonstration of catecholamines. Theory, practice and application. J. Histochem. Cytochem. 21:293, 1973.

2. Bjorklund, A., Flack, B., and Owman, C.: Fluorescence microscopic and microspectrofluorometric techniques for the cellular localization and characterization of biogenic amines. In Berson, S. A. (ed.): Methods in Investigative and Diagnostic Encocrinology, vol. 1. London, North-Holland Publishing Co., 1972, p. 318.

3. Duke-Elder, S., and Leigh, A. G.: Diseases of the Outer Eye. Cornea and Sclera. In Duke-Elder, S. (ed.): System of Ophthalmology, vol. 8, pt. 2. St. Louis, C. V. Mosby, 1965, pp. 811-856.

Abbreviations of periodicals are listed in Index Medicus: Am. J. Ophthalmol.; Surv. Ophthalmol.; Br. J. Ophthalmol; and the like. If there is any doubt about an abbreviation, the name of the publication should be spelled out completely.

TABLES: Each table must be titled, numbered with an arabic numeral, and cited sequentially in the text. The title should appear directly below the designation, Table 1. Below the title, a double rule (two parallel horizontal lines) should span the width of the table. Between this double rule and a similar, full-width single rule, a heading must be given for each column. Partial rules spanning several column heads may be used to join main headings with applicable subheadings, if necessary. Vertical lines should not be used anywhere in the table. Abbreviations used in the table must be explained in a footnote. The following symbols are used for footnotes in the order indicated: *(asterisk), †(dagger), ‡(double dagger), § (section mark), ‖(parallels), ¶(paragraph mark), and #(number sign). Each table must be double-spaced and nothing in the table should be underlined. Photographically reproduced or mechanically reduced tables are not acceptable.

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CBE Style Manual Committee: Council of Biology Editors Style Manual: A Guide for Authors, Editors and Publishers in the Biological Sciences, 4th ed. Arlington, Virginia, Council of Biology Editors, 1978.

A Manual of Style, 12th ed. Chicago, University of Chicago Press, 1969.

Strunk, W., Jr., and White, E. B.: The Elements of Style, 3rd ed. New York, Macmillan Co., 1979.

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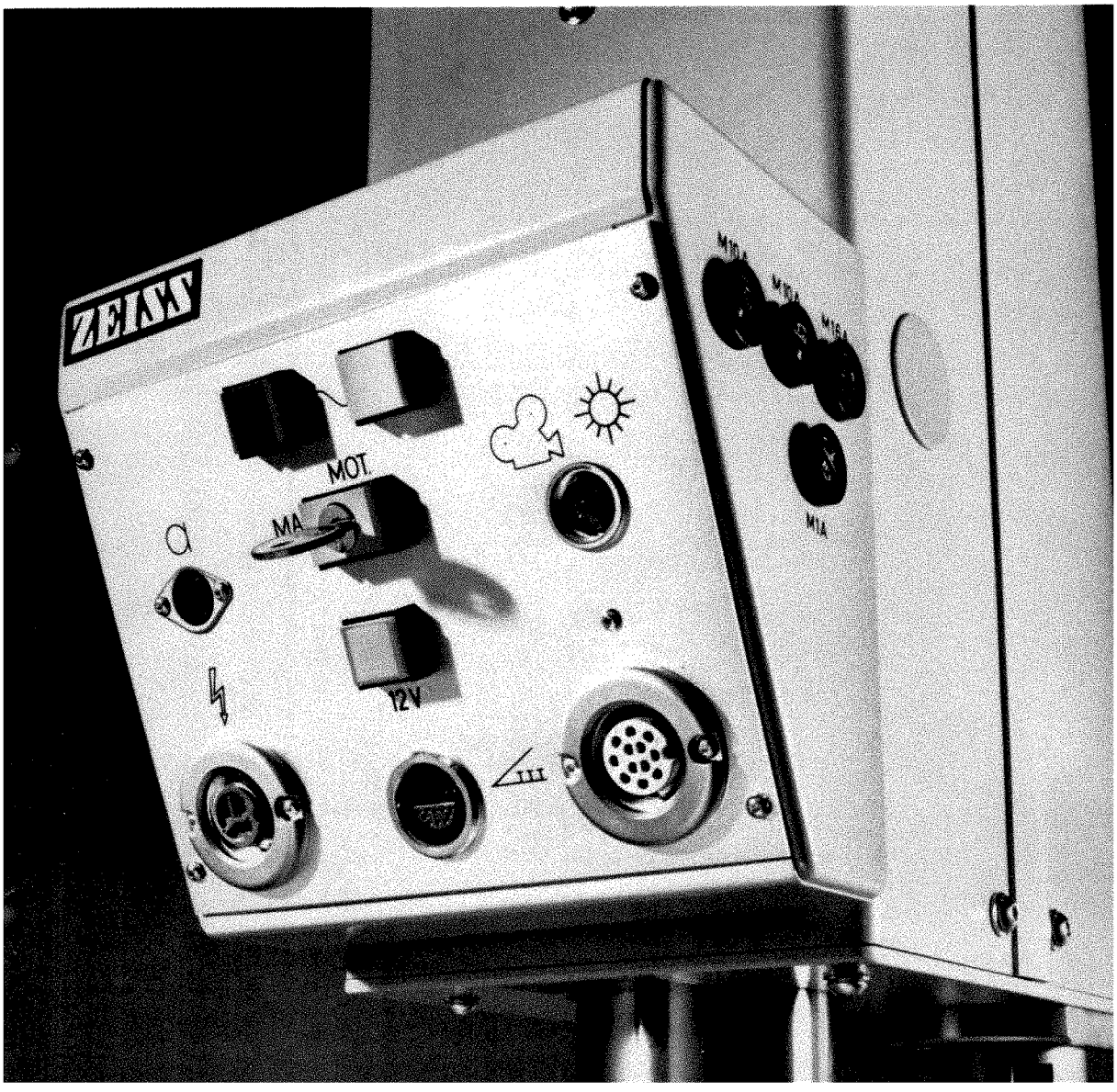
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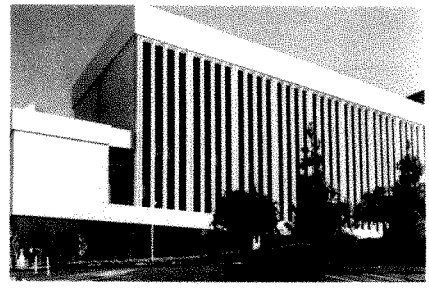
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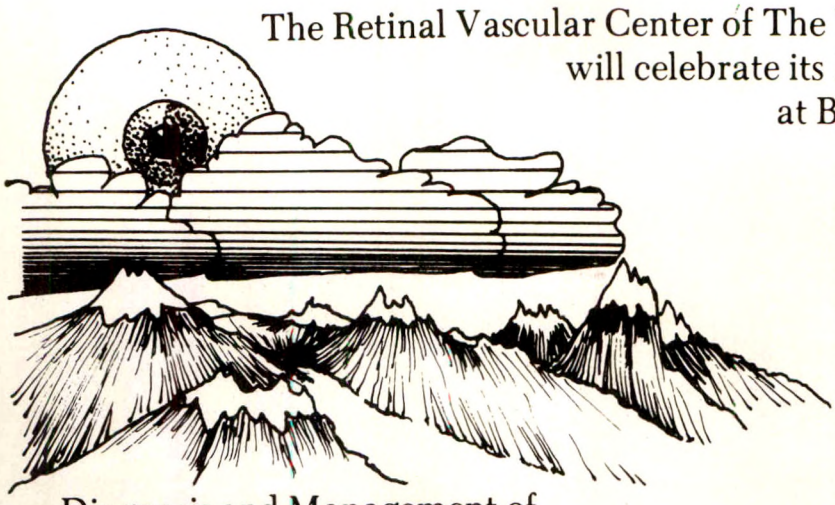
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The Retinal Vascular Center of The Wilmer Institute will celebrate its 11th anniversary at Big Sky, Montana July 12-17, 1981

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Radial Keratotomy
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Guest speaker:
Professor Svatoslav N. Fyodorov

Speakers:

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D. Boyaner, M.D.	Leeds Katzin, M.D.
R. D. Binkhorst, M.D.	Steven Ostbaum, M.D.
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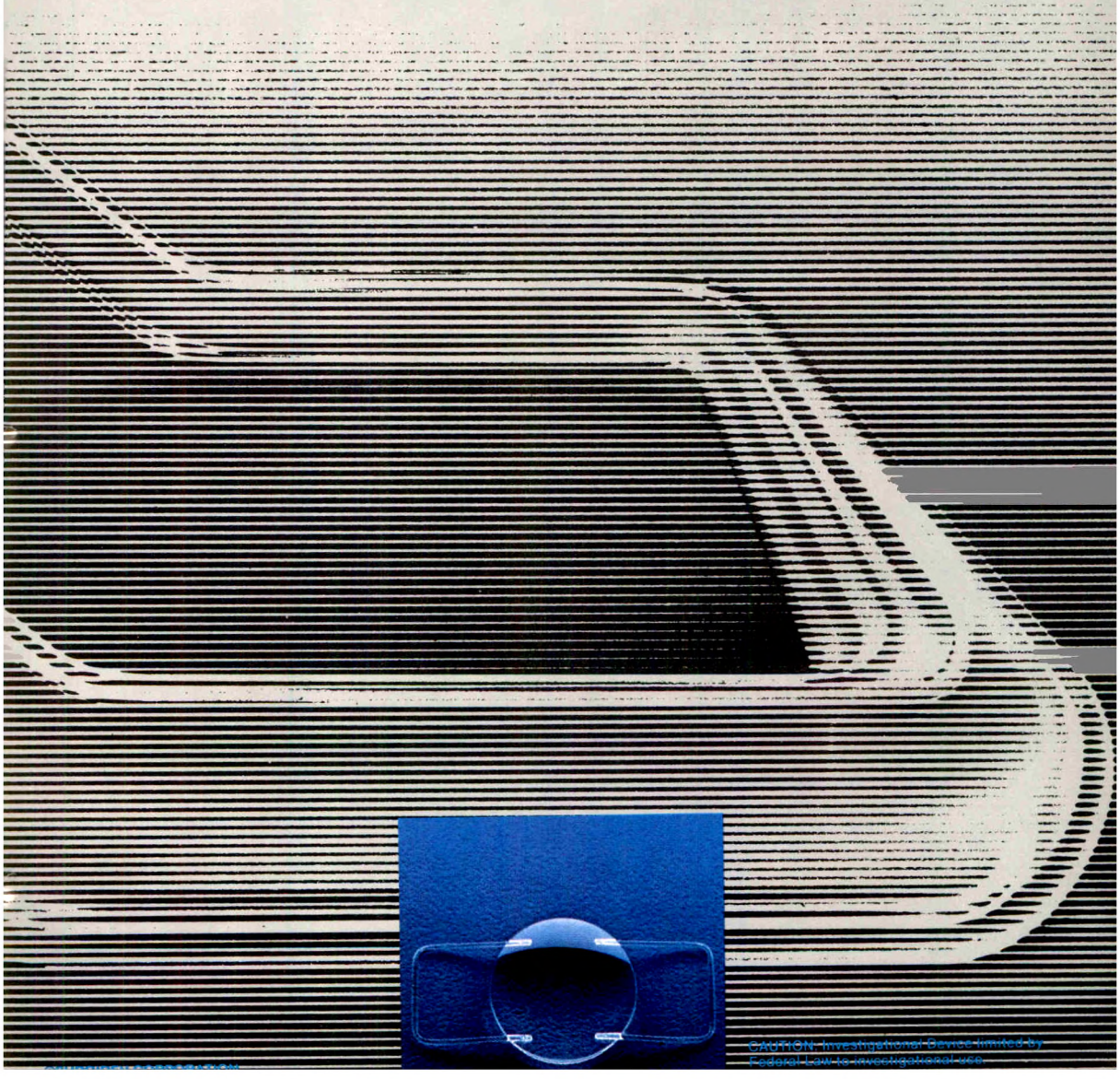
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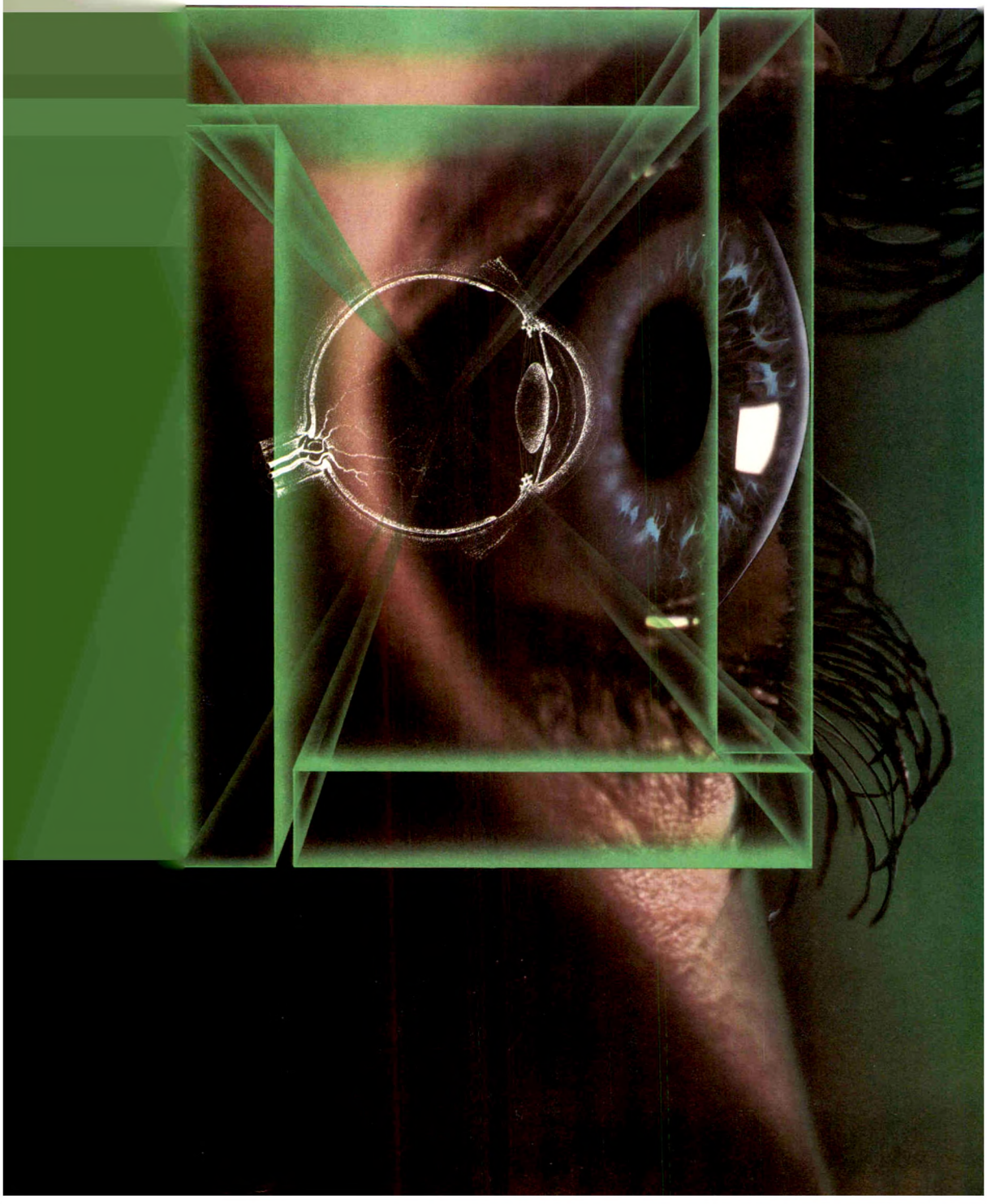
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1. Data on file at Alcon Laboratories.
2. Zimmerman, T. J., Ophthalmology Audio Digest, Vol. 17, No. 1

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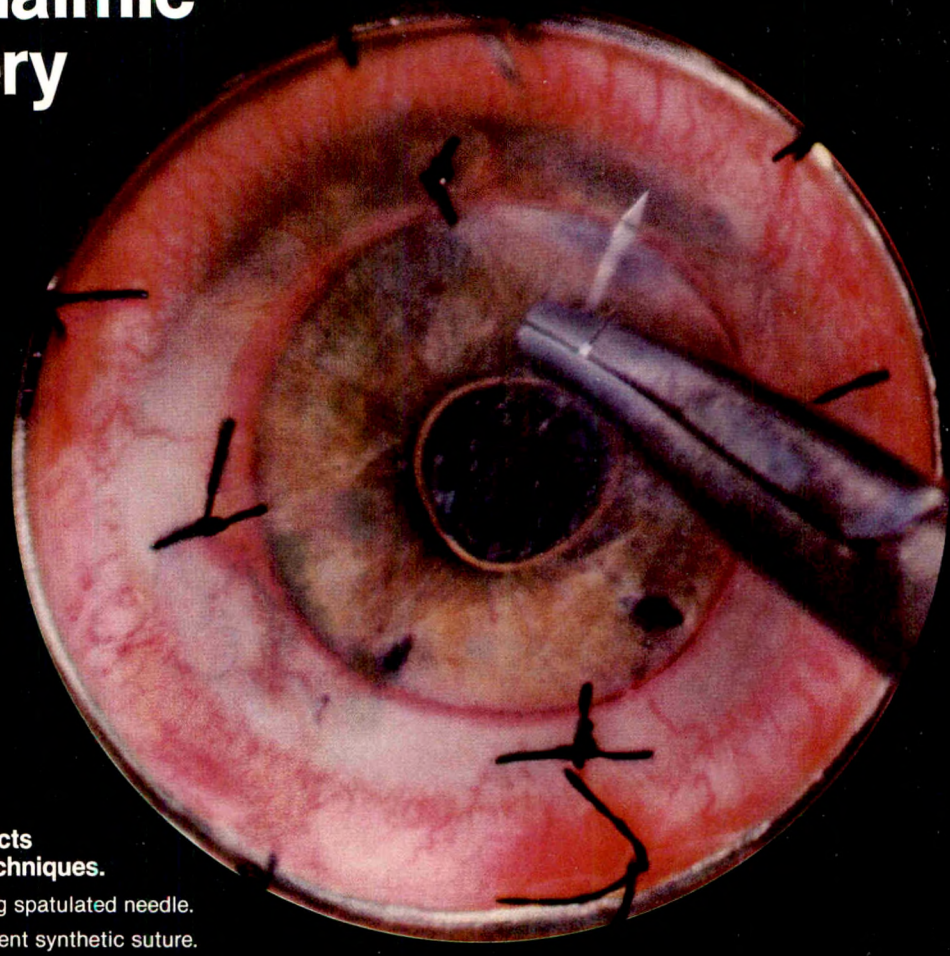
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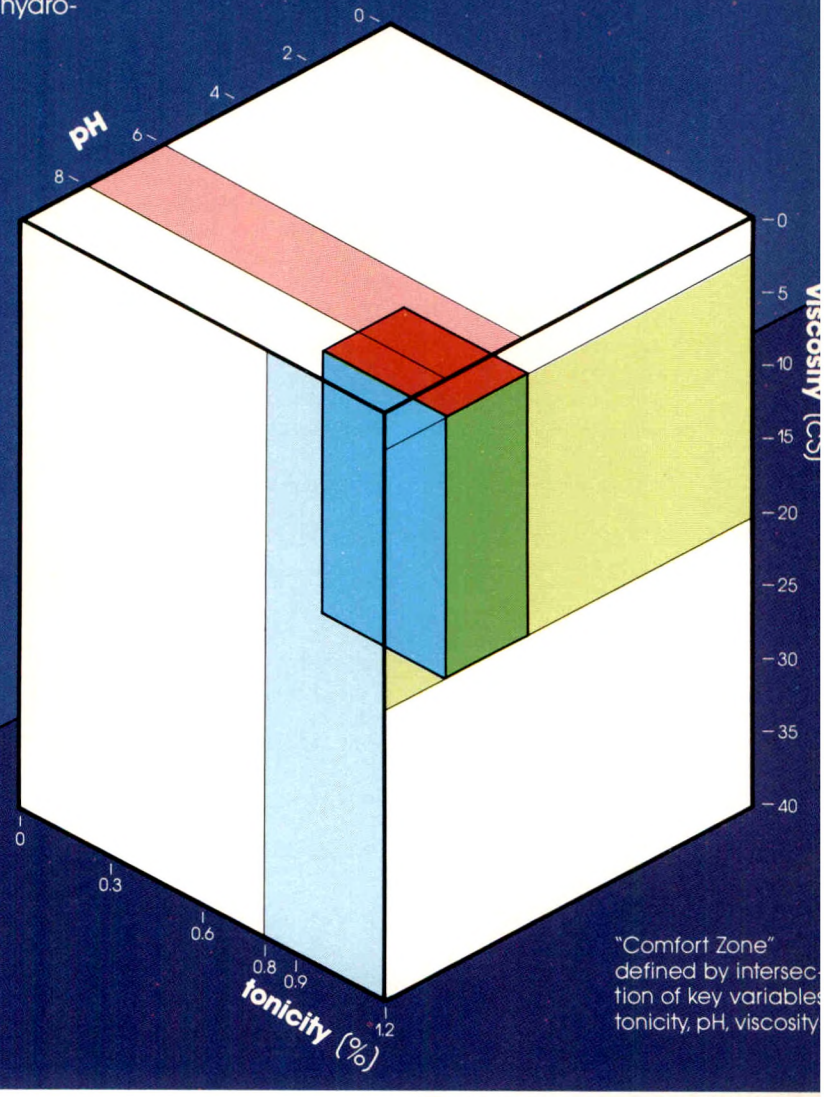
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1. Leibowitz HM, Kupferman A. Bioavailability and therapeutic effect of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 1975 Jan/Feb; 79(1):OP78-OP88.
2. Ibid.

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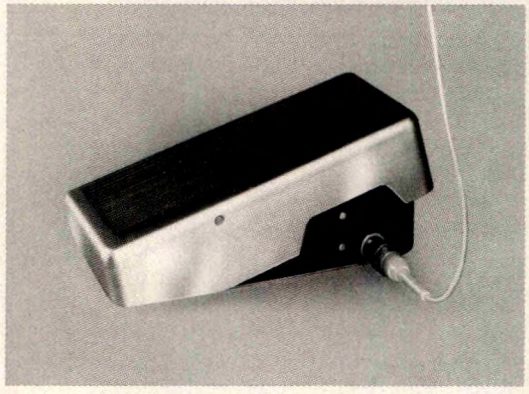
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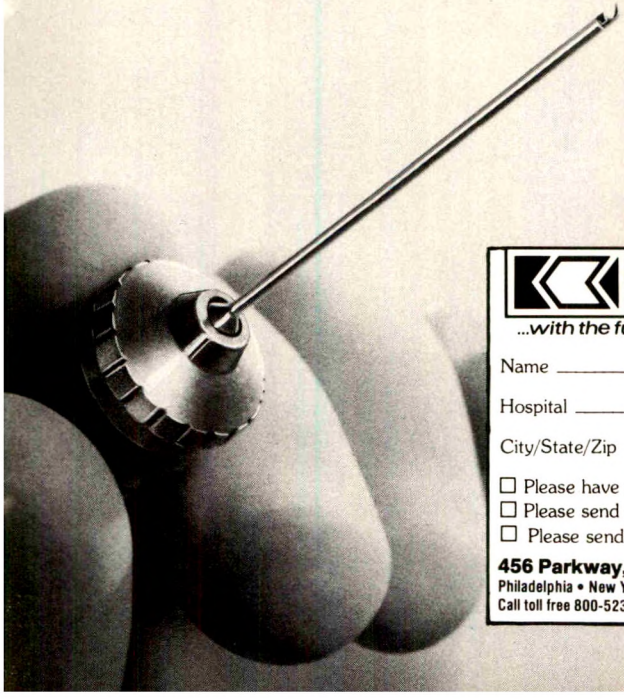
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Diabetic Renal-Retinal Syndrome

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This book provides a comprehensive analysis of the syndrome of eye and kidney failure that affects over half of all long term insulin-dependent diabetics. It contains up-to-date reviews and suggestions for therapy by ophthalmologists, endocrinologists, and nephrologists. It presents recent therapeutic advances in dialysis and renal transplantation. Also included is material on future diabetic management employing pancreas and islet of Langerhans transplants.

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An Extended Analysis

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Foreword by PROF. dr. HANS GOLDMAN

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CONTENTS: Psychophysical Localization of Retinal Lesions Methodology for Psychophysical Testing. Remission of Anomalies: An Important Aid to Analysis. Outer Retinal Pathology. Inner Retinal Pathology. Retinal Receptive Field-like Properties in Primary Open-Angle Glaucoma. Functional Time-Dependent Reductions in Sensitivity in Diseases of the Optic Nerve and the Visual Pathways: Use of the Flashing Repeat Static Test (FRST).

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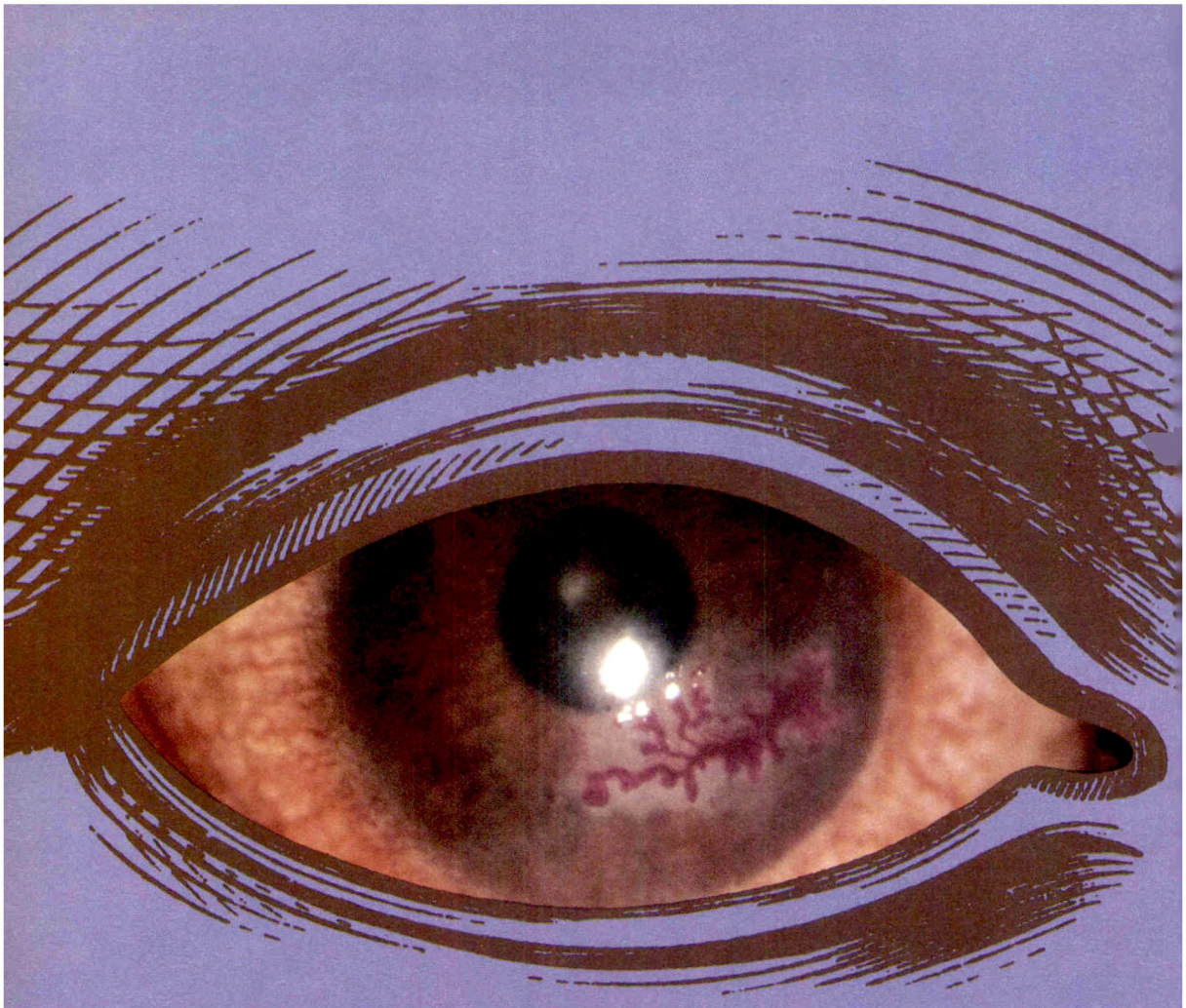
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Indication: Herpes simplex keratitis (topical use only).

Contraindications: Known or suspected hypersensitivity to any of the components.

Precautions: If there is no response after 7 or 8 days, other therapy should be considered. Recommended frequency and duration of administration should not be exceeded (usually not longer than 21 days). Not effective in corneal inflammations if herpes simplex is not present. Boric acid should not be used concomitantly. To insure that stability is maintained, the solution should not be mixed with other medications. Studies of mutagenicity and oncogenicity have been inconclusive.

Administer with caution in pregnancy or women of childbearing potential. In animal studies, fetal abnormalities have been reported. It is not known whether 'Stoxil' is secreted in human milk. In general, nursing should not be undertaken while on drug therapy, since many drugs and their metabolites are excreted in human milk.

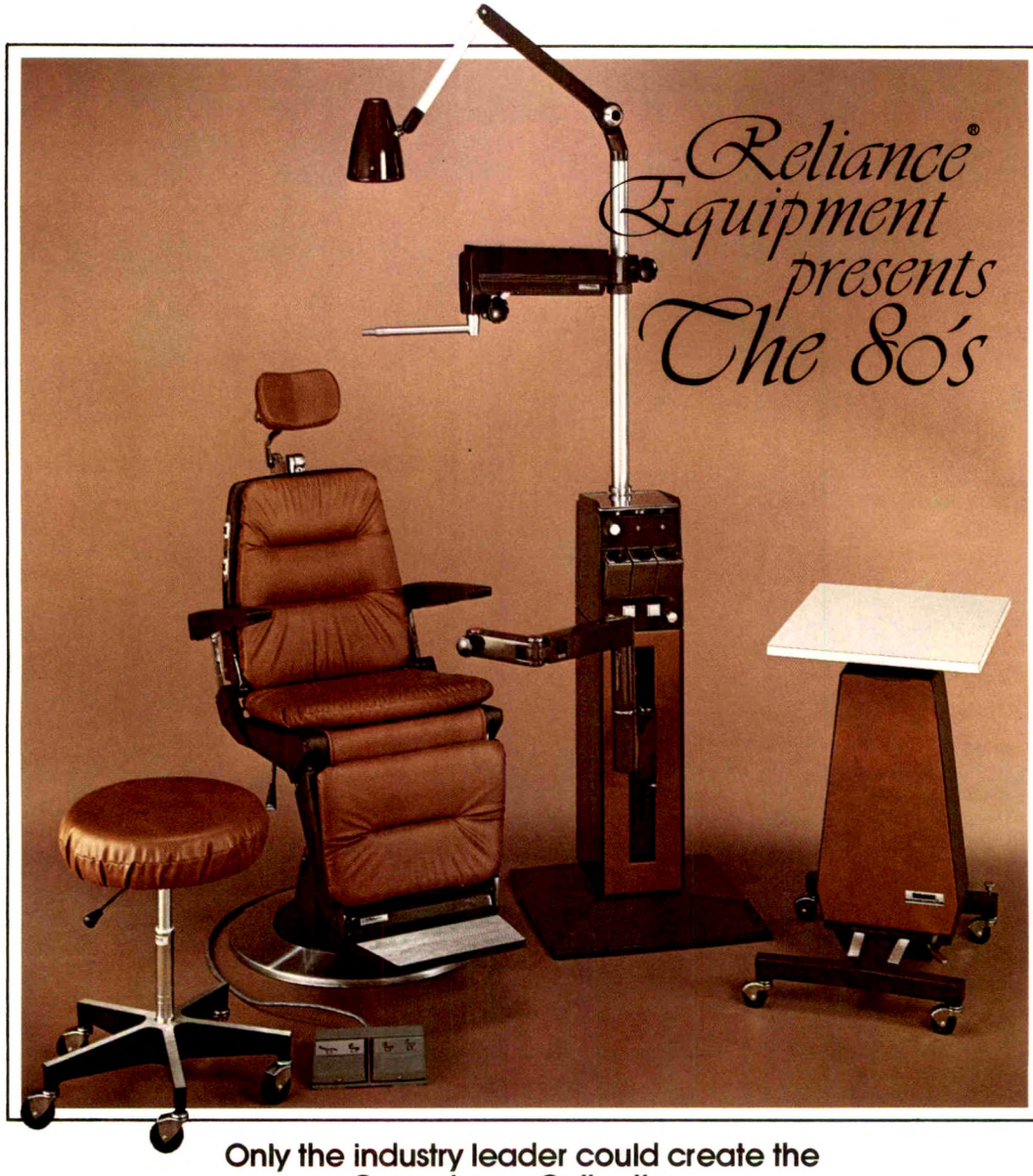
Adverse Reactions: Occasionally, irritation, pain, pruritus, inflammation, or edema of the eye or lid; rarely, allergic reactions have been reported. Photophobia has occurred. Occasionally, small punctate defects (which may be a manifestation of the infection), corneal clouding, and stippling of the corneal epithelium have been observed.

Supplied: 0.1% Ophthalmic Solution (1 mg./ml.) in 15 ml. bottles with dropper; 0.5% Ophthalmic Ointment (5 mg./gram) in 4 gram tubes.

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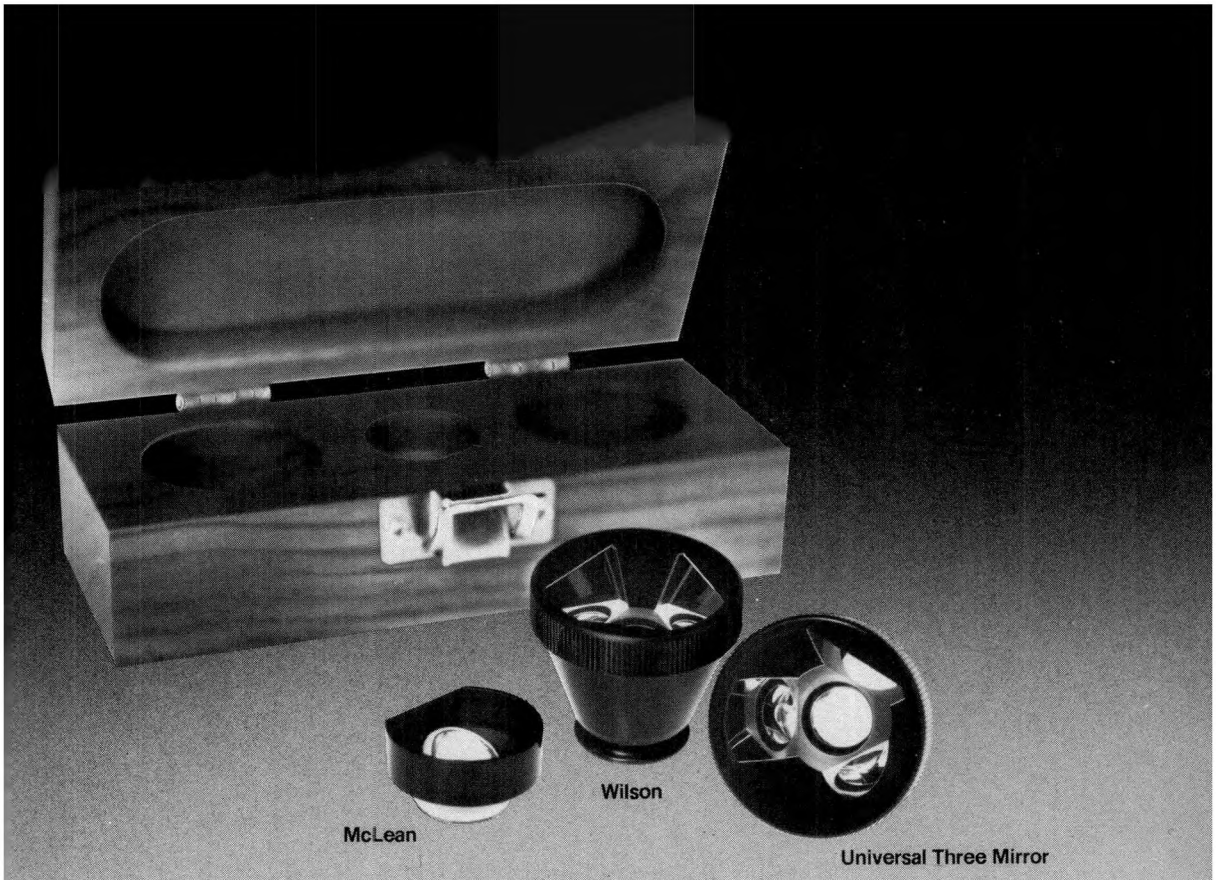
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The fee for ophthalmologists is $300. And a special one-day course for technicians on Saturday April 25, is $50.

So plan now to attend the 1st Annual International Eye Congress in Atlanta. Attendance is limited to 300.

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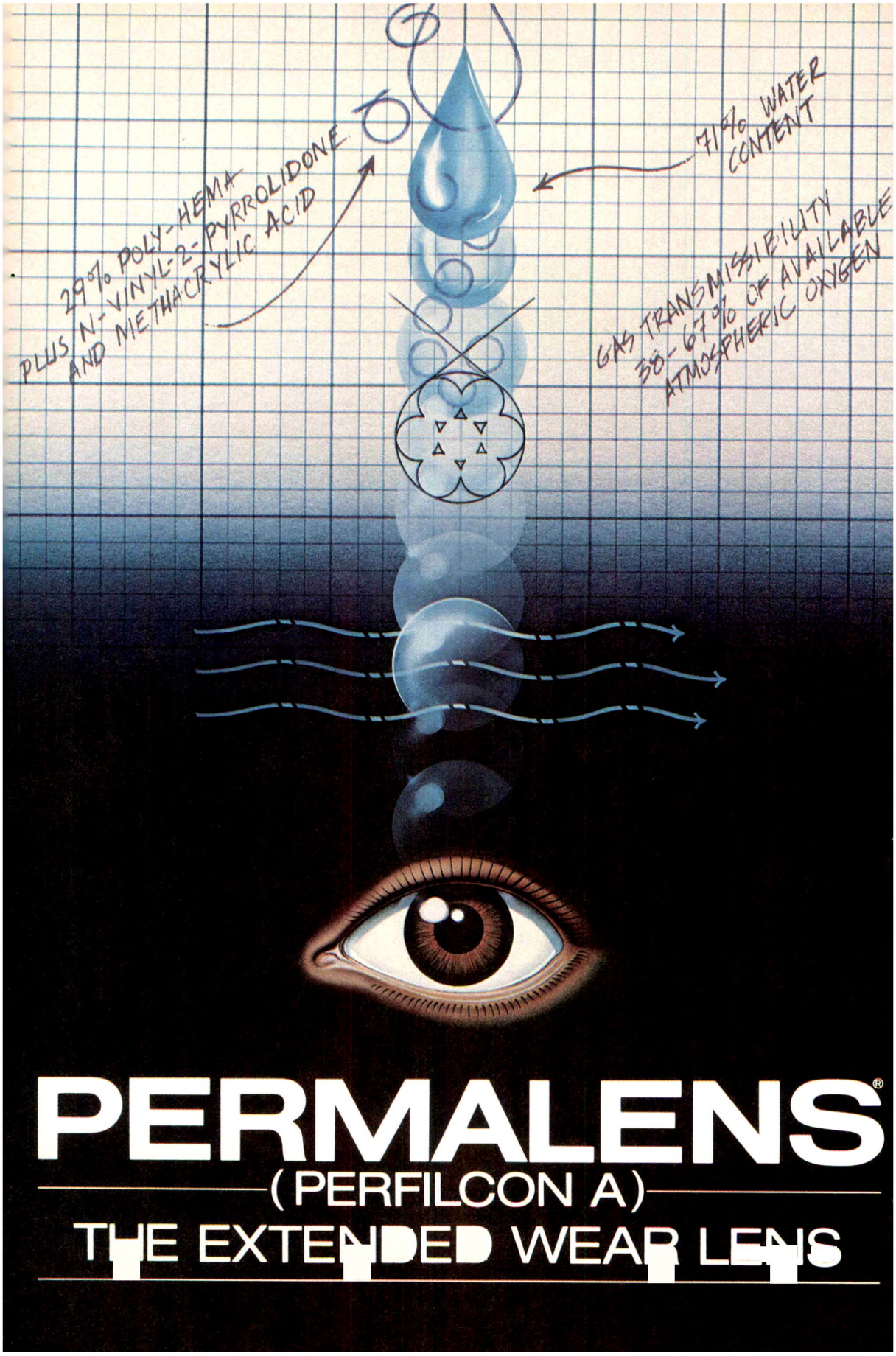
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This response is partly due to the Permalens® material, perfilcon A, which combines high water content, high oxygen permeability, and dimensional stability. These properties were designed into the lens to facilitate oxygen transfer directly through the lens, so the cornea can receive an oxygen supply adequate to maintain itself in a healthy state.

These properties also make Permalens® Contact Lenses more supple, and therefore comfortable on the patient's eyes. Patients have reported, in fact, they're the next best thing to wearing no lens at all.

Easy to fit, Permalens® Contact Lenses are available in a range of powers from +11.00 to +17.00 diopters. And with just four parameters, the lens fits most corneal sizes and curvatures.

Maintenance, too, is uncomplicated. Since Permalens® Contact Lenses usually need only be removed once a month, cleaning and disinfection are simplified. The preferred regimen is Preflex®/Flexsol®/Normol® (Thermal regimens can also be used.) For re-hydration during lens wear, Clērz® or Adapettes® eye drops are available.

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Permalens®
(perfilcon A)

Hydrophilic Contact Lens
For Extended Wear
Post-Cataract (Aphakic) Use

For full prescribing information, see next page.

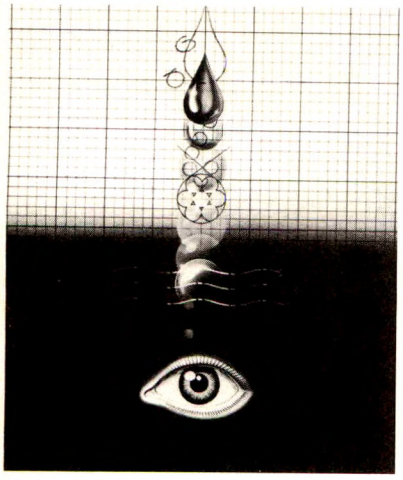
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Permalens®
(perfilcon A)

Hydrophilic Contact Lens For Extended Wear Post-Cataract (Aphakic) Use

DESCRIPTION

The **Permalens**® (perfilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The lens material, perfilcon A, is a terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone, and methacrylic acid, with ethyleneglycol dimethacrylate as a crosslinking agent. The hydrated lens consists of 29% perfilcon A and 71% water by weight. The gas permeability of **Permalens**® Hydrophilic Contact Lenses, using the Fatt method,[1] has been determined to be: 42.0×10^{-11} $cm^2 \cdot ml\ O_2/sec \cdot ml \cdot mm\ Hg$ at 36°C.

ACTIONS

When placed on the human cornea, the hydrated **Permalens**® Hydrophilic Contact Lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS AND USAGE

Aphakia

Permalens® (perfilcon A) Hydrophilic Contact Lenses are indicated for extended-wear aphakic vision correction in persons with non-diseased eyes.

Note: Supplemental spectacles may be required, in some conditions, to provide near vision or to compensate for uncorrected refractive astigmatism.

CONTRAINDICATIONS

Permalens® Hydrophilic Contact Lenses are contraindicated by the presence of any of the following conditions:

1. Acute and subacute inflammations of the anterior segment of the eye.
2. Any eye disease which affects the cornea or conjunctiva.
3. Insufficiency of lacrimal secretion.
4. Corneal hypoesthesia.
5. Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.

WARNINGS

Abrasions and Infections

If a lens becomes less comfortable than it was when it was first placed on the wearer's cornea, the lens should be removed and inspected for the presence of a foreign body, lens coating, deposits, or lens damage. If examination of the patient's eye reveals any eye abrasion, ulceration, irritation or infection, or any other abnormal eye condition is observed concurrently with lens wear, the lens should be removed immediately and a physician consulted.

Patients should not be fitted with Permalens® (perfilcon A) Hydrophilic Contact Lenses during the post-operative period until, in the opinion of the surgeon, the eye has healed completely.

Medications and Eye Drops

Permalens® Hydrophilic Contact Lenses must be stored in an appropriate solution when off the eyes. The solution used is dependent on the system selected for disinfection. When the lenses are disinfected with the thermal disinfection system, they may be stored in PERMASOL™ Rinsing and Storage Solution (sterile buffered balanced salt solution preserved with edetate disodium 0.1% and thimerosal 0.001%) or BOILnSOAK® Sterile Saline Solution (sterile buffered isotonic solution containing boric acid, sodium borate, sodium chloride 0.7%, preserved with Thimerosal (Lilly) 0.001% and edetate disodium 0.1%). When lenses are disinfected with the chemical disinfection system, they may be stored in FLEXSOL® Sterile Disinfection and Storage Solution (sterile buffered isotonic solution of sodium chloride, sodium borate, boric acid, polyvinylpyrrolidone, polyoxyethylene and polyoxypropylene, preserved with Thimerosal (Lilly) 0.001%, chlorhexidine 0.005% and edetate disodium 0.1%).

Only the above solutions and the following may be used with **Permalens**® Hydrophilic Contact Lenses:

1. PREFLEX® Sterile Cleaning Solution (sterile solution of a nonionic cleaner preserved with Thimerosal (Lilly) 0.004% and edetate disodium 0.2%).
2. NORMOL® Sterile Rinsing Solution (sterile buffered isotonic aqueous solution of sodium chloride, sodium borate and boric acid, preserved with Thimerosal (Lilly) 0.001%, edetate disodium 0.1% and chlorhexidine 0.005%).
3. CLERZ® Lubricating and Rewetting Eye Drops (sterile solution preserved with edetate disodium 0.1% and Thimerosal (Lilly) 0.001%).
4. ADAPETTES® Sterile Lubricating Solution (buffered isotonic aqueous solution containing Adsorbobase® (polyvinylpyrrolidone with other water-soluble polymers), Thimerosal (Lilly) 0.004%, and edetate disodium 0.1%).

No conventional (hard) contact lens solution should be used. Only those solutions specifically recommended by your eye-care practitioner should be used during wear.

WEARING RESTRICTIONS

Permalens® (perfilcon A) Hydrophilic Contact Lenses should not be worn while swimming or in the presence of noxious or irritating vapors.

Lens-Care Regimen

Patients must adhere to the recommended lens-care regimen of **Permalens**® Hydrophilic Contact Lenses. Failure to follow this procedure may result in the development of serious ocular infections which might result in corneal ulcers.

PRECAUTIONS

Storage

Permalens® Hydrophilic Contact Lenses must be stored in the appropriate storage solution: PERMASOL™ Rinsing and Storage Solution, BOILnSOAK® Sterile Saline Solution, or FLEXSOL® Sterile Disinfection and Storage Solution, depending on disinfecting methods used. If left exposed to air, the lenses will dehydrate, become brittle and break readily. If a lens dehydrates, it should be soaked in one of the following until it returns to a hydrated (soft) pliable state:

1. PERMASOL™ Rinsing and Storage Solution
2. BOILnSOAK® Sterile Saline Solution
3. FLEXSOL® Sterile Disinfection and Storage Solution

PERMA-case™ Contact Lens Storage and Carrying Case should be used for storing lenses.

Hygiene

Hands must be washed and rinsed thoroughly, and dried before handling the lenses. Cosmetics, lotions, soaps and creams must not come in contact with the lenses since eye irritation may result. If hair spray is used while the lenses are being worn, the eyes must be kept closed until the spray has settled.

Cleaning and Disinfecting

Prior to wearing, **Permalens**® (perfilcon A) Hydrophilic Contact Lenses must be both cleaned and disinfected.

Cleaning: Monthly removal for cleaning is recommended. However, at the discretion of the eye-care practitioner, the lenses may be removed at a frequency adjusted to the needs of each patient. Each time the lenses are removed from the wearer's eyes, both surfaces of the lenses must be cleaned using several drops of PREFLEX® Sterile Cleaning Solution. Between regular cleaning periods, CLERZ® Lubricating and Rewetting Eye Drops or ADAPETTES® Sterile Lubricating Solution may be used to maintain lens hydration while being worn.

Routine Thermal Disinfection Method: Permalens® Hydrophilic Contact Lenses may be effectively disinfected using a low-heat disinfecting unit approved for use with hydrophilic contact lenses. After cleaning with PREFLEX® Sterile Cleaning Solution, rinse lenses with PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution, and disinfect in a RINCON* low-heat Thermal Disinfection Unit or other approved unit. The lens storage containers must be emptied and filled with fresh PERMASOL™ Rinsing and Storage Solution or BOILnSOAK® Sterile Saline Solution just prior to disinfecting the lenses.

Alternate Thermal Disinfection Method: If a thermal unit is not available, the following disinfecting system can be used temporarily. After cleaning the lenses according to instructions, fill each chamber of the PERMA-case™ with PERMASOL™ or BOILnSOAK® Bring a pan of water to a rolling boil. Remove the pan from heat. Place the sealed carrying case into hot water and allow to cool. When the water is cool, lenses have been disinfected.

NOTE: USE OF THE RINCON* THERMAL DISINFECTION UNIT SHOULD BE RESUMED AS SOON AS POSSIBLE.

Chemical Disinfection Method: Disinfection with PREFLEX® Sterile Cleaning Solution, FLEXSOL® Sterile Disinfection and Storage Solution and NORMOL® Sterile Rinsing Solution has been shown to be an effective disinfection system. Following each removal, **Permalens**® Hydrophilic Contact Lenses must be cleaned with PREFLEX® Sterile Cleaning Solution and rinsed with NORMOL® Sterile Rinsing Solution. Each time the lens is stored, the PERMA-case™ Contact Lens Storage and Carrying Case must be emptied and refilled with fresh FLEXSOL® Sterile Disinfection and Storage Solution. Fresh FLEXSOL® Sterile Disinfection and Storage Solution must also be used when disinfecting a lens.

WARNING: FLEXSOL® STERILE DISINFECTION AND STORAGE SOLUTION SHOULD *NEVER* BE USED WIT

LENS EJECTION

Occasionally, a lens may become partially dehydrated dur sleep. This may result in the lens being dislodged from the lid action or rubbing the eyelids. Dislodged or routinely re lenses should be thoroughly rehydrated in one of the follo solutions:

1. PERMASOL™ Rinsing and Storage Solution
2. NORMOL® Sterile Rinsing Solution
3. BOILnSOAK® Sterile Saline Solution

After the lens returns to a soft, supple state it should be cle and disinfected prior to replacement on the eye.

ADVERSE REACTIONS

Serious corneal damage may result from wearing lenses w have been soaked in conventional hard contact lens solutio Eye irritation may occur within a short time after putting on stored in a solution of improper pH or tonicity. Removal of lens will relieve the irritation.

Excessive tearing or redness, unusual eye secretions, redu visual acuity, and photophobia are not normal; if these symp occur, the patient should be examined by an eye-care prac to determine the cause.

DOSAGE AND ADMINISTRATION

Fitting

Conventional methods of fitting contact lenses do not apply **Permalens**® (perfilcon A) Hydrophilic Contact Lenses. For a detailed description of the fitting technique, refer to the **Per lens**® Hydrophilic Contact Lens Professional Fitting Guide, copies of which are available from Cooper Laboratories, Inc N. Whisman Road, Mountain View, California 94043.

WEARING SCHEDULE

Permalens® Hydrophilic Contact Lenses are indicated for extended-wear (24-hour) use, without a gradual daily increa wearing time required. Cleaning is recommended at monthl intervals. However, at the discretion of the eye-care practitio the lenses may be removed at a frequency adjusted to the n of each patient.

Lens Care and Handling

Care must be taken on the initial visit to ensure that the patie is supplied with all necessary accessory products and fully u stands all care and handling instructions for the lenses. Regu post-fitting visits are necessary to assure patient health and compliance with instructions.

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expirat date of the lens.

Accessories

Patient Care Kit	Cat. #80
Wearer's Manual	Cat. #80
PERMA-case™ Contact Lens Storage and Carrying Case	Cat. #80
FLEXSOL® Sterile Disinfection and Storage Solution*	Cat. #90
NORMOL® Sterile Rinsing Solution*	Cat. #90
PREFLEX® Sterile Cleaning Solution*	Cat. #90
CLERZ® Lubricating and Rewetting Eye Drops	Cat. #80
PERMASOL™ Rinsing and Storage Solution	Cat. #80
RINCON® Thermal Disinfection Unit**	Cat. #90
BOILnSOAK® Sterile Saline Solution*	Cat. #90
ADAPETTES® Sterile Lubricating Solution	Cat. #90

CAUTION: Federal Law Prohibits Dispensing Without Prescription

[1] Fatt I, Morris JA. A survey of gas-permeable contact lenses. *The Optician*: 27-36 (November 4, 1977)

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The VISC-X system offers you more options than any similar instrument. Approaches and techniques with multifunctional or single function probes may be employed.

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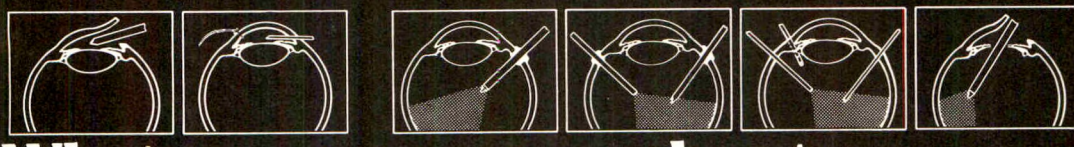
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What are your approaches to Anterior/Posterior Segment Surgery?

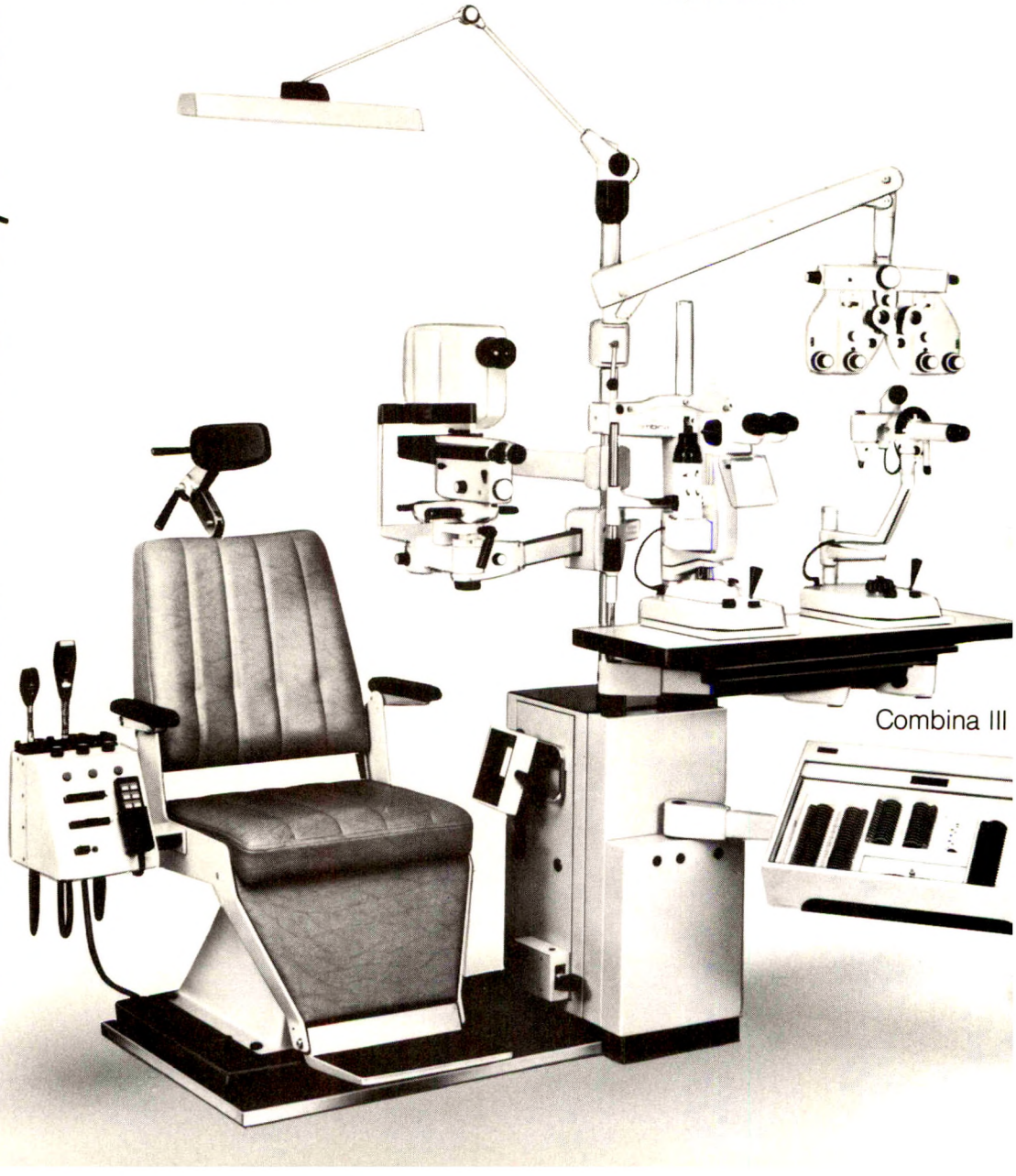
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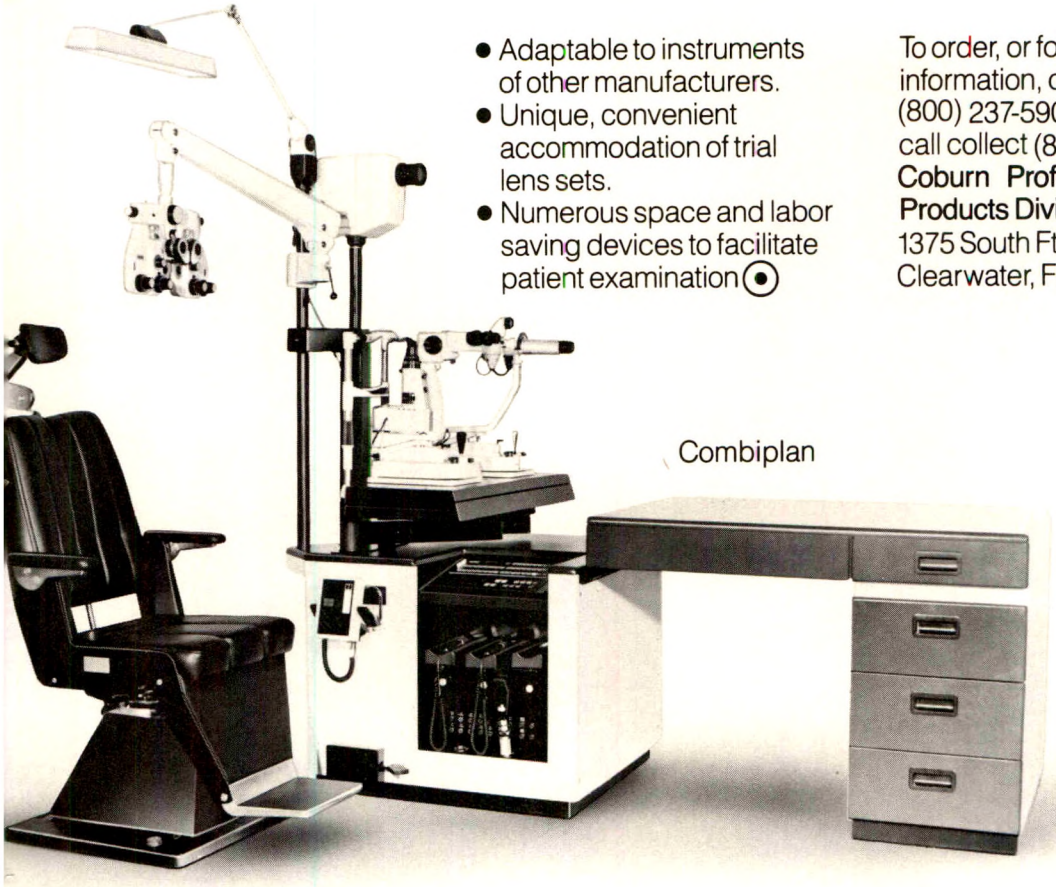


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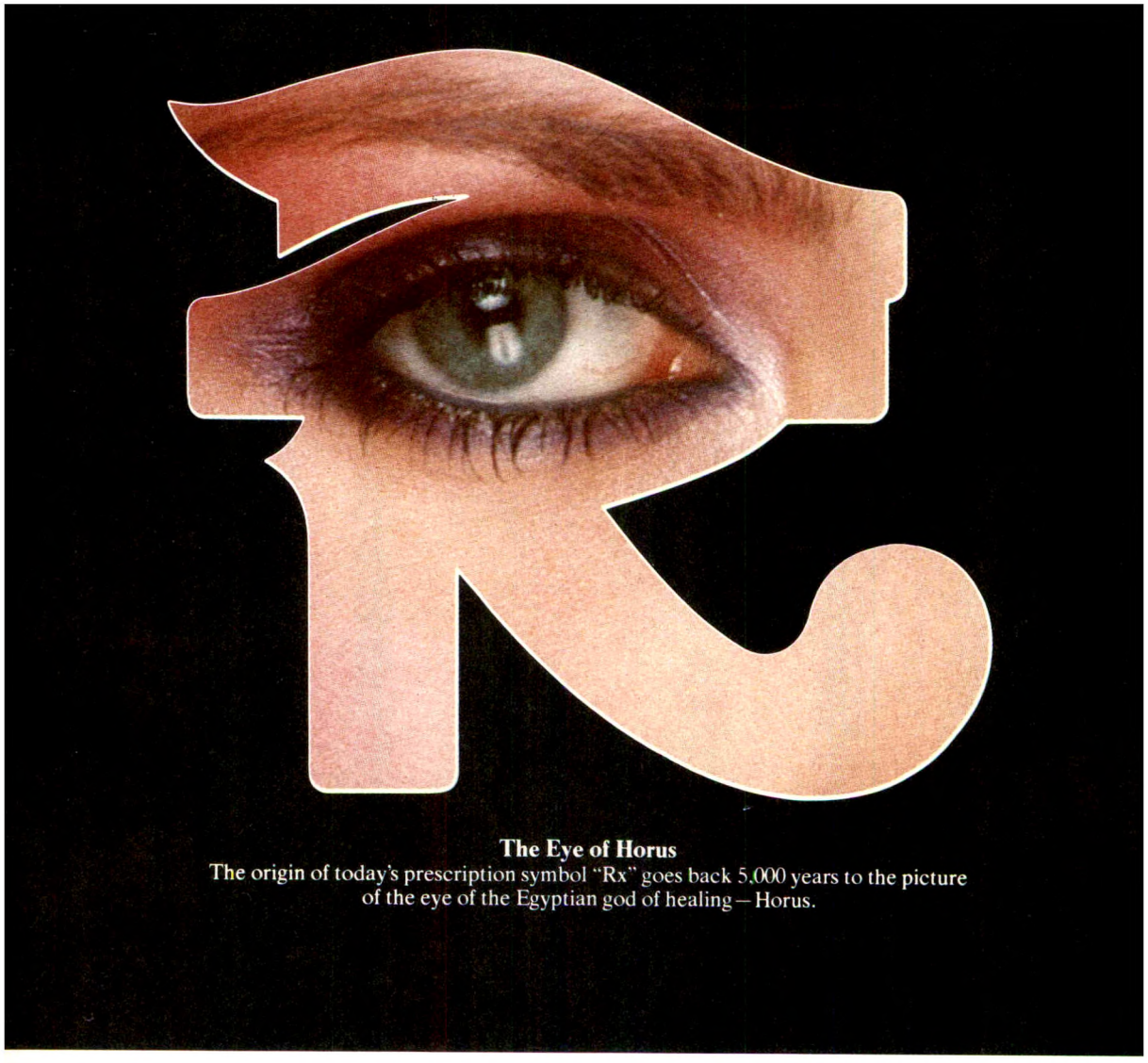
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Combiplan



Combistyle



The Eye of Horus

The origin of today's prescription symbol "Rx" goes back 5,000 years to the picture of the eye of the Egyptian god of healing—Horus.

DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2'-deoxyuridine.

VIROPTIC sterile opthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2'-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

For Topical Treatment of
Epithelial Keratitis Caused by Herpes Simplex Virus

VIROPTIC®

(Trifluridine)

OPHTHALMIC SOLUTION, 1%

STERILE

95% of Patients Responded*

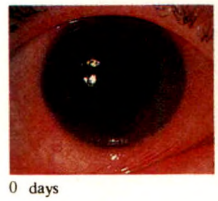
During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

Significantly More Convenient

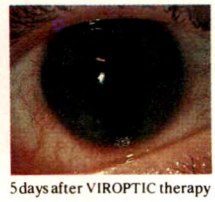
- Less frequent applications
- No middle of the night dosage
- None of the blurring and inconvenience of ointments

5 to 9 Drops Daily

One drop every 2 hours while awake until herpetic lesion has completely re-epithelialized, then one drop every 4 hours for 7 days post-re-epithelialization.



0 days



5 days after VIROPTIC therapy

Effective in 138 of 150 patients* Unresponsive or Intolerant to Idoxuridine or Vidarabine

In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

*Data on file, Medical Division, Burroughs Wellcome Co.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml. Store under refrigeration 2° to 8° C (36° to 46° F).

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MARCH 15-21, 1981
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President: R. Witmer, M.D.
Vice President: Mikhail Krasnov, M.D.

Monday, March 16

Speakers

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ROBERT F. AZAR, MD
STANLEY C. BECKER, MD
HUGH BECKMAN, MD
ERNST van BEUNINGEN, MD
G. BISCHOF, MD
JAMES C. BOBROW, MD
I.E. CAIRNS, MD
DIETER FRIEDBURG, MD
MAURICE LANGHAM, PhD
NORMAN S. LEVY, MD, PhD
MICHAEL E. LIEPPMAN, MD
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RALPH S. SANDO, MD
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John Bellows, M.D. Director

Sunday, March 15 **OCULOPLASTIC SURGERY SEMINAR.** Chairman: Pierre Guibor, M.D.

PARTICIPANTS

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Anders Borthne, MD
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William R. Thornton, MD
David E. Savar, MD
Ernst Nicolitz, MD
John G Yassin, MD
Roger Kohn, MD
Marsha Guibor, CO

Monday, March 16 **GLAUCOMA SEMINAR (See Above)**

Tuesday, March 17 **CATARACT SEMINAR.** *Chairman:* Richard Troutman, M.D. *Speakers:* Donald Willard, M.D. (Easton, Herbert Kaufman, M.D. (New Orleans), Charles D. Kelman, M.D. (New York), Robert F. Azar, M.D. (New Orleans), Harold Scheie, M.D. (Philadelphia), Edward Shaw, M.D. (Phoenix), Donald L. Praeger, M.D. (Poughkeepsie), Steven P. Shearing, M.D. (Las Vegas), David Miller, M.D. (Boston), Robert Stegmann, M.D. (Boston), Charles W. Bechert II, M.D. (Ft. Lauderdale), Miles Galin, M.D. (New York), Jack Hartstein, M.D. (St. Louis), William S. Harris, M.D. (Dallas), William. C. Simcoe, M.D. (Tulsa), Jack Kayes, M.D. (St. Louis).

Wednesday, March 18 **RETINA-CHOROID-VITREOUS SEMINAR.** *Chairman:* Howard Schatz, M.D. *Speakers:* Marvin F. Kraushar, M.D. (New York), Daniel F. Finkelstein, M.D. (Baltimore), Lawrence J. Singerman, M.D. (Cleveland), Peter H. Morse, (Chicago), Robert Massof, Ph.D. (Baltimore), Howard Schatz, M.D. (San Francisco). Ronald G. Michels, M.D. (Baltimore), Theodore F. Schaegel, Jr, M.D. (Indianapolis).

Thursday, March 19 **CONTACT LENS SEMINAR.** *Chairman:* Jack Hartstein, M.D.*Speakers:* Mathea R. Allansmith, M.D. (Boston), H. Dwight Cavanagh, M.D. (Atlanta), Joseph A. Baldone, M.D. (New Orleans), Paul R. Honan, M.D. (Indianapolis), James M. Gordon, M.D. (St. Louis), John F. Morgan, M.D. (Kingston), Harold A. Stein, M.D. (Toronto), Bernard Slatt, M.D. (Scarborough), Ellis Gruber, M.D. (Rochester), Dean Clements, (Rochester) Gerald Feldman, Ph. D. (San Diego), Louis A. Wilson, M.D. (Atlanta). Oliver H. Dabezies, Jr., M.D. (New Orleans).

Friday, March 20 **CORNEA AND EXTERNAL DISEASES SEMINAR.** *Chairman:* Claes Dohlman, M.D. *Speakers:* Claes Dohlman, M.D. (Boston), Mathea Allansmith, M.D. (Boston), H. Dwight Cavanagh, M.D. (Atlanta), David Miller, M.D. (Boston), George O. Waring, M.D. (Atlanta), Lance S. Forstot, M.D. (Denver), Robert Abel, M.D. (Wilmington), Herbert Kaufman, M.D. (New Orleans).

Saturday, March 21 **REFRACTIVE SURGERY SEMINAR.** *Chairman:* Herbert E. Kaufman, M.D. *Speakers:* Herbert E. Kaufman, M.D. (New Orleans), S. Fyodorov, M.D. (Moscow), Leeds Katzen, M.D. (Baltimore), Theodore P. Werblin, M.D. (New Orleans), Keith S. Morgan, M.D. (New Orleans), Leo Bores, M.D. (Detroit), Anthony Nesburn, M.D. (Los Angeles), Richard Troutman, M.D. (New York), Herbert Katzin, M.D. (New York), Miles Friedlander, M.D. (New Orleans).

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Pathology
Ocular Motility
Optics and Refraction
Pharmacology and Therapeutics
Anterior Segment Diseases
Vitreoretinal Disorders
Neuro-ophthalmology

SPECIAL COURSES
Workshops, Tutorials and Review/Refresher Courses

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3	External Ocular Diseases	2	8	$125
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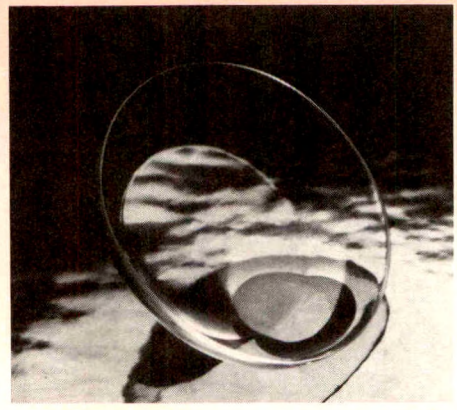
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For more information on the high water Sauflon PW lens, call us or your local Sauflon distributor.

1. Fatt, I. and Morris, J., "A Survey of Gas-Permeable Contact Lenses." The Optician, November, 1977.

2. Clinical data on file at Heyer-Schulte Medical Optics Center.

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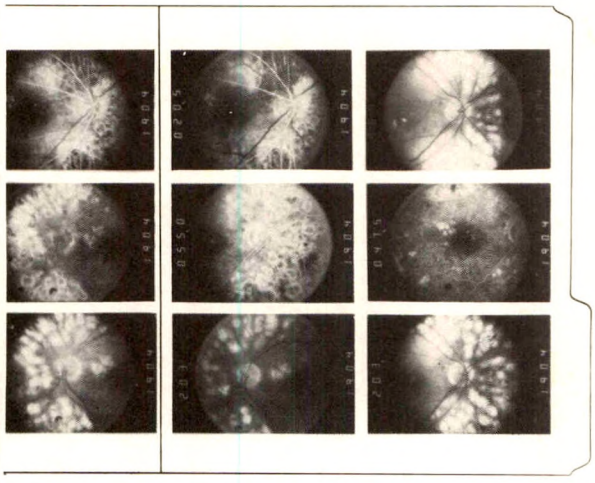
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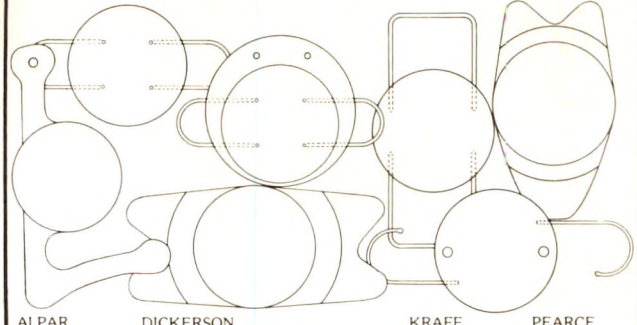
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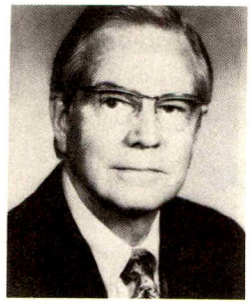
SEVENTEENTH ANNUAL SPRING MEETING

Friday and Saturday,
March 27-28, 1981

Pittsburgh Hilton
Pittsburgh, Pa.

FACULTY

D. Jackson Coleman, M.D., New York
Richard H. Keates, M.D., Columbus
Steven M. Podos, M.D., New York
Norman J. Schatz, M.D., Philadelphia



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A. EDWARD MAUMENEE, M.D.

William Holland Wilmer Professor Emeritus (Active Staff)
of Ophthalmology
and
Former Director, Department of Ophthalmology,
The Wilmer Ophthalmological Institute
The Johns Hopkins University School of Medicine
and The Johns Hopkins Hospital,
Baltimore, Maryland

Dr. Maumenee will present the First Annual Harvey E. Thorpe Lecture

FOR NURSES AND ALLIED OPHTHALMIC PERSONNEL
A full day symposium on "The Role of the Nurse and the Technician in Ophthalmology," Saturday, March 28.
Please indicate interest when responding for registration information.

REGISTRATION FEES:
Physicians – $150. (Meals included.)
Residents – $75. (Meals included.)

Approved for AMA Continuing Education Credit Category 1
(12 Credit Hours) by the Pennsylvania Academy of Ophthalmology
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For registration and Nurses' Symposium information, contact:
Frank Cignetti, M.D.
Program Co-Chairman, Spring Meeting
Pittsburgh Ophthalmology Society
2545 Mosside Blvd.
Monroeville, Pa. 15146
(412) 356-8815

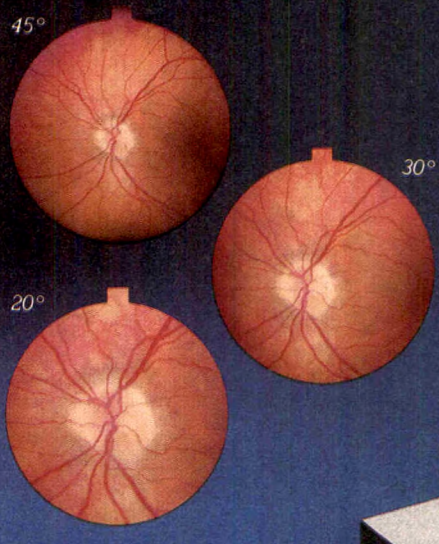
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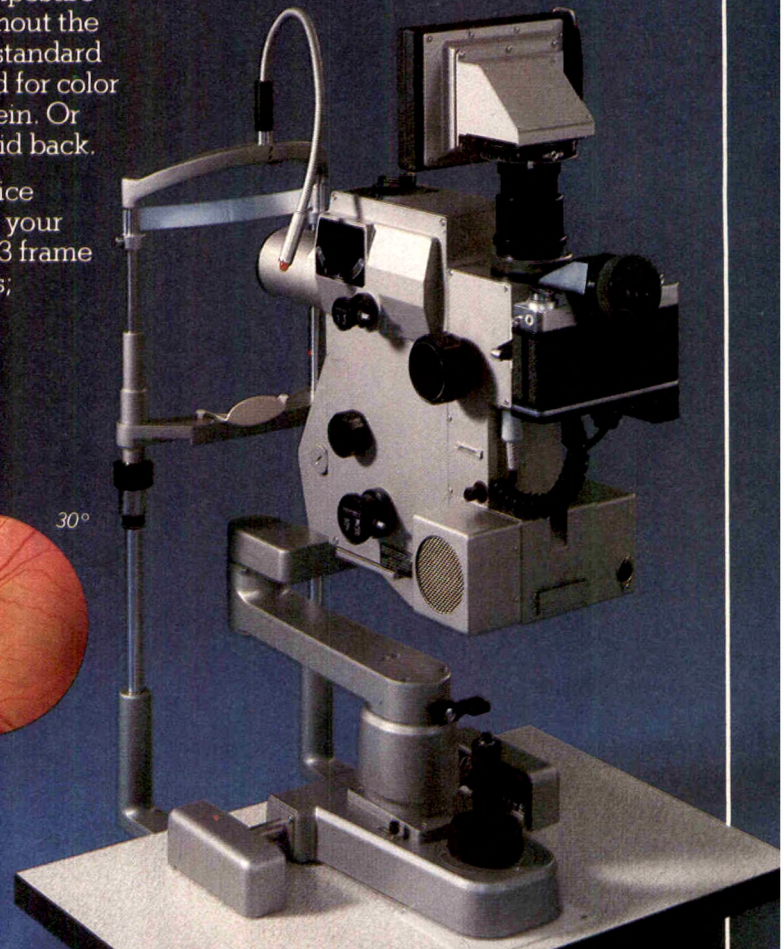
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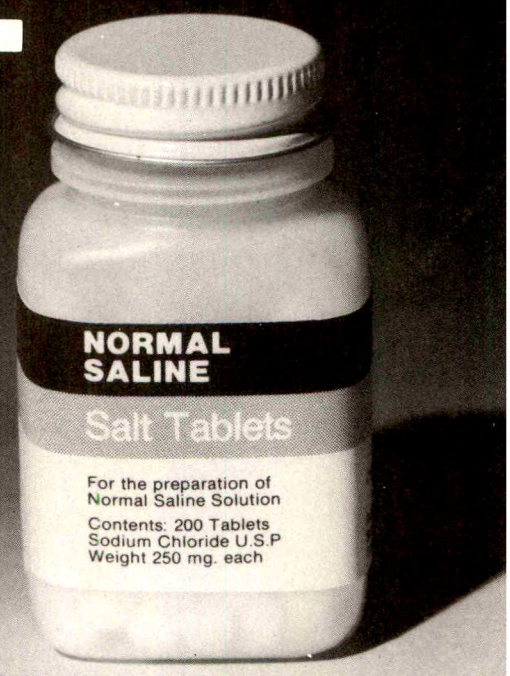
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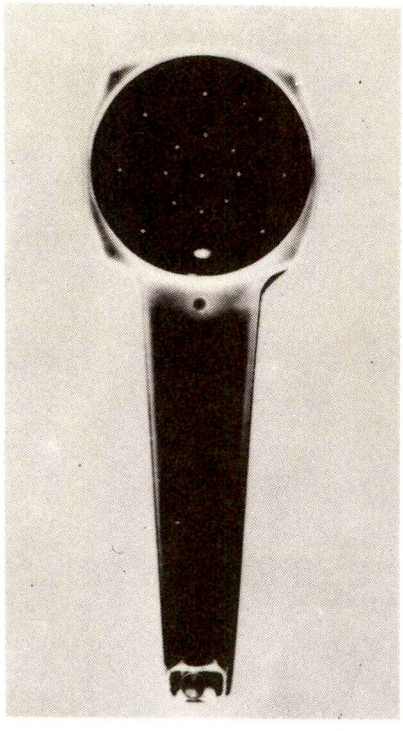
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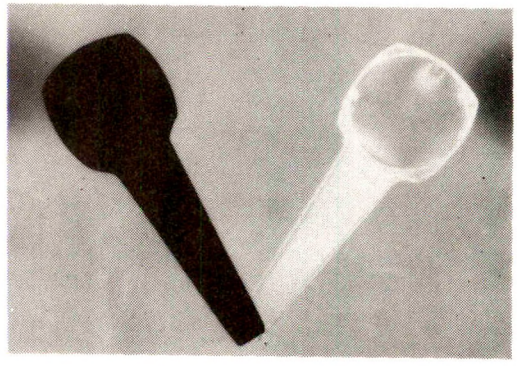
A PLUS FOR OPHTHALMOLOGY WITH VELOTRON'S PINHOLE PLUS

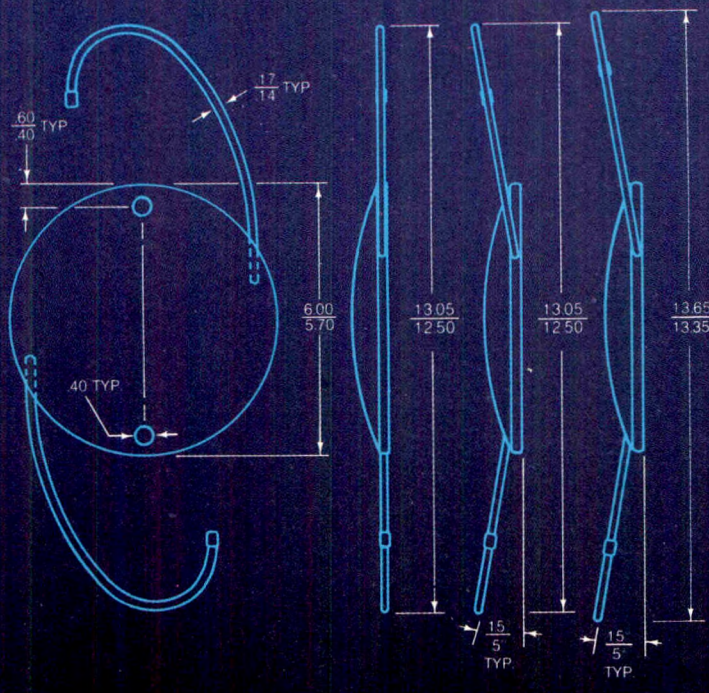
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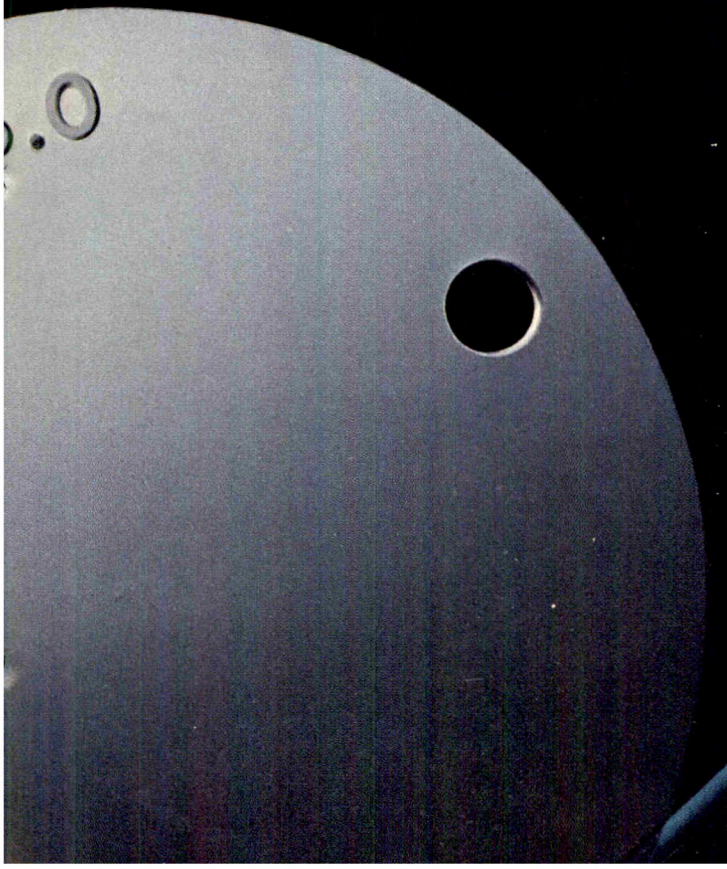
MODEL 103 J/M/L SINSKEY

Starting in 1977, with the introduction of the IOLAB Model 101, a surge of interest has developed among ophthalmologists for positioning intraocular lenses in the posterior chamber.

This year, IOLAB introduced the Model 103 J/M/L lens designed by Robert M. Sinskey, M.D., of Santa Monica, California. This posterior chamber lens has loops which are more open than the Model 101 and, while made of the same 5.0 PROLENE* polypropylene, the loops are continuously curved from the point of attachment to the optic so that the compression force is always at an angle to the main axis of the loop fiber. This design results in a reduction of the force required to compress the loop.

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On all IOLAB lenses the IODENTIFICATION™ marking system is clearly visible, with the dioptric power number and the IOLAB name embossed right on the lens. This unique concept insures that at the time of implantation the surgeon can be confident that the patient will receive a lens with the proper refraction. The IODENTIFICATION™ marking system guarantees the stated dioptric power to within ± 0.25d of the power embossed.



MODEL 103J
6mm optic with plano-convex design. 13mm haptic diameter, two positioning holes at 6 and 12 o'clock. Loops parallel to the plano surface.

MODEL 103B
Model 103J with blue loops.

MODEL 103M
Loops are angled at 10° toward the convex side.

MODEL 103N
Model 103M with blue loops.

MODEL 103L
Haptic diameter of 13.5 mm, loops angled 10° toward the convex side.

MODEL 103Q
Model 103L with blue loops.

CAUTION: Investigational device. Limited by Federal law to investigational use.

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ESTELLE DOHENY EYE FOUNDATION
AND
THE DEPARTMENT OF OPHTHALMOLOGY
UNIVERSITY OF SOUTHERN CALIFORNIA
ANNOUNCE
EXTRACAPSULAR CATARACT EXTRACTION
A PRACTICAL WORKSHOP
(INCLUDING POSTERIOR CHAMBER IOL'S)
FRIDAY AND SATURDAY, March 20-21, 1981
(Limited to 40 Participants)



ESTELLE DOHENY EYE FOUNDATION

OBJECTIVES:

1. Review indication, contraindications, complications, pre-op management and surgical techniques, etc. for extracapsular cataract extraction and posterior chamber lenses.
2. Supervised practice surgery on cadaver and animal eyes using various extracapsular techniques and posterior chamber IOL's.
3. Review video tapes of human surgery.

USC FACULTY

Course Director:
Ronald E. Smith, M.D.
Ronald Akashi, M.D.
George Baerveldt, M.D.
Kenneth R. Diddie, M.D.
Francis Hertzog, M.D.
Arthur Hurt, M.D.
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Nicholas Kokoris, M.D.
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James R. Wilson, M.D.

PROGRAM
DIDACTIC SESSIONS
History of ECCE Techniques
Extracapsular Techniques
Use of I and A Tip in Planned ECCE
Management of the Posterior Capsule
Problems Related to Late Capsule Clouding
Indications and Selection of Patients
Contraindications and Management
Posterior Chamber IOL's
When to Abort an IOL
Secondary Implantation
FDA Reporting Requirements
Pre-Operative Evaluation
Specular Microscopy and Ultrasound
Lens Design
Instrumentation
Calculation of Lens Power

LABORATORY SESSIONS
Practice surgery on cadaver and animal eyes
Insertion of posterior chamber lenses

Tuition: $350 Accredited for 16 hours, Category I AMA-CMA
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THIRD ANNUAL DALLAS SPRING OPHTHALMOLOGY SYMPOSIUM
MARCH 13, 14, 15, 1981 FAIRMONT HOTEL DALLAS, TEXAS

VISITING FACULTY

Leo Bores, MD	Santa Fe
Edward Cotlier, MD	New Haven
David Hiles, MD	Pittsburgh
Herbert Kaufman, MD	New Orleans
James Kingham, MD	Tampa
Donald Mousel, MD	Reno
Marshall Parks, MD	Washington, DC
Gary Rogers, MD	Columbus
Ronald Smith, MD	Los Angeles
Howard Slavin, J.D.	Los Angeles
Walter Stark, MD	Baltimore
William Tasman, MD	Philadelphia

DALLAS FACULTY

Priscilla Berry, MD
Gary Fish, MD
Dwain Fuller, MD
William Hutton, MD
John Lynn, MD
Everett Moody, MD
William Phelps, MD
William Snyder, MD
David Stager, MD
Albert Vaiser, MD

TOPICS

- REFRACTIVE SURGERY (Friday, March 13)
  Radial Keratotomy (Theory, Animal Studies, Technique, Human Results, Proposed Collaborative Study), Keratophakia, Keratomileusis, Epikeratophakia, Intraocular Lenses
- PEDIATRIC ANTERIOR SEGMENT SURGERY (Saturday, March 14)
  New Approaches to Congenital Cataracts, Subluxated Lenses, Secondary Membranes, Anterior Vitrectomy, Management of Pediatric Ocular Trauma
- UPDATE ON RETROLENTAL FIBROPLASIA (Sunday, March 15)
  Historical Perspectives, Differential Diagnosis, Medical Treatment, Surgical Treatment, Medical-Legal Implications

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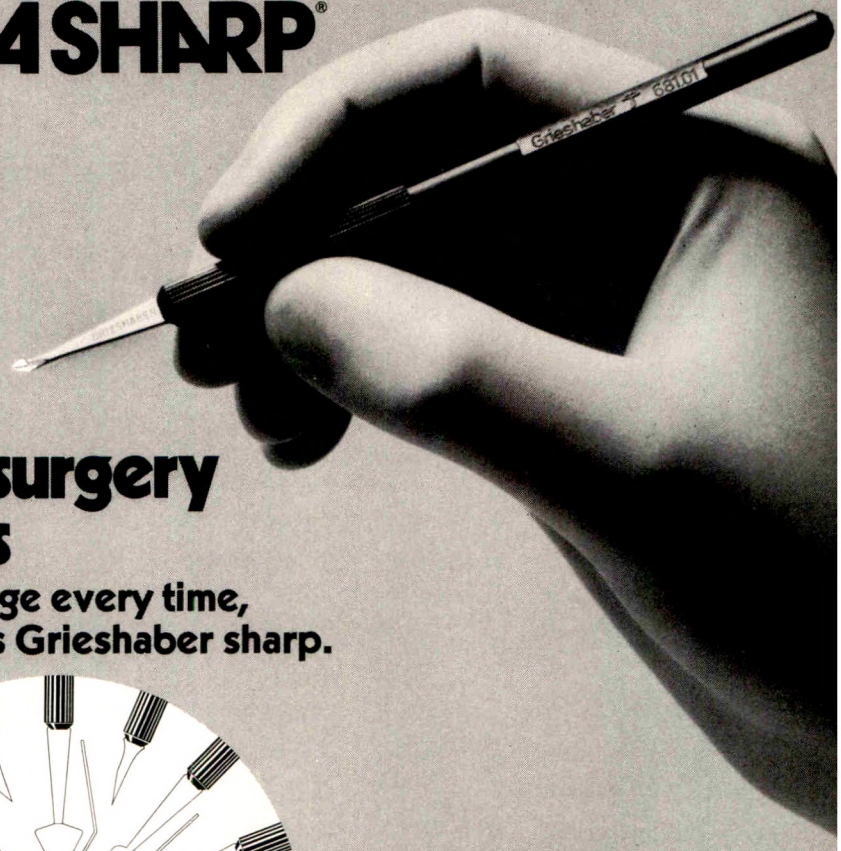
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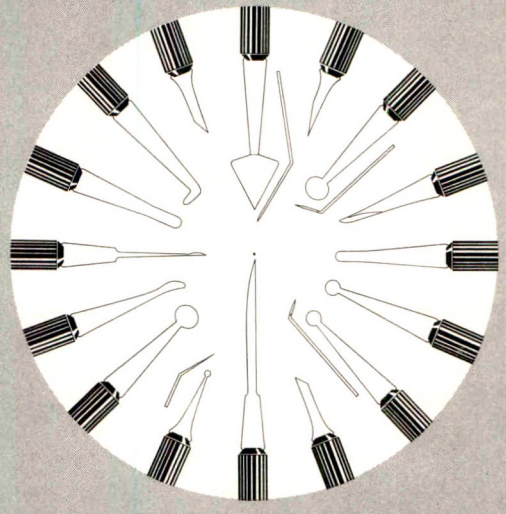
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NEW 40-MONTH STUDY REPORTS LONG-TERM EFFICACY AND TOLERABILITY

In an open, long-term maintenance study,* TIMOPTIC was given for up to 40 months to patients with chronic open-angle glaucoma or elevated IOP at sufficient risk to require lowering of the ocular pressure.

Results: 96% of the patients exhibited IOP values of ≤ 21mm Hg at each measurement during treatment with TIMOPTIC...and the drug was generally well tolerated. (See graphs and data on facing page.)

A better quality of life for many patients

Patients on TIMOPTIC generally do not experience the following therapy-limiting side effects: night blindness/blurred vision/dimming of vision.**

Patients on TIMOPTIC have significantly fewer and less severe side effects than those on pilocarpine or epinephrine, i.e., a much lower incidence of ocular discomfort—such as less burning, smarting, itching, browache, and headache.**

TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product. As with other topically applied ophthalmic drugs, this drug may be absorbed systemically.

As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients.

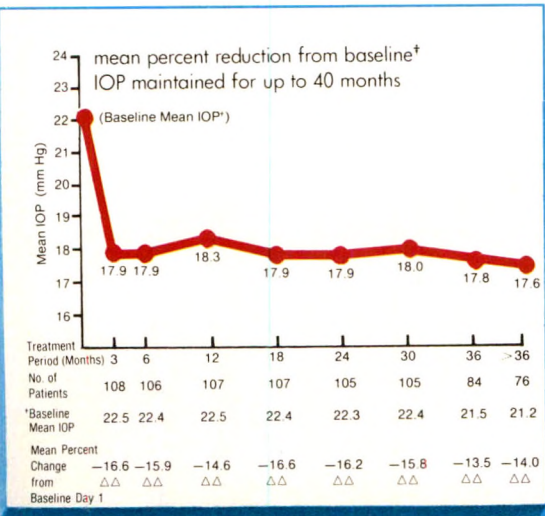
*Additional information available upon request, Merck Sharp & Dohme, West Point, Pa. 19486.

**Based on controlled multiclinic studies. Data on file, Merck Sharp & Dohme Research Laboratories. Additional information available upon request.

indicated in the treatment of chronic open-angle glaucoma

Timoptic®

(TIMOLOL MALEATE | MSD) OPHTHALMIC SOLUTION

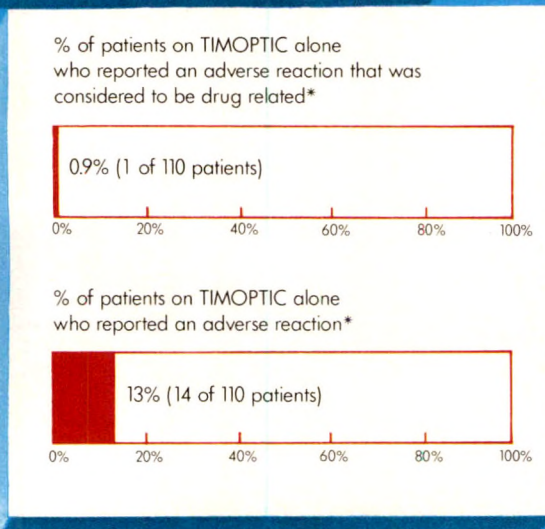


long-term efficacy of Timoptic® (Timolol Maleate/MSD)

There was virtually no loss of therapeutic effect in patients treated for up to 40 months with TIMOPTIC alone, i.e., the IOP of almost all patients was lowered to 21 mm Hg or below throughout the study.*

†Of the 110 patients on TIMOPTIC alone, 91 were on previous antiglaucoma treatment and had a treated baseline mean IOP of 21.4 mm Hg. The 19 patients with no previous antiglaucoma treatment had an untreated baseline mean IOP of 27.3 mm Hg. Thus, the mean IOP at baseline for the combined group is 22.5 mm Hg.

ΔΔSignificant percent change from baseline, $p < 0.01$.



long-term tolerability of Timoptic® (Timolol Maleate/MSD)

During the 40-month study,* 14 of 110 patients (13%) on TIMOPTIC alone reported an adverse reaction, but only one of these, an allergic reaction, was considered to be drug related.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

*Additional information available upon request, Merck Sharp & Dohme, West Point, Pa. 19486.

For a brief summary of prescribing information, please see following page.

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Timoptic®

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a beta-adrenergic blocking agent in ophthalmic solution

a significant advance for the reduction of IOP in:

- chronic open-angle glaucoma
- aphakic glaucoma
- some patients with secondary glaucoma
- patients with elevated intraocular pressure at sufficient risk to require lowering of the ocular pressure
- some patients who respond inadequately to multiple antiglaucoma drug therapy. The addition of TIMOPTIC may produce a further reduction of IOP.

How to start patients on TIMOPTIC:
Usual starting dosage: one drop 0.25% TIMOPTIC in each eye twice a day.

How to transfer from a single antiglaucoma agent to TIMOPTIC:
1. On the first day: continue with the agent already being used and add one drop 0.25% TIMOPTIC in each eye b.i.d.

2. On the second day: discontinue the previously used agent and continue with TIMOPTIC in each eye b.i.d.

If clinical response is not adequate:
Dosage may be increased (from the 0.25% solution) by changing to one drop 0.5% TIMOPTIC twice a day in each eye. Dosages above one drop of 0.5% TIMOPTIC twice a day generally have not been shown to produce further reduction of IOP. If the intraocular pressure is maintained at satisfactory levels, the dosage schedule may be changed to one drop once a day in each eye.

Because of diurnal variations in intraocular pressure, satisfactory response to the once-a-day dose is best determined by measuring the intraocular pressure at different times during the day.

In patients with a history of severe cardiac disease, pulse rates should be checked.

CONTRAINDICATIONS: TIMOPTIC is contraindicated in patients who are hypersensitive to any component of this product.
WARNINGS: As with other topically applied ophthalmic drugs, this drug may be absorbed systemically.
PRECAUTIONS: Should be used with caution in patients with known contraindications to systemic use of beta-adrenergic receptor blocking agents. These include bronchospastic disease; sinus bradycardia and greater than first-degree block, cardiogenic shock; cardiac failure; concomitant use with adrenergic-augmenting psychotropic drugs. In patients with a history of severe cardiac disease, pulse rates should be checked. Patients who are already receiving a beta-adrenergic blocking agent orally and who are given TIMOPTIC should be observed for a potential additive effect either on the intraocular pressure or on the known systemic effects of beta blockade.

Sufficient clinical data have not been obtained to show safety or efficacy in narrow-angle or angle-closure glaucoma.

As with the use of other antiglaucoma drugs, diminished responsiveness to TIMOPTIC after prolonged therapy has been reported in some patients. However, in one long-term study in which 96 patients have been followed for at least 3 years, no significant difference in mean intraocular pressure has been observed after initial stabilization.

Pregnancy: TIMOPTIC has not been studied in human pregnancy. The use of TIMOPTIC in pregnant women requires that the anticipated benefit be weighed against possible hazards.

Use in Children: Since clinical studies in children have not been done, TIMOPTIC is not recommended for use in children at this time.

Animal Studies: No adverse ocular effects were observed in rabbits and dogs administered TIMOPTIC topically in studies lasting one and two years respectively.

Lifetime studies with timolol have been completed in rats at oral doses of 25, 100, and 300 mg/kg/day and in mice at oral doses of 5, 50, and 500 mg/kg/day. In male and female rats and male mice at all dose levels and in female mice at dose levels of 5 and 50 mg/kg/day, timolol demonstrated no carcinogenic effect. There was a slight increase in the incidence of mammary adenocarcinomas in female mice that received 500 mg/kg/day (about 500 times the maximum recommended human oral dose on a mg/kg basis). Timolol caused dose-related elevations of serum prolactin in female mice at doses of 100 mg/kg or more, but only very slight transient elevations were found in male mice at doses of 500 mg/kg. Since numerous studies have demonstrated that drugs which cause elevations of serum prolactin are associated with mammary tumors in rodents, the mammary tumors in the female mice in the highest dosage group of this study were considered to have resulted from an increased serum prolactin. In humans, no such association between serum prolactin and mammary carcinoma has been established. Furthermore, in adult human female subjects who received oral dosages of up to 60 mg of timolol, the maximum recommended human oral dosage, there were no clinically meaningful changes in serum prolactin.

ADVERSE REACTIONS: TIMOPTIC Ophthalmic Solution is usually well tolerated. The following adverse reactions have been reported either in clinical trials of up to 3 years duration prior to release in 1978 or since the drug has been marketed.

Signs and symptoms of ocular irritation, including conjunctivitis, blepharitis, and keratitis, have been reported occasionally.

Hypersensitivity reactions, including localized and generalized rash, and urticaria have been reported rarely.

Visual disturbances including refractive changes (due to withdrawal of miotic therapy in some cases) have been infrequently associated with therapy with TIMOPTIC. Rarely, aphakic cystoid macular edema has been reported, but a causal relationship to therapy with TIMOPTIC has not been established.

Aggravation or precipitation of certain cardiovascular and pulmonary disorders has been reported, presumably related to effects of systemic beta blockade (see PRECAUTIONS). These include bradyarrhythmia, hypotension, syncope, and bronchospasm (predominantly in patients with pre-existing bronchospastic disease). In clinical trials, slight reduction of the resting heart rate in some patients (mean reduction 2.9 beats/minute, standard deviation 10.2) has been observed.

The following adverse effects have been reported rarely, and a causal relationship to therapy with TIMOPTIC has not been established: headache, anorexia, dyspepsia, nausea, dizziness, CNS effects (fatigue, confusion, depression, somnolence, and anxiety), palpitation, and hypertension.

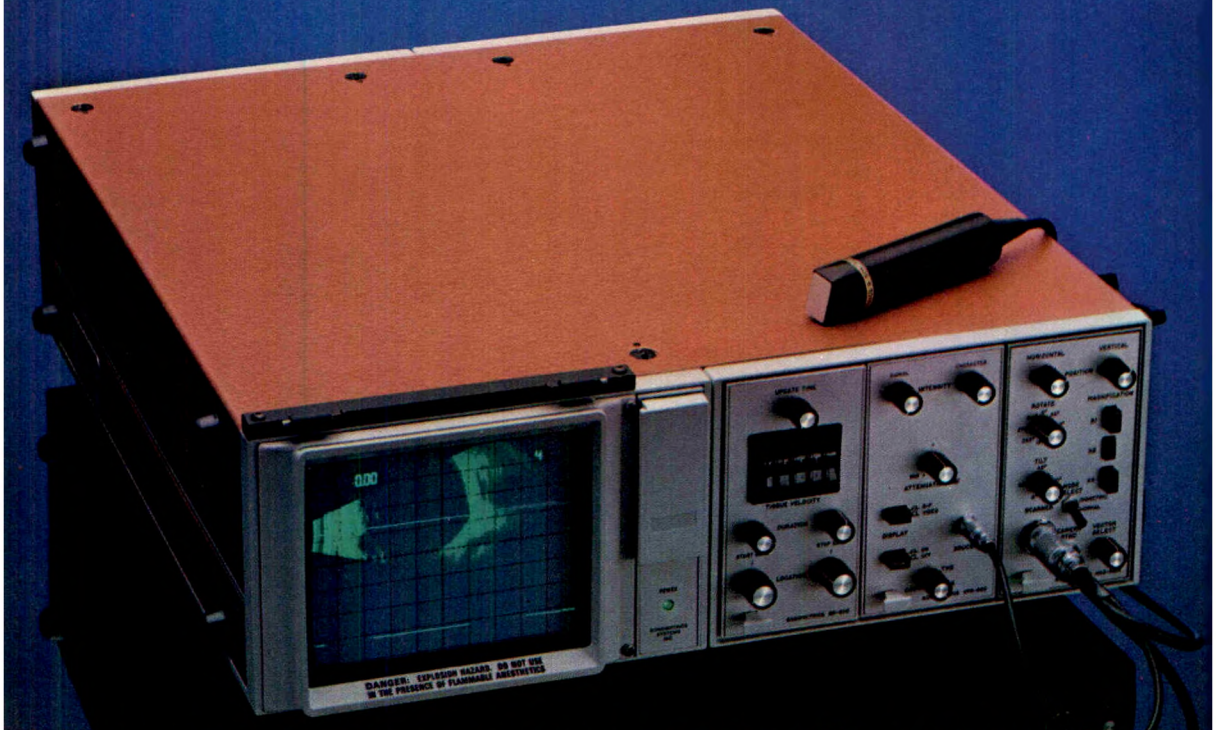
HOW SUPPLIED: TIMOPTIC Ophthalmic Solution, 0.25% and TIMOPTIC Ophthalmic Solution, 0.5%. Both are available in 5-ml and 10-ml plastic OCUMETER® Ophthalmic Dispensers with a controlled drop tip.

980(DC7046011)

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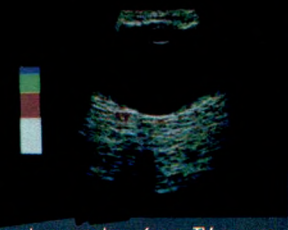


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AUTOMATED PERIMETRY—*Moderator:* William O. Sass, M.D.
Speakers: Professor H. Bebie, Steven Feldon, M.D., Charles Kurtzman, Ph.D., G. Peter Halberg, M.D., David Miller, Ph.D. George Spaeth, M.D., G. Zuehlke, Ph.D.

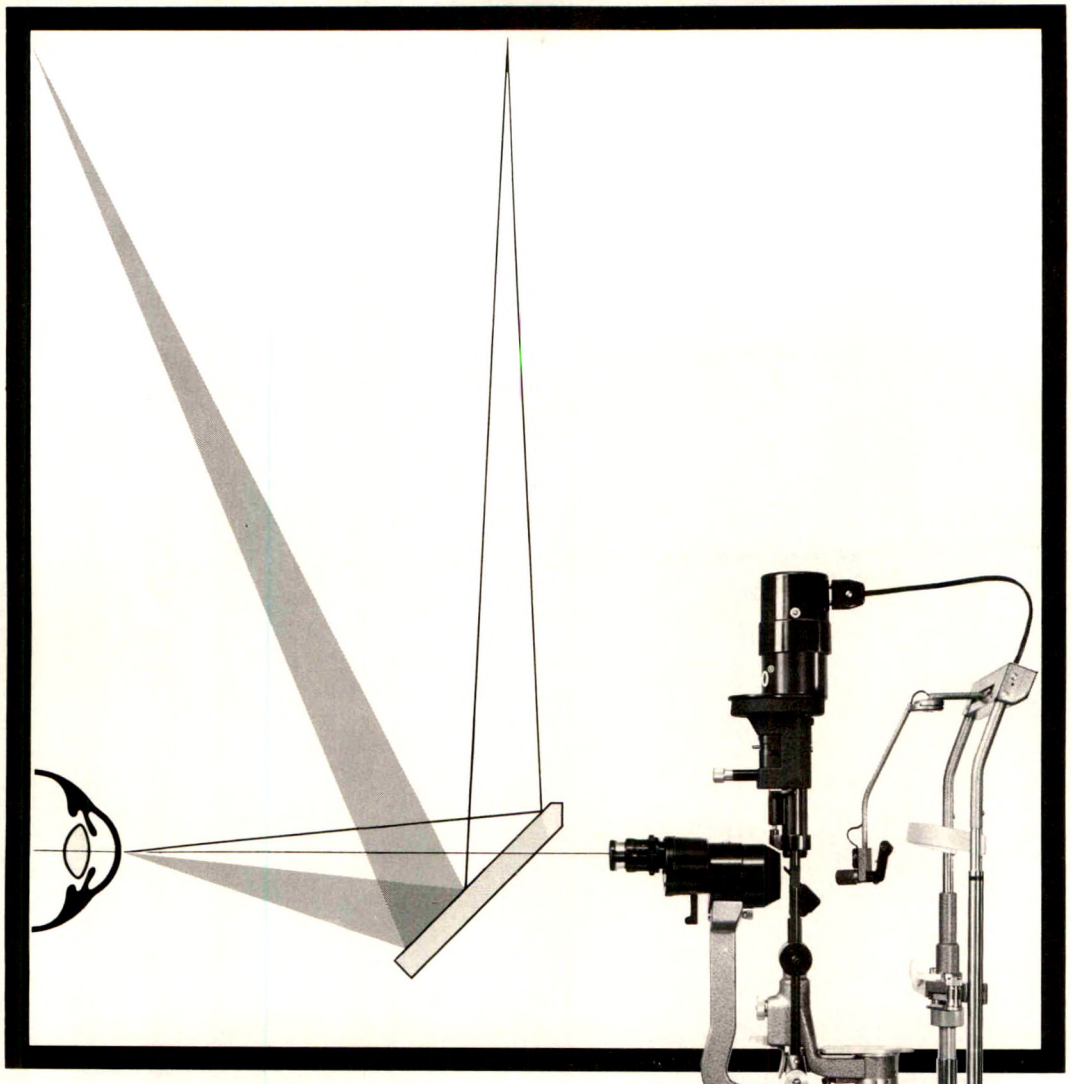
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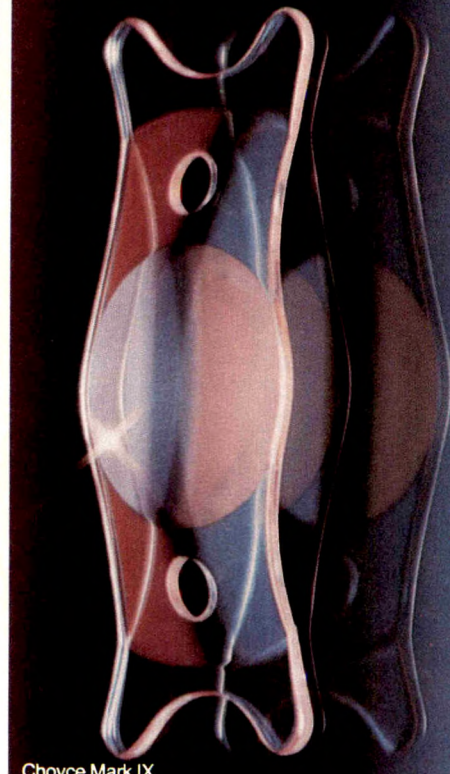
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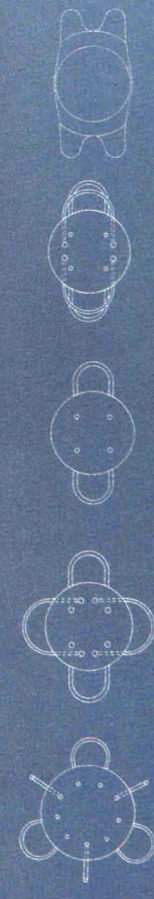
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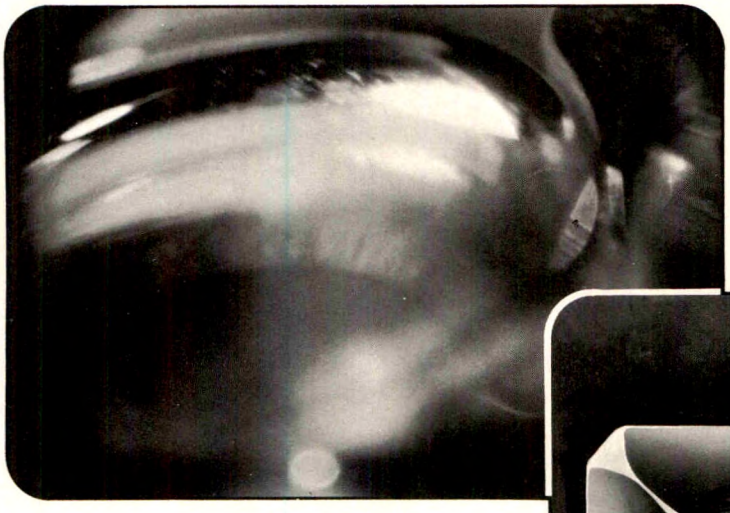
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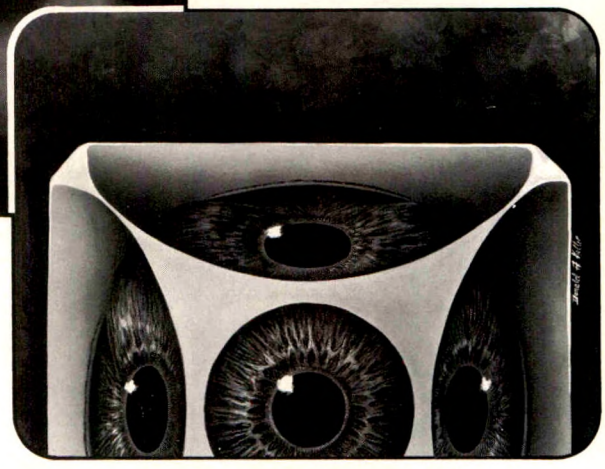
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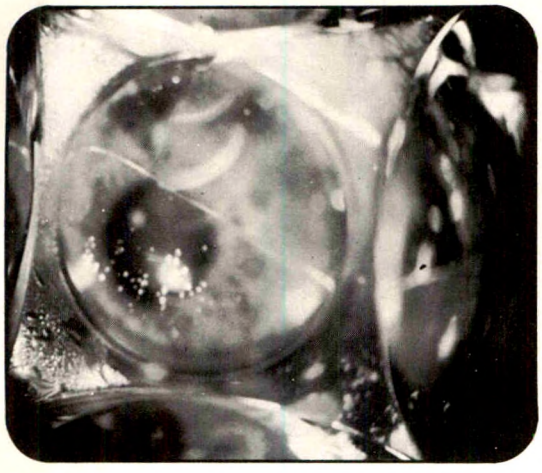
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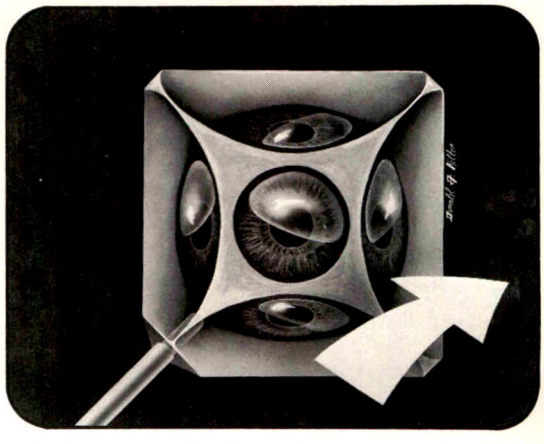


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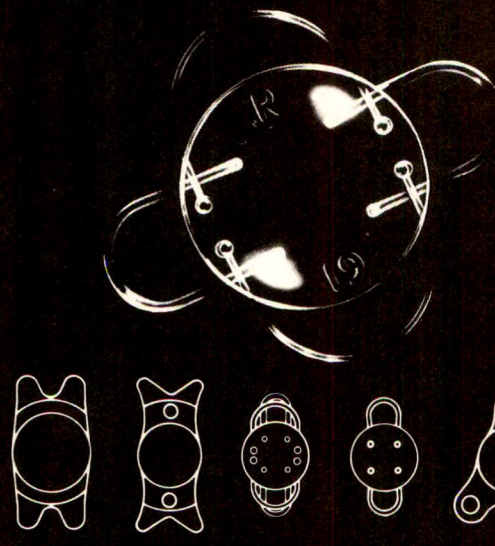
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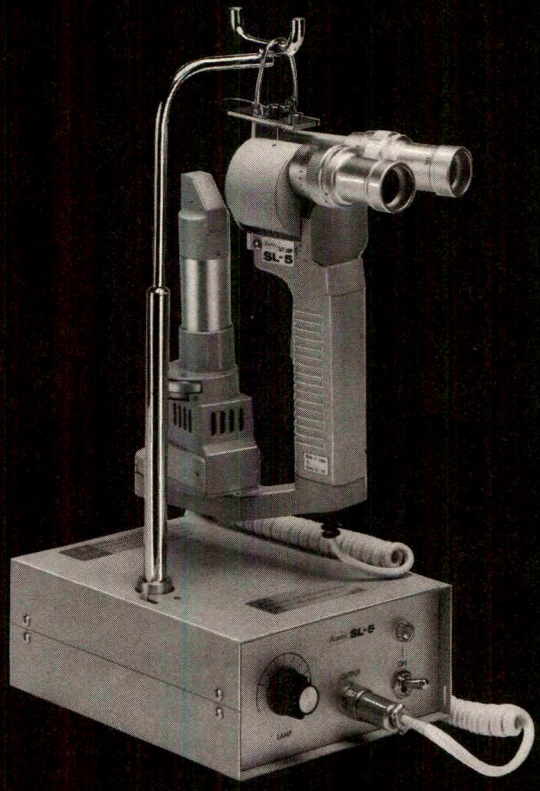
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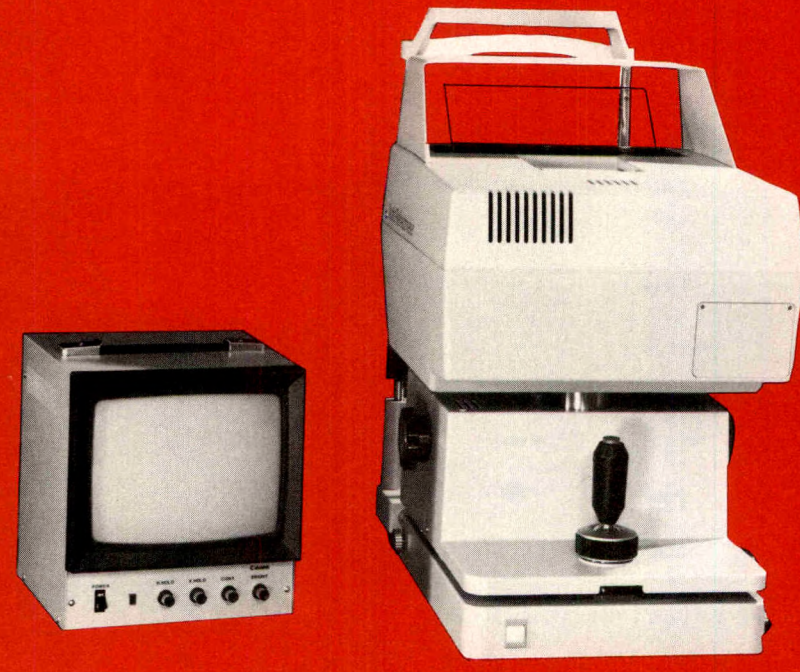
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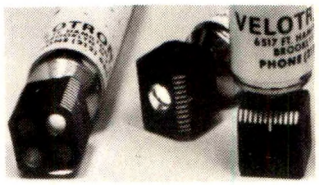
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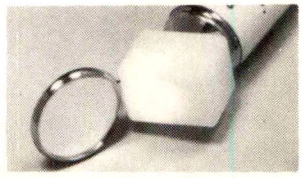
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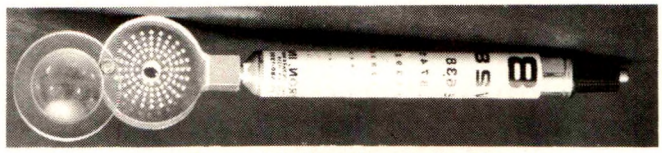
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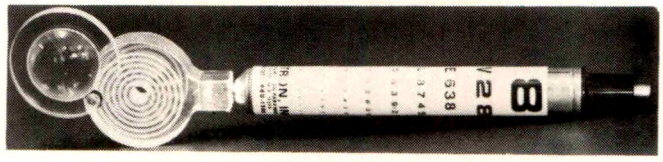
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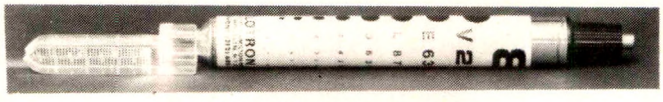
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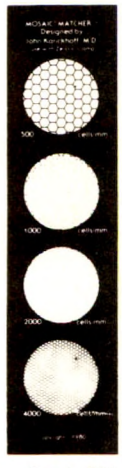
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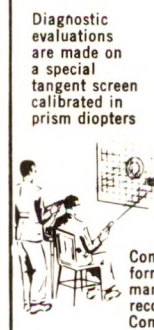
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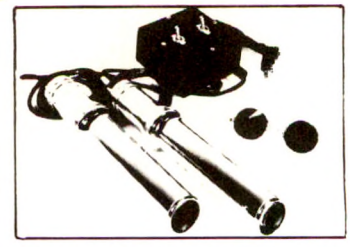
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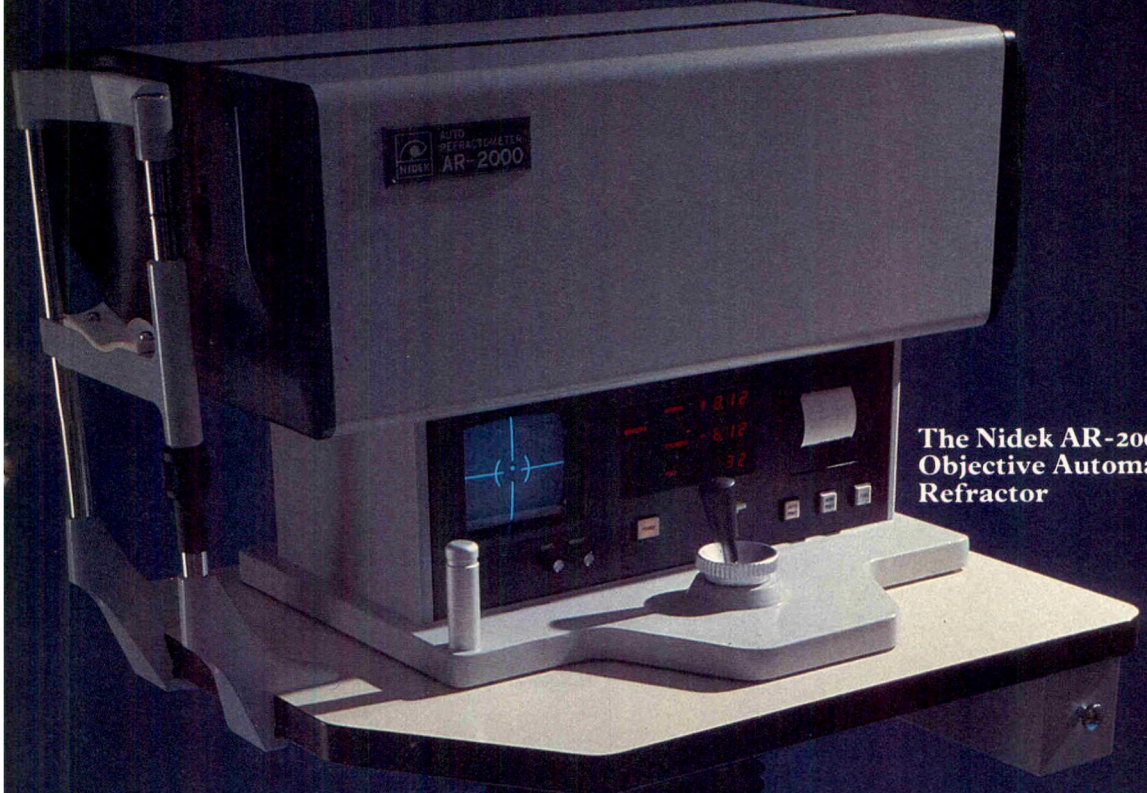
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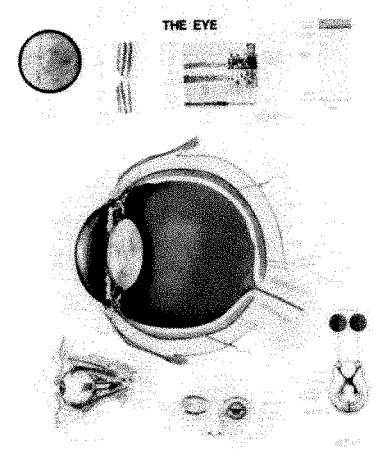
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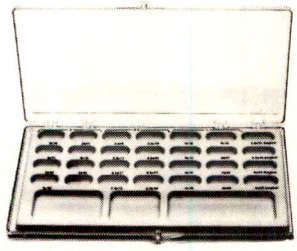
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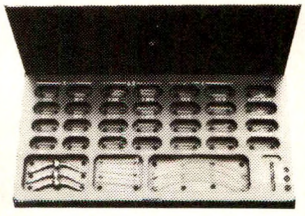
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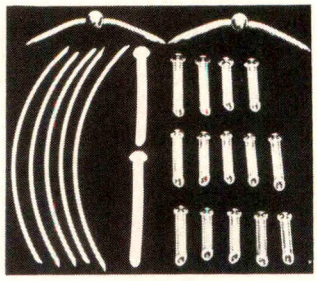
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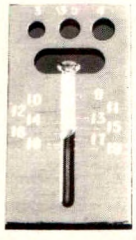
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* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
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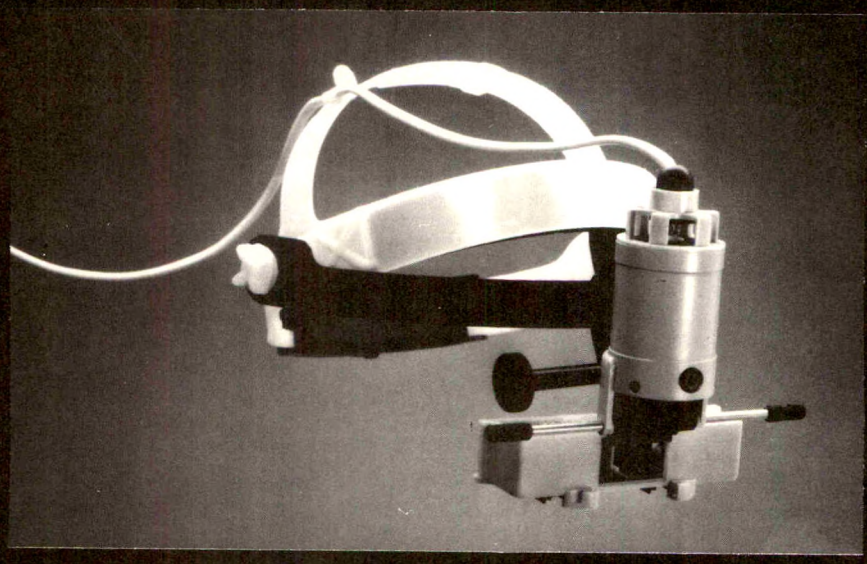
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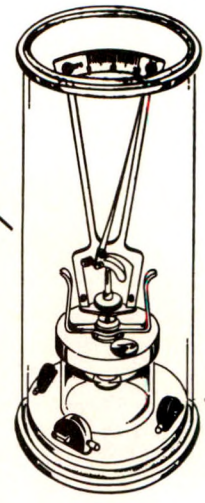
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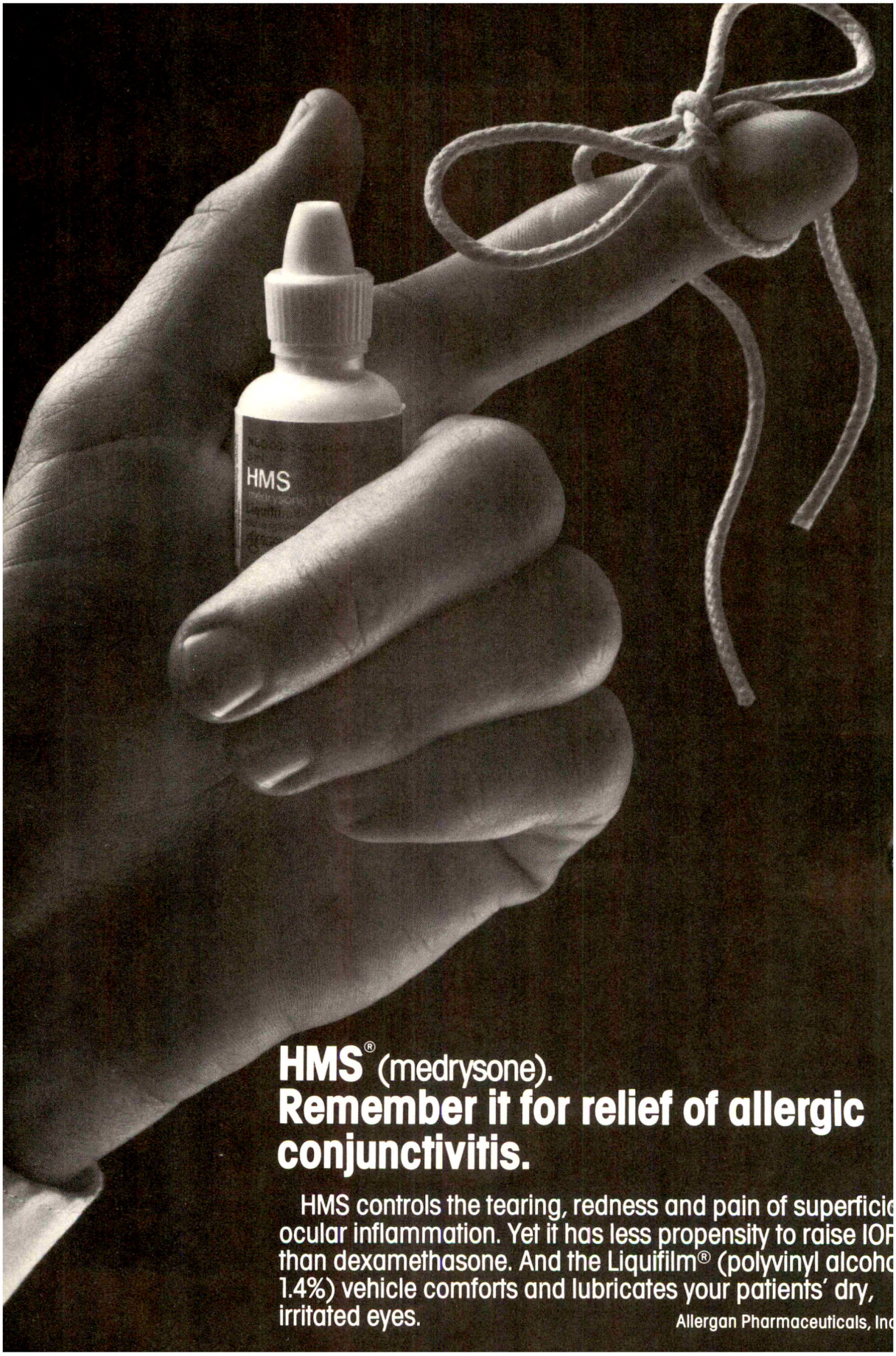


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CONTAINS: medrysone*, 1.0%, with: Liquifilm (polyvinyl alcohol) 1.4%; benzalkonium chloride; edetate disodium; sodium chloride; potassium chloride; sodium phosphate monobasic, monohydrate; sodium phosphate dibasic, anhydrous; hydroxypropyl methylcellulose; purified water, and sodium hydroxide or hydrochloric acid if needed to adjust pH. *Licensed under U.S. Patent 2,968,655. Medrysone (previously known as hydroxymesterone) belongs to the family of steroids, related structurally more to progesterone than to other available corticosteroids. It is a white crystalline powder, sparingly soluble in water, with a melting point of approximately 162°C, and empirical formula of $C_{22}H_{32}O_3$ and a molecular weight of 344.50. The specific rotation of a 1% solution of medrysone in chloroform is between +184° and +192°. It has a characteristic ultraviolet absorption maximum of 242±1 millimicrons in 95% ethanol solution. **Chemical Name:** 11β-Hydroxy-6α-methylpregn-4-ene-3, 20 dione. **ACTIONS:** HMS (medrysone) is a synthetic steroid with topical anti-inflammatory and anti-allergic activity. Steroids inhibit the inflammatory response to inciting agents of mechanical, chemical, or immunological nature of edema, fibrin deposition, capillary dilatation and leucocyte migration, capillary proliferation, deposition of collagen and scar formation. No generally accepted explanation of this steroid property has been advanced. HMS (medrysone) has less anti-inflammatory potency than 0.1% dexamethasone. Data from 2 uncontrolled studies[1,2] indicate that in patients with increased intraocular pressure and in those susceptible to a rise in intraocular pressure, there is less effect on pressure with HMS than with dexamethasone or betamethasone.
INDICATIONS: HMS (medrysone) is effective in the treatment of allergic conjunctivitis, vernal conjunctivitis, episcleritis, and epinephrine sensitivity.
CONTRAINDICATIONS: HMS (medrysone) is contraindicated in the following conditions: Acute superficial herpes simplex. Viral diseases of the conjunctiva and cornea. Ocular tuberculosis. Fungal diseases of the eye. Hypersensitivity to any of the components of the drug. **WARNINGS:** 1. Acute purulent untreated infections of the eye may be masked, enhanced or activated by the presence of steroid medication. 2. Corneal or scleral perforation occasionally has been reported with prolonged use of topical steroids. In high dosage they have been associated with corneal thinning. 3. Prolonged use of topical steroids may increase intraocular pressure, with possible resultant glaucoma, damage to the optic nerve, and defects in visual acuity and fields of vision. However, data from 2 uncontrolled studies[1,2] indicate that in patients with increased intraocular pressure and in those susceptible to a rise in intraocular pressure upon application of topical steroids, there is less effect on pressure with HMS than with dexamethasone or betamethasone. 4. Prolonged use of topical corticosteroids may rarely be associated with development of posterior subcapsular cataracts. 5. Systemic absorption and systemic side effects may result with the use of topical steroids. 6. HMS (medrysone) is not recommended for use in iritis and uveitis as its therapeutic effectiveness has not been demonstrated in these conditions. 7. Steroid medication in the presence of stromal herpes simplex requires great caution; frequent slit lamp microscopy is suggested. 8. Prolonged use may aid in the establishment of secondary ocular infections from fungi and viruses liberated from ocular tissue. **Use In Pregnancy:** The use of topical steroids in pregnancy should be limited to conditions serious enough to warrant such treatment, so that possible risk to the fetus may be justified by the expected benefit to the mother. **PRECAUTIONS:** With prolonged use of HMS (medrysone) the intraocular pressure and lens should be examined periodically. In persistent corneal ulceration where a steroid has been used, or is in use, fungal infection should be suspected. **ADVERSE REACTIONS:** Occasional transient stinging and burning may occur on instillation. **DOSAGE AND ADMINISTRATION:** One drop instilled in the conjunctival sac up to every four hours. Shake well before using. DO NOT FREEZE. **HOW SUPPLIED:** As a sterile suspension in 5 ml. and 10 ml. plastic dropper bottles. On prescription only. NOTE: 5 ml bottle filled to 1/2 capacity and 10 ml bottle filled to 2/3 capacity for proper drop control.

REFERENCES: 1. Becker, B. and Kolker, A.E.: "Intraocular Pressure Response to Topical Corticosteroids" in Ocular Therapy, Complications and Management, Irving Leopold, Ed. St. Louis, C.V. Mosby, 1967, pp. 79-83. 2. Spaeth, G.: Hydroxymethylprogesterone, Arch. Oph. 75: 783-787, 1966.

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CLINICAL AND RESEARCH ASPECTS OF SUBTOTAL OPEN-SKY VITRECTOMY

XXXVII EDWARD JACKSON MEMORIAL LECTURE

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Boston, Massachusetts

During the past ten years, vitrectomy has become a surgical procedure of great value. The main indications are removal of dense vitreous opacities and release of vitreoretinal traction. The overwhelming majority of vitrectomies performed today are closed vitrectomies, which have the advantage that the globe incision is small and, as a result, the intraocular pressure can be maintained at a near-normal level. The surgical field is observed through the dilated pupil. Many difficult closed vitrectomies are made easier by a two-instrument technique. However, in spite of improved techniques, some cases still cannot be operated on successfully by closed vitrectomy.

During the past several years, I have attempted to develop a technique of subtotal open-sky vitrectomy—not as a substitute for closed vitrectomy but rather as an extension of it—in the hope of salvaging some eyes that cannot now be helped by closed vitrectomy. I have been assisted in this task by a number of able co-workers, and by Tatsuo Hirose, M.D., in particular.

From the Eye Research Institute of Retina Foundation, Boston, Massachusetts. Presented before the 85th annual meeting of the American Academy of Ophthalmology, Chicago, Nov. 3, 1980.

Reprint requests to C. L. Schepens, M.D., Eye Research Institute, 20 Staniford St., Boston, MA 02114.

The advantages of open-sky vitreous surgery include easier access for surgical instruments to the site of operation and the possibility of switching instruments readily and of changing their position. The surgical field is observed directly through the microscope. Anterior fundus structures are more readily accessible and more visible than with the closed method. A disadvantage is that the intraocular pressure is equalized with atmospheric pressure and, as a consequence, the intraocular tissues tend to become edematous after a period of time.

The subject of this article is limited to subtotal open-sky vitrectomy; I will not discuss anterior open-sky vitrectomy. The latter procedure consists of surgical work on the anterior segment plus a vitrectomy that does not extend much further than the globe's equator.

In all the cases discussed here, the surgeon removed as much vitreous as was safe, including the peeling of preretinal membranes in an overwhelming majority of cases. However, a vitrectomy must always remain subtotal because it is extremely dangerous to excise the corona of rigid gel that forms the vitreous base. Such attempts often cause an extensive detachment of the nonpigmented epithelium of the pars plana ciliaris, followed by a detachment of the ora serrata from the choroid. In the course of excising the gel

of the vitreous base, the detached nonpigmented epithelium is frequently excised or torn, causing a giant retinal break. In most eyes subjected to vitrectomy, the adhesion of the vitreous base to the retina extends more posteriorly than the ora serrata, sometimes as far back as the equator of the globe. Therefore, removal of vitreous material near the retina, anterior to the equator, should be performed with great care, and the most extensive vitrectomy must remain subtotal if catastrophies are to be avoided.

Subtotal open-sky vitrectomy is a long and difficult operation, to be undertaken only when no other procedure is likely to succeed. Because the joint experience of Hirose and myself covers well over 300 cases, operated on during a span of nine years, our data can be presented with a degree of confidence. The procedure has some strong detractors among vitreoretinal surgeons. However, their experience with open-sky vitrectomy is generally nil and therefore their mostly unpublished criticisms can be discounted.

Clinical aspects

My first attempt at subtotal open-sky vitrectomy was in 1960, on a case of giant retinal tear with a fixed, rolled retinal flap that adhered to the retracted vitreous gel. After the operation, the retina could not be reattached because of massive preretinal retraction. One year later, the cornea, which had remained clear, gradually became covered by a retrocorneal membrane. This initial failure discouraged me from further trials until Kasner and associates,[1] in 1968, reported some successful cases and others in which the cornea opacified. I then thought that if the technique of open-sky vitrectomy could be improved, it would offer definite possibilities for some cases that had been incurable.[2]

I have divided the discussion of the present status of subtotal open-sky vitrectomy into four parts: instrumentation and material, surgical procedure, indications and results, and complications.

Instrumentation and material

Instrumentation—Clinical experience showed that special instruments were needed to perform the operation safely and efficiently.[3] When most of the ocular contents are surgically removed, the walls of the globe tend to collapse. This must be prevented by using a scleral supporter[4] (Fig. 1), which is more effective than a simple Flieringa ring.

The instruments used inside the ocular cavity must fit under a surgical microscope with a focal length of 125 mm, which is the most useful focal length for this type of work. Therefore, these instruments should have handles no more than about 100 mm long and should be angulated to keep the surgeon's hands out of the field of observation. Since the instruments are used in a surgical field

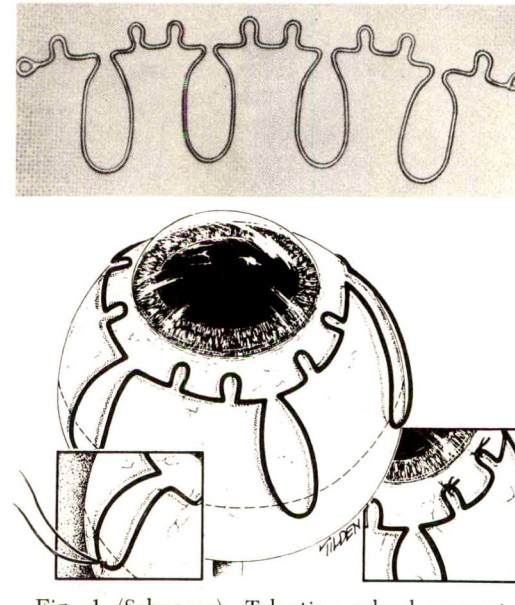


Fig. 1 (Schepens). Tolentino scleral supporter prevents collapse of globe during open-sky vitrectomy. Top, Total length of supporter. Bottom, Supporter molded to fit globe. Insets, One 6-0 suture is placed on each loop.

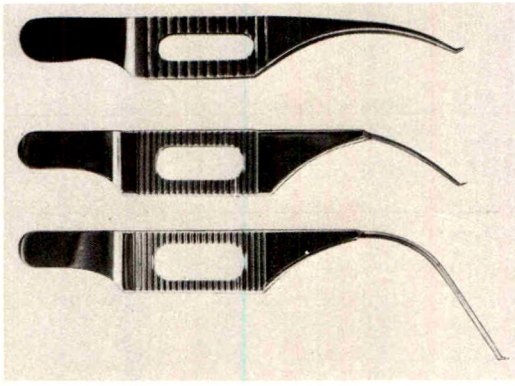


Fig. 2 (Schepens). Colibri forceps for vitreous surgery. At top, regular Colibri forceps; at middle, Colibri forceps with greater curve; at bottom, Colibri forceps with greater curve and longer shaft for work deep inside vitreous cavity.

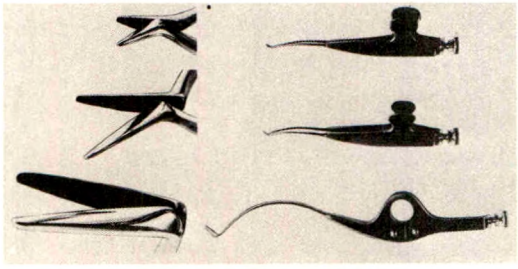


Fig. 3 (Schepens). Vitreous scissors. At top and middle, miniaturized de Wecker scissors have short (1.5 mm) or long (2.5 mm) blades; at bottom, long-handled, modified de Wecker scissors have blades at right angle to shaft.

located 17 to 25 mm from the patient's pupillary opening, they must have fairly long shafts to allow access to the posterior pole.

The forceps used are of a modified Colibri design (Fig. 2); scissors are modified and miniaturized de Wecker instruments, curved slightly backward (Fig. 3). Preretinal dissection is done with two types of disposable aluminum spatulas, one blunt and one relatively sharp (Fig. 4). Liquid is removed from the vitreous cavity with a suction spatula, which prevents the retina from being damaged by suction (Fig. 5). When bulging retinal folds need to be gently moved out of the way, a tiny retractor is used (Fig. 6). To explore the area of the optic disk and macula, a gold ring is placed over the posterior pole (gold was selected because of its high specific gravity). A shielded electrode, adapted for work under water,[5] is used to prevent or stop intraocular bleeding.

Material—A new generation of purified high-molecular-weight hyaluronic acid or sodium hyaluronate solutions has become an important adjunct in intraocular surgery. These new solutions of noninflammatory hyaluronic acid were first introduced in 1974,[6,7] when it was shown, by quantitative analysis, that single primary injections in the owl monkey eye were as well tolerated as physiologic saline or buffer solutions. Further experiments showed that a solution of hyaluronic acid injected into the vitreous diffuses slowly, via the anterior cortical gel, into the anterior chamber and leaves the eye via Schlemm's canal.[8] Hyaluronic acid injected into the vitreous or aqueous is not degraded by inflammation or experimental hemorrhage.[8,9] On the contrary, hyaluronic acid may actually prevent the influx of inflammatory proteins

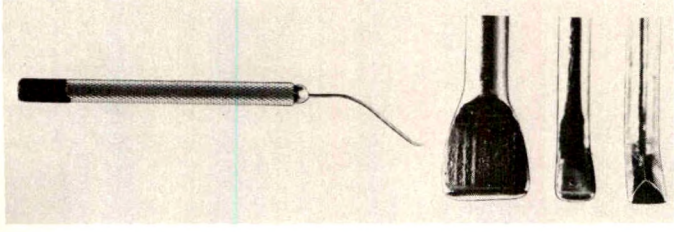


Fig. 4 (Schepens). Retinal dissection microspatulas. At left, disposable microspatula screwed in handle; at right, enlarged views of two blunt microspatulas, wide and narrow, and relatively sharp microspatula.

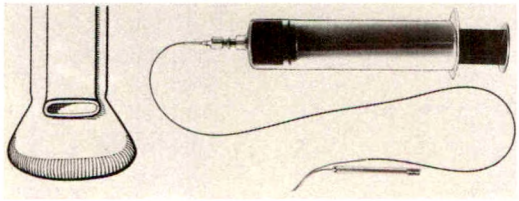
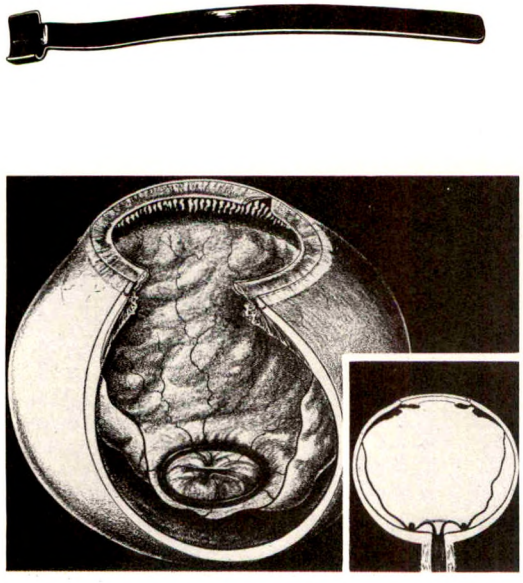


Fig. 5 (Schepens). At left, blunt spatula shields tube, preventing suction of retina; at right, spatula with suction tube and syringe.

into the vitreous cavity.[8,9] Of further importance is the presence of a novel factor that prevents the degradation of hyaluronic acid by the ascorbic acid present in the vitreous and aqueous. This has been termed the hyaluronic acid-depolymerization prevention factor.[10] Thus, hyaluronic acid that is injected intraocularly is retained for various lengths of time in the vitreous[7-9] and aqueous humor,[11] depending on the molecular weight and concentration of the polymer, the integrity of the cortical gel in the anterior vitreous, and the outflow capacity of Schlemm's canal. It was shown experimentally[12] that laser therapy did not cause the degradation of vitreous hyaluronic acid.

My associates and I have used hyaluronic acid (HYVISC) extensively in subtotal open-sky vitrectomy.[13] The marked viscosity of this substance makes it very cohesive so that it tends to remain as a globule where injected. Since it is strongly hydrophilic, it mixes freely with water. The material does not adhere to surgical instruments. In the owl monkey eye, the intraocular inflammation it may cause is no greater than that produced by intraocular injection of a physiologic solution (E. Hultsch, unpublished data, 1980). This purified hyaluronic acid solution is similarly well tolerated in human eyes.[13] In agreement with the experimental data, the material was not degraded by the human intraocular fluids and provides a long-term tamponading effect (E. Hultsch and C. L. Schepens, unpublished data).

During an open-sky vitrectomy, hyaluronic acid is used mainly on the anterior



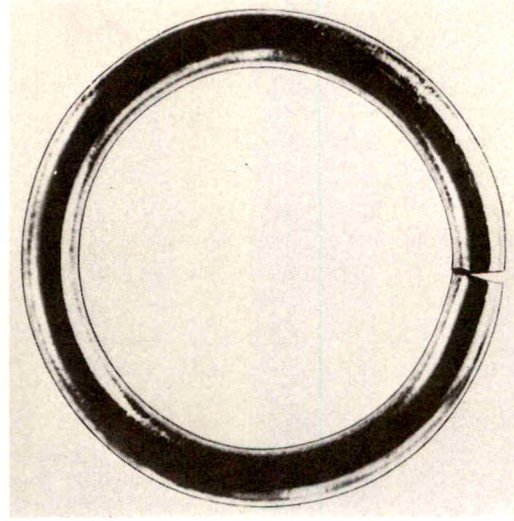


Fig. 6 (Schepens). Top left, Pliable retinal retractor. Top right, Gold ring used to expose posterior pole. Bottom left, Ring in place exposes optic disk area. Inset, Membrane that bridges optic disk is visible inside ring.

surface of the iris and in the vitreous cavity. After radial iridotomies to enlarge the pupil and cryoextraction of the lens, hyaluronic acid is slowly injected through an olive-tip needle on the anterior surface of the iris and in the filtration angle. The purpose is to prevent filtration angle closure by an adhesion between the iris and the scleral edge of the corneal incision. Such an adhesion is generally rendered sturdy by the production of a fibrinous membrane that attaches the anterior surface of the iris to the corneal wound.

Hyaluronic acid is injected into the vitreous cavity to offset problems caused by the accumulation of transudative fluid in the subchoroidal and subretinal spaces (Fig. 7). The fluid balloons the retina forward, compromising surgical exposure. By injecting hyaluronic acid in the vicinity of the optic disk through an olive-tip needle, the large balloons of detached retina are pushed aside somewhat, exposing the posterior pole. If further preretinal surgery is needed around the posterior pole, it is carried out beneath the surface of this clear viscous solution.

Since hyaluronic acid has a high surface tension, it tends to form a convex meniscus at the air-fluid interface. This may cause severe distortion that interferes with precise dissection of preretinal membranes. In this circumstance, the eye is filled with hyaluronic acid to the edge of the corneal incision (Fig. 8). The hyaluronic acid then forms a convex meniscus that acts optically like an artificial cornea and through which visualization with the surgical microscope is impossible. A small, flat contact lens, mounted on a tripod of fine metallic wire, is then placed over the convex meniscus of hyaluronic acid. The tripod rests on the sclera in such a way that there is a space between the edge of the contact lens and the scleral edge of the corneal incision. A good view of the patient's vitreous cavity is now available through the flat contact lens. It is through the space between contact lens and scleral edge of the corneal incision that instruments are introduced into the vitreous cavity for dissection around the posterior pole.

At the end of the procedure, the intraocular cavity is filled with hyaluronic acid. This helps to push the iris backward, thereby deepening the anterior chamber and increasing the intraocular pressure to a normal level. The clinical impression is that postoperatively hyaluronic acid helps to prevent the formation of adhesions between iris and cornea and between retina and other tissues. So far, glaucoma has not been observed in humans as a result of filling the intraocular cavity with hyaluronic acid.

Surgical procedure

The surgical procedure is divided into four stages—preparatory steps, operating

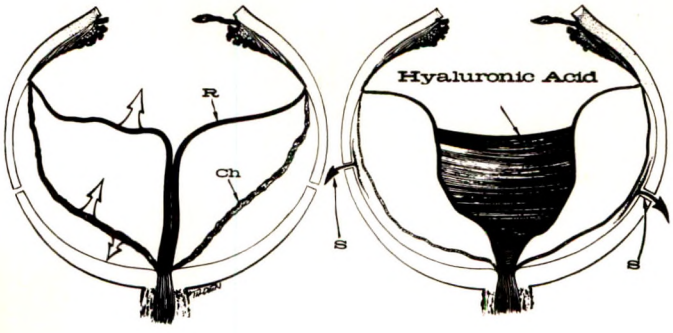


Fig. 7 (Schepens). At left, toward the end of open-sky vitrectomy, the choroid (Ch) exudes fluid, often causing marked choroidal and retinal (R) elevation. At right, partially filling the vitreous cavity with hyaluronic acid tends to redistribute subchoroidal and subretinal fluids and to facilitate release of subchoroidal fluid through sclerotomies (S) made at the beginning of the operation. This exposes posterior pole temporarily.

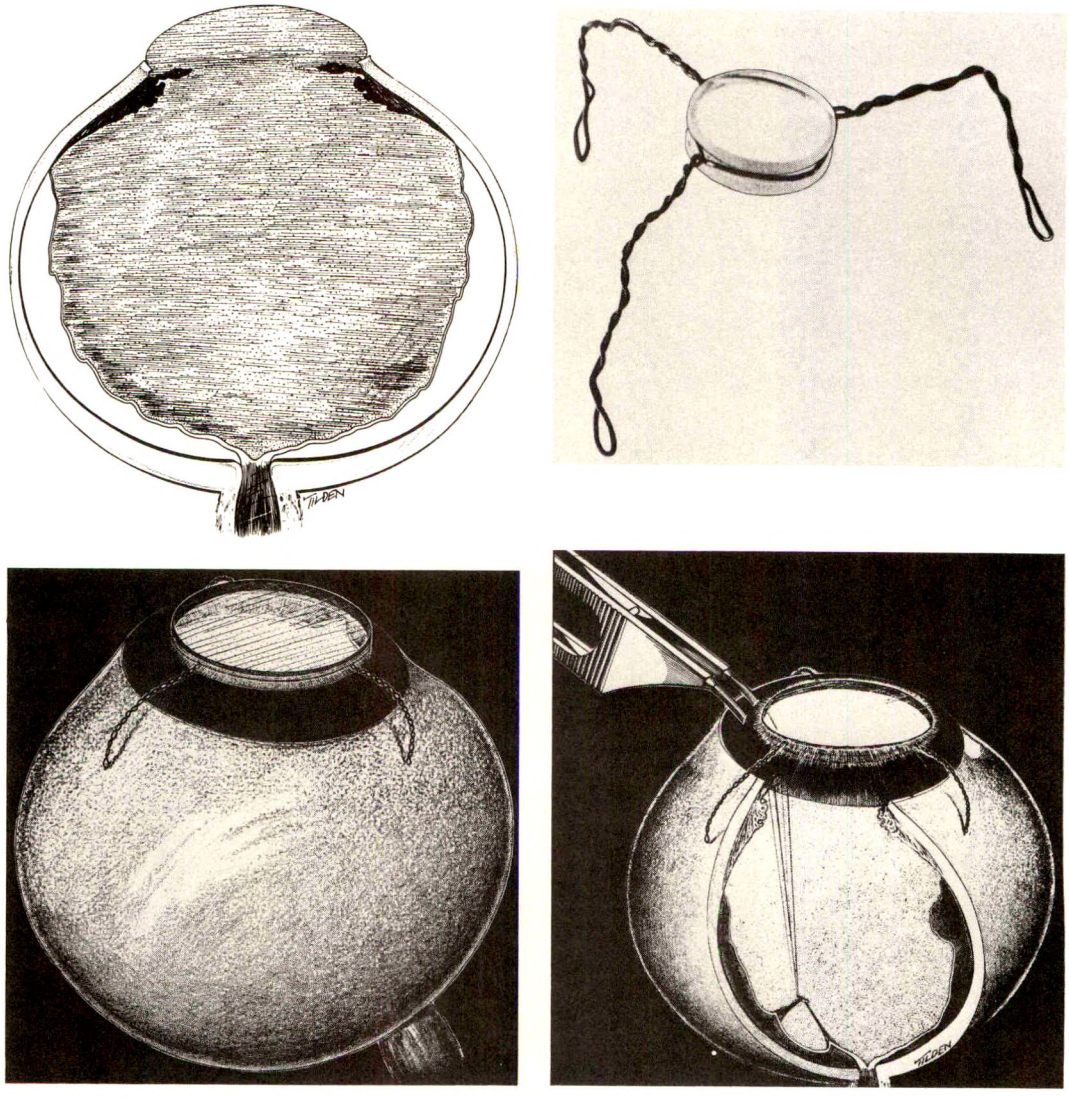


Fig. 8 (Schepens). Top left, To improve visualization of posterior pole, entire vitreous cavity may be filled with hyaluronic acid. Top right, Flat contact lens, 6 mm in diameter, with tripod of fine wire. Bottom left, Contact lens rests on hyaluronic acid and tripod rests on sclera. Bottom right, Dissection by inserting instruments between contact lens and sclera; observing is done through contact lens.

inside the vitreous cavity, wound closure, and retinal reattachment.

Preparatory steps—If a large amount of subretinal fluid is present, it is released and the intraocular pressure is reestablished with a vitreous injection of physiologic solution. One or two sclerotomies are made, preferably 180 degrees apart, to avoid the formation of large subchoroidal effusions during the operation. The sclerotomies are temporarily closed with a slipknot to permit safe trephining of the cornea. A trephine is used that leaves a rim of 1 to 1.5 mm of clear cornea all around the corneoscleral limbus. The corneal button is kept in

tissue culture fluid during the operation.[14] Two radial iridotomies are made at the 12 and 6 o'clock positions and the lens is removed by cryoextraction, generally without prior irrigation with alpha-chymotrypsin. Then the slipknots on the scleral incisions are loosened.

Operating inside the vitreous cavity—The central portion of the vitreous is removed with a nibbler without infusion or fiberoptic illumination. The next and most difficult step is preretinal dissection. Before removal, preretinal membranes must be carefully loosened from the underlying retinal surface, using smooth vitreous forceps and a retinal dissection spatula (Fig. 9). Once separated from the retina, the membranes are excised with vitreous scissors. The process of dissection may involve more than the whole posterior hemisphere of the globe from the optic disk to an area anterior to the equator. In general, dissection should not be attempted in the extreme fundus periphery because atrophic retina in this area often adheres strongly to preretinal membranes. In many cases, such adhesions do not prevent subsequent retinal reattachment; exceptions are eyes in which the entire vitreous base has shrunk or the retina has become adherent to an anterior structure such as the pars plana ciliaris, the ciliary processes, or the iris.

When membranes are extensively adherent to the retina, frequently the main difficulty is locating a free edge from which to start dissecting. Such an edge can usually be found in the vicinity of the optic disk. Pigmented membranes (possibly derived from the pigment epithelium) are often easier to peel off the retina than are whitish membranes, some of which may have a different origin. Dissection of vitreous membranes of recent formation is generally easy. Long-standing membranes tend to be more adherent, and the retina to which they are attached is often atrophic, making membrane dissection more risky. When new-formed tissue is adherent to the area surrounding the optic disk, its dissection often causes bleeding, probably from new vessels that are not clearly visible before dissection.

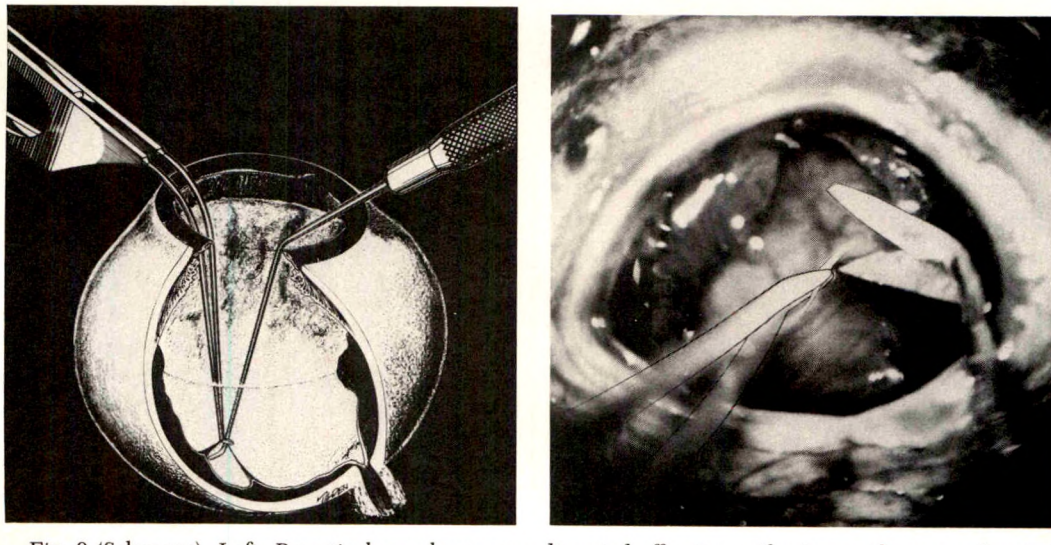


Fig. 9 (Schepens). Left, Preretinal membranes are dissected off retina with vitreous forceps and retinal microspatula. Right, Freed membrane is excised with forceps and scissors.

Therefore, stubborn attempts should not be made to remove preretinal membranes close to the optic disk; it is often advisable to stop after exposing the macular area.

During the operation, a preretinal membrane that is adherent to the optic disk area may become opaque, to the point of covering the large retinal vessels with a structureless white material, although this area looked completely transparent preoperatively. The opacification probably results from swelling of the preretinal membrane during the operation. Such a membrane is thinner more peripherally, but it may prevent reattachment of the retina.

It is remarkable that membranes that are completely invisible preoperatively can be identified under the surgical microscope after the corneal button and lens are removed. Removal of the membranes is generally the key to successful flattening of the retina. Some cases of recent severe massive preretinal retraction have a very thin, almost invisible preretinal membrane that cannot be dissected off the retina. It appears as a soft, whitish preretinal coating that, in places, shows myriad preretinal capillaries. Attempts at dissection cause many areas of bleeding. I have been unable to identify such cases preoperatively and they have not been helped by open-sky vitreous surgery.

The surgeon should be on the constant lookout for the possible existence of retroretinal bands and membranes, which are not rare and which may make retinal reattachment impossible. They may be invisible preoperatively. When diagnosed during the operation, their extent should be evaluated since they may make all further surgical efforts useless. In rare cases it has been possible to section or remove a retroretinal membrane by approaching it through a sizable retinal break. Such membranes are elastic and cut easily. Some retroretinal membranes are particularly developed around the area of the optic disk, making their surgical removal impossible by present techniques.

During the operation it may be possible to remove subretinal fluid by sucking it out through a retinal break. Therefore, it is important to examine the vitreous cavity for the presence of retinal breaks. They are located from information obtained by preoperative ophthalmoscopy, or they may be found after membranes that obstructed their visualization are removed. Sometimes an unseen retinal break is detected by a change in the appearance of the remaining fluid vitreous. As a rule in cases undergoing open-sky vitrectomy, subretinal fluid is easily distinguished from fluid vitreous because of its yellowish tinge, syrupy consistency, and different refractive index. Subretinal fluid mixed with fluid vitreous often looks like syrup mixed with water. Once a retinal break of appropriate size and location is found, subretinal fluid can be sucked through it with a 1-ml syringe equipped with a blunt 30-gauge irrigator tip. It is often unnecessary to introduce the needle inside the subretinal space. If the break is under liquid vitreous, placing the needle tip in front of the break is sufficient (Fig. 10). One should be careful not to enlarge the break or suck its edge into the needle. If the retina surrounding the retinal break flattens and subretinal fluid remains trapped more posteriorly, changing the position of the patient's eye or head may force the remaining fluid to accumulate around the break. One should never introduce the needle into the subretinal space, in a location where it cannot be observed, because the needle may traumatize the choroid and cause a hemorrhage.

In difficult cases, it may be preferable

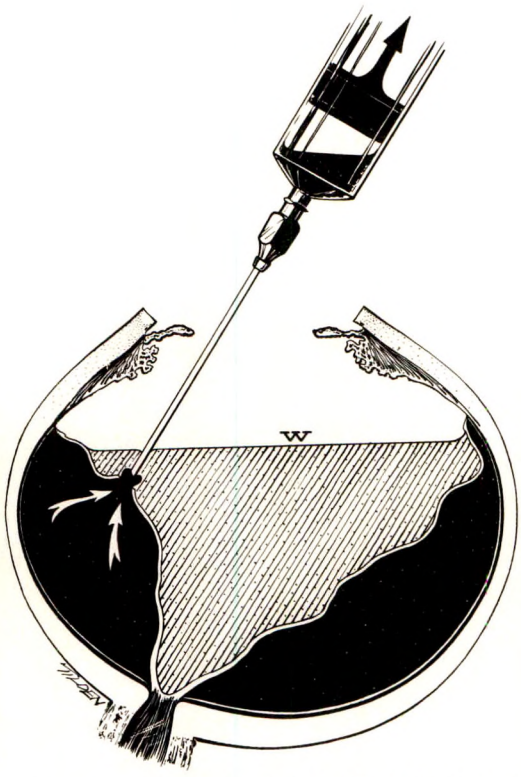


Fig. 10 (Schepens). Subretinal fluid drained through retinal break. Break is placed under water (W), then 1-ml syringe armed with blunt-tip 30-gauge needle is placed in front of break. Steady suction generally succeeds in emptying subretinal cavity.

not to insist on total mobilization of fixed retinal folds because this may not help a subsequent retinal reattachment. It may be better to cut a membrane that unites two tight retinal folds than to attempt its laborious and often futile total blunt dissection. However, retina of normal thickness has been found unexpectedly resistant to the trauma caused by membrane dissection from the retinal surface.

Wound closure—Each radial iridotomy is sutured with one 10-0 monofilament nylon suture; the knots are tied posteriorly (Fig. 11). It is important to place the suture about 1 mm from the pupillary edge in order to have a sufficiently large pupil for postoperative examination. If a sector iridectomy was done at a previous operation, the edges of the iridectomy are sutured. Since iris tissue tears easily, very little traction should be exerted on the suture. If the iridectomy was broad, its edges cannot be apposed. The sutured iris makes reformation of the anterior chamber easier and helps to avoid anterior iris synechiae during the postoperative period because a somewhat rigid iris diaphragm is formed.

Since open-sky vitrectomy is a lengthy procedure, the cornea becomes markedly swollen, and accurate suturing of the corneal button is more difficult than in a corneal transplant. Two sets of sutures are used. First, three separate sutures of 9-0 virgin silk maintain the cornea in proper position. Then, continuous 10-0 monofilament nylon is used to make five to six loops per quadrant. To guarantee closure of the wound even after the cornea deturgesces, an elastic 10-0 monofilament nylon suture must be used and pulled tight (Fig. 12). However, if the suture is too tight, it may tear through some of its corneal loops postoperatively. After the cornea has been sutured, 0.2 ml of a solution containing 4 mg of dexamethasone (Decadron) per milliliter is injected into the vitreous cavity, and the vitreous cavity and anterior chamber are filled with hyaluronic acid.

Retinal reattachment procedure—The open-sky vitrectomy decreases or eliminates vitreoretinal traction. It does not close retinal breaks. Therefore, this operation does not bring about reattachment of the retina except when no open retinal breaks were present. The timing of a scleral buckling procedure to close retinal breaks varies according to the case; most often it is done within two weeks of the open-sky vitrectomy. Sometimes the scleral buckling is performed at the same session as the open-sky vitrectomy. In general, however, results appear to be

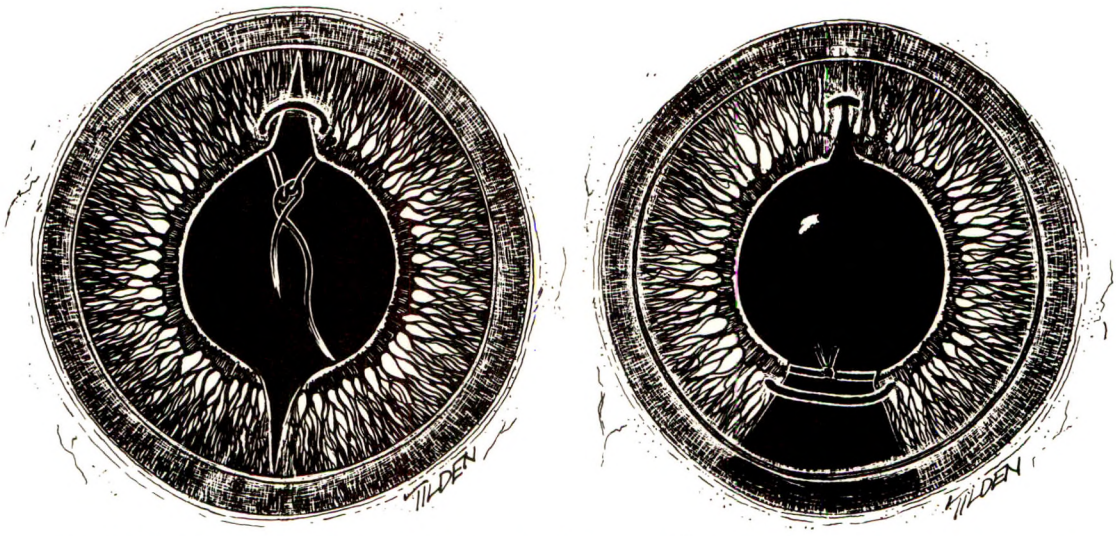


Fig. 11 (Schepens). Left, Iridotomies are sutured with 10-0 monofilament nylon, tied backward. Sutures are placed distal to pupillary edge to avoid making pupil too small. Right, In eye with previous broad iridectomy, iris suture is tied with enough tension to maintain rigid iris plane and prevent anterior synechiae. No effort is made to join iris edges.

best when the buckling can be performed within two weeks after the vitrectomy.

Indications and results

The indications for subtotal open-sky vitrectomy have changed with increased experience.[15] At present, they encompass a wide variety of cases. Of 297 consecutive operations, performed between 1971 and 1979 by Dr. Hirose or myself, complete information is available for 290 cases. Retinal reattachment was obtained in 61 of 290 eyes (21%) and substantial improvement in visual function in 36 of 290 eyes (12.4%). The best visual improvement was from finger counting to 6/12 (20/40). It must be remembered that all eyes operated on by subtotal open-sky vitrectomy had been considered inoperable by a simpler procedure. Many of the patients were referred by other competent vitreoretinal surgeons. In 98% of the cases, best corrected preoperative visual acuity was finger counting or worse. Other surgical procedures had been performed on 261 (87.9%) eyes: scleral buckling, cryopexy, or both, in 117.6% of the total number of cases (the percentage above 100% indicates that some eyes underwent the same procedure more than once), anterior segment operation in 54.1%, closed vitrectomy in 23.8%, and other operations, including silicone oil injection, photocoagulation, keratoplasty, or choroidal tap, in smaller percentages.

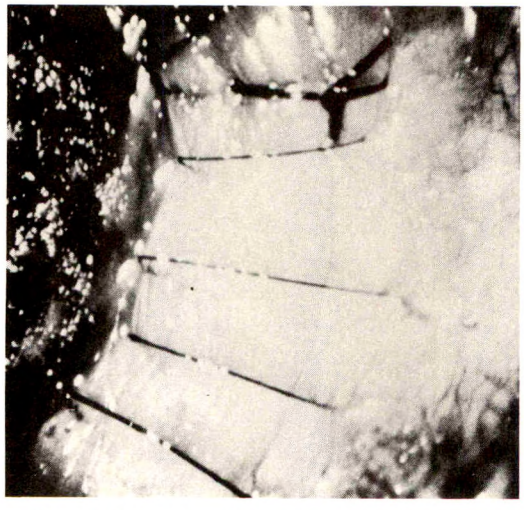


Fig. 12 (Schepens). Sutured cornea, showing elastic nylon suture under tension over swollen cornea.

An analysis of results according to preoperative condition showed that different groups have yielded different results. The Table gives a breakdown of some categories.

Tractional retinal detachment—In most patients in this group a tractional retinal detachment was not diagnosed preoperatively because initially they had a severe rhegmatogenous retinal detachment, treated by one or several scleral buckling operations and followed by massive preretinal retraction. Upon completion of the open-sky vitrectomy, the retina reattached without further retinal surgery, indicating that a previous scleral buckling operation had closed the retinal breaks and that the remaining retinal detachment with massive preretinal retraction was purely tractional. As the Table shows, eyes with tractional detachment are the group in which open-sky vitrectomy was most successful. The problem is that the purely tractional nature of these cases cannot generally be diagnosed preoperatively because the absence of a visible retinal break, even after the most exhaustive preoperative ophthalmoscopic examination, is not solid evidence that no break exists. Vitreous haze and multiple retinal folds can easily hide retinal breaks from view.

TABLE

RESULTS OF 297 SUBTOTAL OPEN-SKY VITRECTOMIES

Preoperative Condition*	No. of Cases	Reattachments	
		No.	%
Tractional retinal detachment	30	16	53.3
Retrolental fibroplasia	10	3	30.0
Retina adherent to anterior segment structures	10	3	30.0
Perforating injury	47	14	29.8
Massive preretinal retraction	201	28	13.9
Giant retinal tear	45	4	8.9

*Some eyes had more than one of the listed conditions.

Case 1—A 57-year-old man with a low degree of myopia developed a temporal giant tear extending from the 7 to almost the 11 o'clock position in the right eye. His first retinal operation, performed elsewhere, had been unsuccessful. Examination showed a giant tear with markedly rolled edge and several large star-shaped folds temporally. A second operation, consisting of a closed vitrectomy and 360-degree scleral buckling, was performed. Five weeks later, extensive fixed folds, resembling the early stage of massive preretinal retraction, developed but the optic disk remained visible (Fig. 13). An open-sky vitrectomy was performed eight weeks after the second operation. Preretinal membranes were peeled over an extensive area of the fundus but the scleral buckling was not revised. Postoperatively the retina slowly reattached during a period of about three months. This course of events indicates that the giant tear had been closed by the second operation and that the extensive fixed retinal folds that ensued caused a purely tractional detachment. Visual acuity improved from hand movements at 4 feet to 6/120 (20/400), after 11 months.

Retrolental fibroplasia—Eyes with this condition that are suitable for subtotal open-sky vitrectomy show an extensive or total nonrhegmatogenous retinal detachment without glaucoma or flat anterior chamber, in a patient who is a few months to about 2 years old. Such patients cannot generally be operated on safely by closed vitrectomy, because the vitreoretinal adhesions and new vessels are located in the fundus periphery where they are hard to treat by that procedure. Another difficulty is that the ora serrata is quite anterior in infants affected by retrolental fibroplasia, making it difficult or impossible to introduce the nibbler safely through a pars plana incision and to perform the vitrectomy without damaging the lens, the peripheral retina, or both.

Infants and small children tolerate an open-sky procedure particularly well; dissection of new-formed tissue in the vitreous is often easy in these patients. The area of the vitreous base, where many new vessels cannot be differentiated from dilated retinal vessels, should not be interfered with. Because of the

young age of these patients, significant data on the degree of functional recovery have not yet been collected.

Retina adherent to anterior segment structures—After a penetrating eye injury with disruption of the anterior segment and vitreous loss, the detached retina may become adherent to the damaged ciliary body, iris, posterior lens capsule, or cornea. Occasionally, a retinal adhesion to an anterior structure follows an anterior segment operation that had a stormy course. For instance, a cataract extraction with vitreous loss may end up with not only the vitreous but also the peripheral retina incarcerated into the cataract wound or adherent to the pupillary edge. When vitreous gel becomes incarcerated into a pars plana incision for closed vitrectomy, the peripheral retina may be dragged toward the sclerochoroidal incision and form a large, intractable fixed retinal fold.

In such desperate cases, attempts to reattach the retina by scleral buckling are futile unless the retina is first mobilized. In simple cases, a narrow retinal adhesion may be severed with vitreous scissors. In severe instances, open-sky vitrectomy should be attempted.

Case 2—An 8-year-old boy was hit in the left eye by a flying piece of wood. He sustained an extensive vertical laceration of the cornea and sclera, with iris and vitreous prolapse. The laceration was repaired immediately. Postoperatively a cataract developed that was absorbed spontaneously over several weeks, and the retina became detached. The eye showed a vertical scar across the cornea and inferior sclera, with the iris adherent to it below. The retinal detachment was total and fixed; a large inferior fixed fold was attached to scar tissue anteriorly, dragged the macula below, and covered the ora serrata. A large horseshoe-shaped tear was present in the fold (Fig. 14). A circular subretinal band of new-formed tissue was visible around the optic disk. A dense white scar had invaded the vitreous base and extended clockwise from the 4 to 9 o'clock positions.

Open-sky vitrectomy was performed to free the retina from its anterior adhesions. During the operation it was found that the retina adhered extensively to the ciliary processes and to the iris, the iris itself being adherent to the corneal scar at the 6 o'clock position. After the retina was freed from its adhesions, a scleral buckling was performed with silicone implant and circling silicone band. The two operations were completed during the same surgical session. The patient made an uneventful recovery and the retina became reattached except for a sharp, narrow retinal fold extending meridionally from the optic disk to the buckle. Visual acuity improved from hand movements at 4 feet to 6/240 (5/200).

Other types of perforating injury—Perforating injuries that involve the lens and vitreous may require a closed vitrectomy within two weeks of repair of the initial damage. In certain cases, a subtotal open-sky vitrectomy may be indicated. Instances in which the retina is adherent to anterior segment structures have been discussed in the preceding section. In other cases, extensive preretinal membranes are adherent to the inner retinal surface, or the eye contains a large intraocular foreign body.

Neglected intraocular foreign bodies are often associated with partially or completely opaque vitreous caused by hemorrhage or a subsequent inflammatory reaction. The retina may be detached. If the vitreous foreign body is nonmagnetic and embedded in scar tissue, surgical exploration by the open-sky technique may be the best method of removing the opaque vitreous, the scar tissue, and the foreign body (Fig. 15). For instance, numerous particles of partly disintegrated wood have been removed successfully from a badly injured eye by the open-sky technique. Small fragments of partly decayed intraocular wood, enmeshed in intraocular scar tissue, may not be detected either by X-rays or by A- or B-scan ultrasonography.

An open-sky vitrectomy may be advisable in an eye with an intraocular foreign body too large for safe removal by the standard approach through the pars plana ciliaris. Such large foreign bodies can be violently irritating, but sometimes the eye harboring them remains quiescent. However, if a large and inert foreign

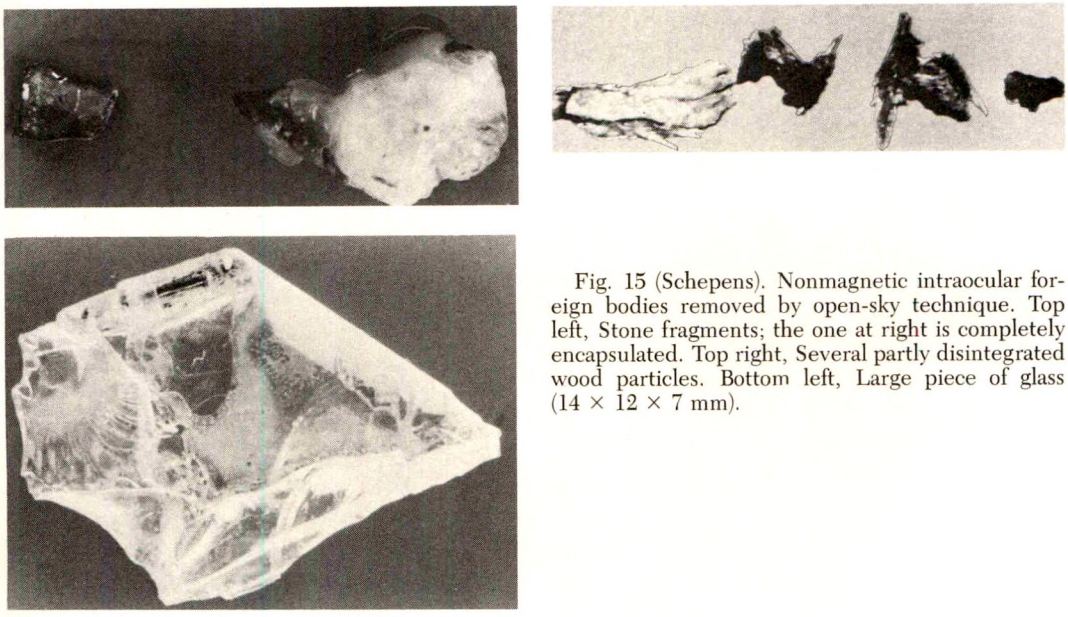


Fig. 15 (Schepens). Nonmagnetic intraocular foreign bodies removed by open-sky technique. Top left, Stone fragments; the one at right is completely encapsulated. Top right, Several partly disintegrated wood particles. Bottom left, Large piece of glass (14 × 12 × 7 mm).

body fails to become encapsulated and floats freely in the vitreous cavity, it gradually breaks down the vitreous gel and results in new-formed preretinal membranes that may destroy sight. In such a case, early operation should be encouraged. A piece of glass measuring 14 × 12 × 7 mm was successfully removed from the vitreous cavity with a subtotal open-sky vitrectomy.

Massive preretinal retraction—A substantial number of cases of early massive preretinal retraction can be treated successfully by a high and wide scleral buckling procedure with circling element and injection of physiologic solution, air, or hyaluronic acid into the vitreous cavity. However, when massive preretinal retraction is severe, the retina either fails to flatten with injections into the vitreous cavity or redetaches days to weeks postoperatively. Additionally, the surgical failure generally makes the case worse. In a number of instances where the above treatment is likely to fail, a two-instrument closed vitrectomy may succeed. In cases in which experience shows that a two-instrument closed vitrectomy would have no chance of success, an open-sky vitrectomy may prove of value. Two types of cases must be distinguished: massive preretinal retraction with breaks that have been closed by previous surgery and massive preretinal retraction with open retinal breaks.

Case 3—A 16-year-old boy had had many discissions for bilateral congenital cataracts by the age of 3 years. His vision was adequate to cope with school until he was 15 years old, when a retinal detachment developed in the left eye. A scleral buckling failed, and the detachment became inoperable because of dense preretinal and vitreous organization. Visual acuity was reduced to light perception with inaccurate projection. The fundus of the right eye could not be examined adequately because of lens remnants. Since vision in this eye was very poor, an iridocapsulectomy was performed. A retinal detachment developed a few days after operation and was judged inoperable.

A few months later, I first examined the patient. His right eye had a somewhat mobile retina but the periphery could not be examined because of a bound-down pupil. After a second iridocapsulectomy, a scleral buckling with air injection was performed unsuccessfully. A second operation, in which the scleral buckling was extended around 360 degrees, was quickly followed by advanced massive preretinal retraction. The optic disk was hidden by fixed retinal folds and vitreous organization made

fundus details only hazily visible. Visual acuity was hand movements at 2 feet. Vitreoretinal adhesions were so numerous and extensive in the right eye that closed vitrectomy was thought to have no chance of success.

Open-sky vitrectomy was done one month after the 360-degree scleral buckling. Fibrotic membranes embedded in formed gel and bridging numerous retinal folds were found, and retracted cortical gel was also present in the sulci between the folds. Individual membranes were dissected off the retina piecemeal, and an intraocular balloon served as a soft-tissue probe to help free the retina. A small retinal break was noted anteriorly, on the buckle, at the 6 o'clock position and was left untreated.

The postoperative course was uncomplicated, and the patient left the hospital with fairly clear media. The detached retina was well visible and mobile after ten days. By 25 days after the operation, the retina had completely reattached, and visual acuity was 6/90 (20/300). By ten months, corrected visual acuity had improved to 6/18 (20/60), and the patient could read J1 with a +5-diopter bifocal addition. This visual acuity has been maintained for more than eight years. The course of recovery indicates that the anterior retinal break became closed by the existing scleral buckling, thus transforming the case into a tractional retinal detachment, which was relieved by open-sky vitrectomy.

In eyes with massive preretinal retraction and open retinal breaks, the retina may be mobilized by open-sky vitrectomy, despite the presence of strong and vascularized preretinal membranes (Fig. 16). However, another scleral buckling operation is often necessary to reattach the retina. The following example illustrates this type of case.

Case 4—A 53-year-old woman had a retinal detachment in the right eye. It was operated on by scleral buckling twice without success. A retinal break in the left eye was treated successfully by photocoagulation.

When first examined here, her best corrected visual acuity in the right eye was hand movements. The fundus presented the typical pattern of massive preretinal retraction with the optic disk still visible. The vitreous was grossly organized between the equator and the extreme fundus periphery, with an area of strong fibrosis and particularly marked retinal traction in the lower nasal quadrant. A medium-size break was present at the 1:30 o'clock position, posterior to the equator.

A closed vitrectomy with two instruments was performed uneventfully, followed by a 360-degree buckling procedure during the same surgical session. Postoperatively, the retina became reattached below but remained detached above and nasally. Marked vitreous organization was still present between equator and extreme fundus periphery, keeping the retina detached all around in the periphery. Visual acuity was unimproved. It was thought that a closed vitrectomy could not effectively deal with such peripheral and strong vitreoretinal adhesions.

An open-sky operation was performed four months after the closed vitrectomy, followed by a revision of the scleral buckling three weeks later. The opera-

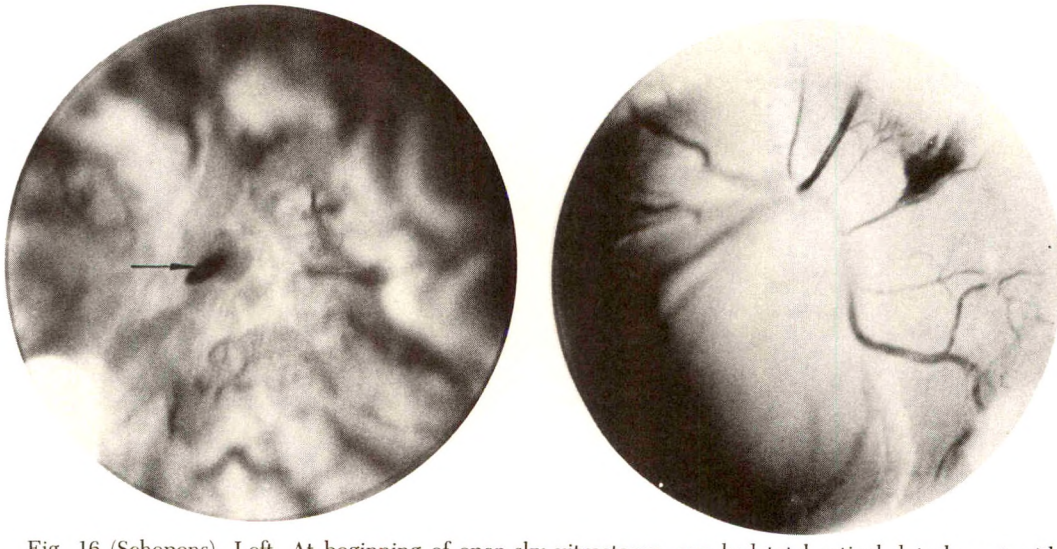


Fig. 16 (Schepens). Left, At beginning of open-sky vitrectomy, eye had total retinal detachment with massive preretinal retraction. Dark area (arrow) is an opening, located in front of the hidden optic disk, in thick, vascularized preretinal membrane. Right, Posterior pole at the end of the operation.

tions and postoperative courses were uneventful and the retina became completely reattached. When the patient was last seen, 14 months after the last operation, corrected visual acuity had improved to 6/60 (20/200).

Giant retinal tear—This type of case may be operated on successfully by a scleral buckling procedure with closed vitrectomy and large air injection. However, when a giant retinal tear is associated with massive preretinal retraction, such treatment almost uniformly fails. Attempts at using open-sky vitrectomy, accompanied or followed by scleral buckling, have shown a low rate of success (four of 45 cases, or 8.9%). Figure 13 depicts a successfully treated case. Recently Hirose developed a technique that seems to improve the success rate considerably.[16] It involves suturing the edge of the giant tear to the peripheral choroid. A report of stable visual results must await a longer follow-up.

Excision of tough tissue—In two cases, not listed in the Table, tissue that had grown into the vitreous cavity was so tough and compact that no nibbler could remove it or even cut into it. It was smooth, glistened like normal tendon, and formed a strand 1 to 1.5 mm thick that extended from the posterior pole toward the anterior portion of the globe. In one case, this tissue may have resulted from severe trauma, but in the other it probably developed as a result of hemorrhage or inflammation. The thick strand shrank with time and caused a severe indentation of the wall of the globe, at the location of the strand's anterior insertion over the ciliary body, thus distorting the globe's external shape. Because of total retinal detachment, the preoperative intraocular pressure was very low and the globe distortion was misinterpreted as representing early phthisis bulbi. The differentiating feature was that the globe was distorted in one area only, instead of its curvature being flattened by the four recti muscles as is the rule in phthisis bulbi.

Case 5—A 28-year-old man suffered from Marfan's syndrome with dislocated lenses. In the right eye, an initial retinal procedure was performed without success. A few weeks later a scleral buckling procedure was done and resulted in a total retinal detachment with vitreous hemorrhage and massive preretinal retraction. The scleral buckling was revised to no avail. These three operations were performed elsewhere. The left eye had no light perception as a result of a retinal detachment of several years' duration that had never been operated on.

I first saw the patient one year after his third retinal operation. The lens was opaque and dislocated into the vitreous in both eyes. In the right eye, the sclera was flattened in the 11 o'clock area and the retina was totally detached with strong vitreous organization that was partly obscured by the opaque dislocated lens. In the left eye, the retina had spontaneously reattached but was atrophic and showed scattered pigmentation.

Open-sky vitrectomy was performed on the right eye. After removal of the opaque lens, extensive vitreous organization was seen, in the middle of which was a dense, glistening, white strand of tissue about 1.5 mm thick, which extended from the ciliary processes, near the 11 o'clock position, to the posterior pole. This strand was invisible preoperatively. After removal of organized vitreous gel, an attempt was made to sever the strand, which was obviously under tension. Since this could not be accomplished with vitreous scissors, it was necessary to use squint scissors (Fig. 17). A 13-mm section of the strand was excised. The cut ends that remained inside the globe retracted but did not bleed, exposing detached retina with extensively occluded retinal vessels. The eye recovered well from this operation, and the retina showed one equatorial retinal break at the 9 o'clock position. Six weeks later, a scleral buckling procedure with a silicone implant and circling silicone band was performed successfully. Postoperatively the patient showed gradual visual improvement but was lost to follow-up after six weeks as he lived abroad.

COMPLICATIONS

Because most eyes selected for open-sky vitrectomy are battered eyes that have already been subjected to many operations and have extremely severe retinal detachments, it is surprising that the number and severity of the complications are relatively limited.

Complications during the operation—Hemorrhage is the most frequent complication during the operation. Fortu-

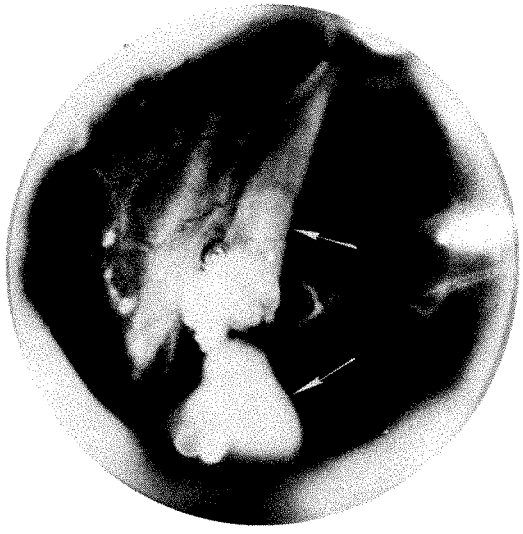


Fig. 17 (Schepens). Glistening tendon-like strand (arrows), 1.5 mm in diameter, extends from posterior pole to ciliary processes up and temporally. Because it could not be cut with vitreous scissors, it had to be severed with squint scissors. Closed retinal vessels (white lines) are visible in detached retina.

nately, most hemorrhages are small and easily controlled with underwater diathermy. Imprudent attempts at dissecting tissue located over the ciliary processes, where visualization is more difficult, may result in bleeding that is sometimes hard to control because its source cannot be pinpointed. New vessels in the vitreous cavity can produce a major hemorrhage. This is why, when these vessels can be seen preoperatively, they should be cauterized with underwater diathermy by the closed technique before starting the open-sky vitrectomy.

Retinal vessels can also bleed, rarely profusely, when attempts are made to dissect vitreoretinal adhesions. These adhesions are always firmer over the course of major retinal vessels. The greatest danger is during dissection around the optic disk because of strong vitreoretinal adhesions and the large caliber of vessels in this area. Choroidal hemorrhage is rare; if it is subchoroidal and large, it may cause an expulsive hemorrhage. When the hemorrhage is subretinal and large, the retina may be extruded through the corneal trephine and then rupture. Such accidents are unpredictable and have occurred in about 3% of open-sky vitrectomies.

There is no known method of controlling a large subretinal hemorrhage. When the bleeding is subchoroidal, incising the sclera may help but, because the extravasated blood clots rapidly, the sclerotomy should be at least 4 to 5 mm long. As soon as possible, the corneal button should be resutured. About five days postoperatively, another sclerotomy can be done to evacuate remaining subchoroidal blood, which by that time has liquefied. Lost volume is replaced with physiologic solution injected into the anterior chamber. This procedure, even when carried out successfully, has so far not prevented phthisis bulbi.

The postoperative complications—The most important postoperative complications of open-sky vitrectomy affect the cornea, iris, and retina.

CORNEAL COMPLICATIONS—Corneal complications have decreased substantially with greater surgical experience. Sometimes, new vessels grow through the closed corneal wound one to three months postoperatively and form a retrocorneal fibrovascular membrane (Fig. 18). Fluorescein angiography is useful in demonstrating the full extent of neovascularization. Factors that seem to precipitate corneal and retrocorneal neovascularization are a corneal incision too close to the corneoscleral limbus, anterior segment ischemia from several previous intraocular operations, preexisting vascularized corneal scar, and perhaps damage to the corneal endothelium. Conditions of systemic origin, such as diabetic retinopathy, may precipitate corneal neovascularization.

Corneal erosion is not rare but may remain undiagnosed until the corneal

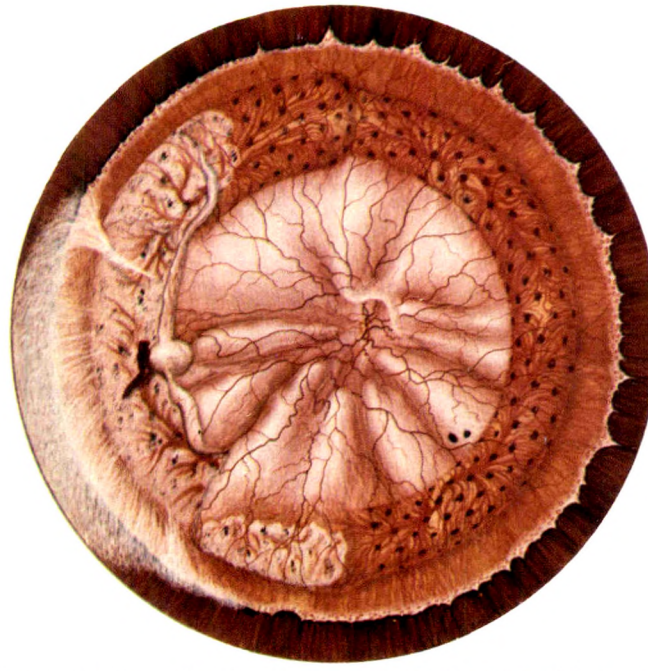


Fig. 13 (Schepens). Case 1. After two scleral bucklings and closed vitrectomy, right eye still shows giant retinal tear temporally. There is total retinal detachment with extensive fixed retinal folds. Giant tear has rolled posterior edge and seems to leak across buckle. There is a blood clot in giant tear at the 9 o'clock position. Scars from cryoapplications are visible in area of giant tear. Scars from diathermy applications can be seen elsewhere on buckle around 360 degrees. Ora serrata is obscured by long-standing hemorrhage remnants from the 7 to 10 o'clock positions.

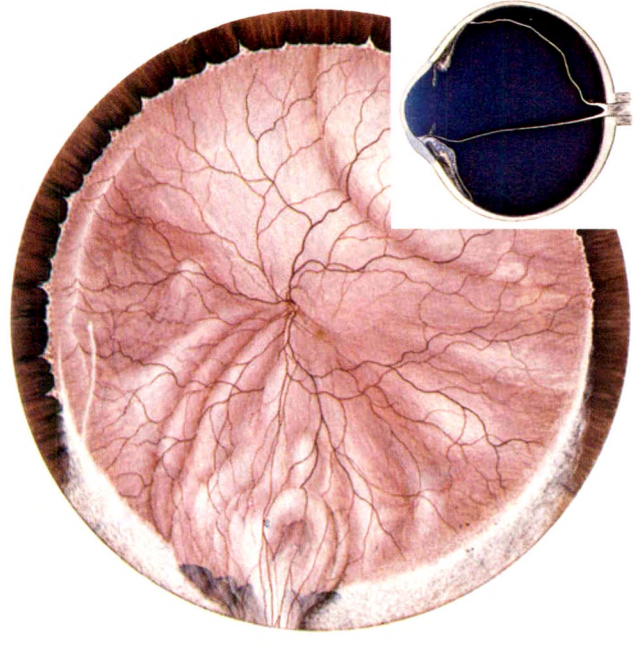


Fig. 14 (Schepens). Case 2. Left eye has total retinal detachment caused by perforating trauma. Fixed retinal fold is dragged out of view, beyond ora serrata at the 6 o'clock position. Vitreous haze, caused by hemorrhage, obscures fundus periphery between the 4 and 8 o'clock positions. There is a horseshoe-shaped retinal tear at the 6 o'clock position. Macula is dragged inferiorly. There is a fixed retinal fold, near the ora serrata, between the 8 and 10:30 o'clock positions. Subretinal band forms a circle around the optic disk. Inset, Cross section of eye showing retina incarcerated into scar and involving the iris and cornea at the 6 o'clock position. Note ring of subretinal tissue near the optic disk.

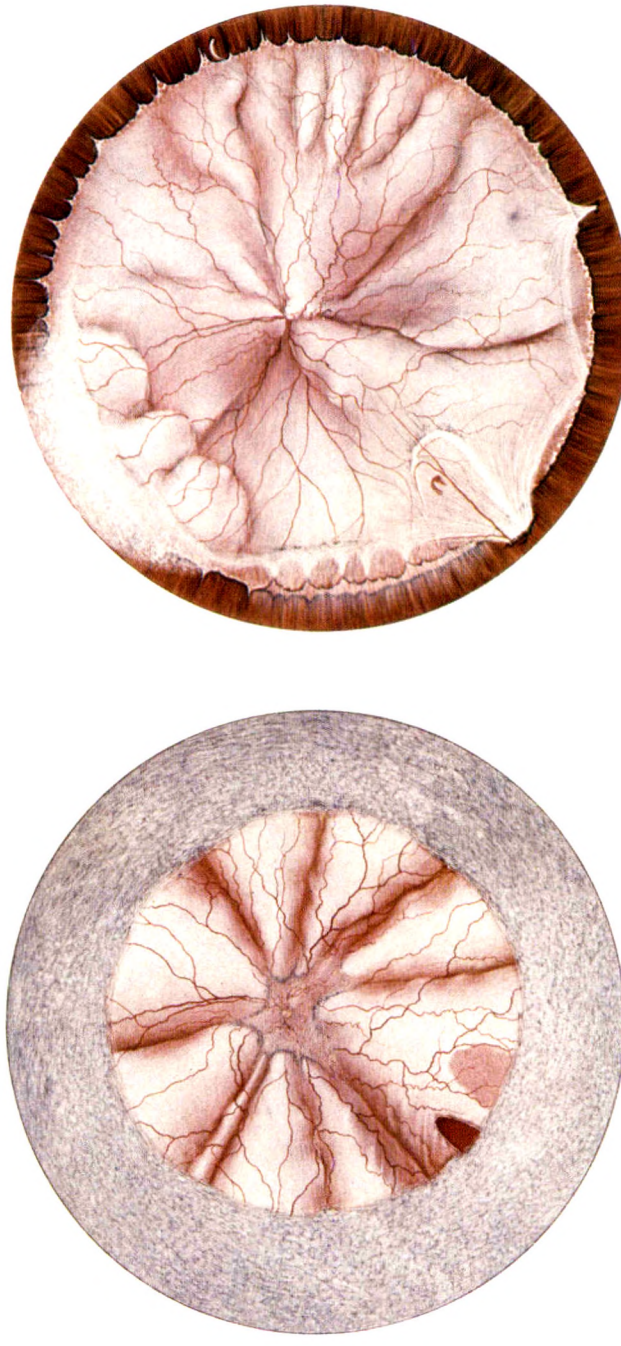


Fig. 21 (Schepens). Case 6. Top, Fundus of left eye after two scleral buckling operations and closed vitrectomy. There is a total retinal detachment with all retinal folds fixed and massive preretinal retraction. Optic disk is invisible. Cellular growth from pars plana into vitreous, at the 11 and 2:30 o'clock positions, results from vitreous injections done at the time of scleral buckling operations. Ingrowth with neovascularization at the 4:30 o'clock position is entry site of nibbler. Partial organization of vitreous base from the 4:30 to 9 o'clock positions has produced detachment of the ora serrata between the 5 and 6 o'clock positions. There is a retinal tear at the 4:30 o'clock position. Scleral buckling is not visible. Bottom, Same fundus, three weeks after open-sky vitrectomy. Optic disk is visible and retinal folds around it are fairly low; retinal detachment has remained total with massive preretinal retraction. Retinal folds are completely fixed by thick, pigmented, newly formed preretinal membranes. The vitreous is more hazy than it was preoperatively. Traction has increased the size of the retinal tear. Fundus periphery was not examined.

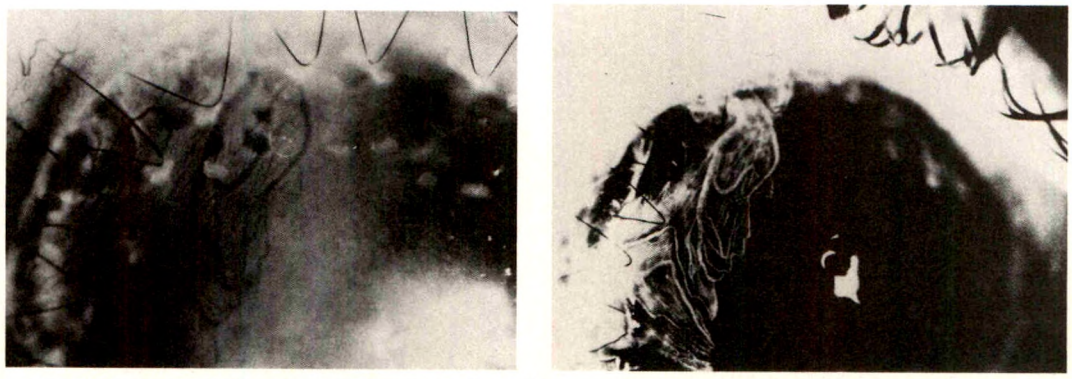


Fig. 18 (Schepens). Vascularized retrocorneal membrane grew through tightly closed corneal incision two months postoperatively, causing gradual opacification of cornea. Left, Standard photograph. Right, Fluorescein angiogram.

damage is severe. The erosion, which begins below the center of the cornea, may not occur for several weeks after operation. There seem to be four basic causes for the condition: (1) preexisting anterior segment ischemia, which is more frequent in older patients and often follows repeated scleral buckling operations; (2) corneal neuropathy manifested by preoperative corneal paresthesia or anesthesia, a condition that is frequent after several ocular operations and in diabetics; (3) imperfect eyelid closure; and (4) a dry eye preoperatively. Other factors, especially the continued intensive use of topical corticosteroids, irritation by loose corneal sutures, and corneal infection, have a precipitating influence.

Because corneal erosion may result in severe corneal damage, the intactness of the corneal epithelium should be checked daily while the patient is in the hospital. In case of doubt, the eye should be examined under the slit lamp after instillation of fluorescein. At the time of discharge, if there is any erosion or even simple roughness of the corneal epithelium, the eye must be kept closed with a mildly compressive dressing. After the patient leaves the hospital, the cornea should be checked weekly for at least six weeks. If erosion is diagnosed, all topical medication is omitted, except an antibiotic ointment once daily, and the eye is closed with a moderately tight patch; if there is any suspicion of infection, a culture of the conjunctival sac is taken and the cultured microorganisms are tested for their sensitivity to antibiotics. If the erosion does not begin to heal after five to seven days of patching, referral to a cornea specialist is indicated. He may recommend the use of a soft contact lens until healing is complete.

If erosion is permitted to progress unchecked, loops of the continuous corneal suture that are too superficial may erode through corneal tissue and cause irritation. The loose suture should be removed but resuturing of the cornea is rarely needed. A corneal ulcer may develop and even perforate if the corneal erosion is untended and becomes infected.

Decompensation of the corneal endothelium may lead to corneal edema and epithelial bullae. The stroma may become vascularized and permanent opacities may develop in the subepithelial area and stroma. This type of complication is precipitated by preexisting damage to the corneal endothelium. In many cases, a corneal transplant can reestablish corneal transparency.

IRIS AND RETINAL COMPLICATIONS—Postoperative rubeosis iridis is rare after open-sky vitrectomy. When it occurs, it may be accompanied by ectropion uveae. The membrane that has grown over the anterior surface of the iris may become continuous with a cyclitic membrane that covers the back surface of the iris and the ciliary processes. The membrane may extend over the vitreous base and the detached retina; it may shrink so much that is causes an adhesion of the retina to the pupillary border. This severe complication cannot be remedied.

The main retinal complication is regrowth of the preretinal membrane. Factors affecting regrowth, which may be very rapid, are largely unknown, except for the development of fibrin membranes described later in this article. In rare cases, a neovascularized preretinal membrane seems to originate from conjunctival vessels that grow under the corneal epithelium, through the tightly closed corneal wound, forming an intraocular membrane that continues its growth on the front and back surfaces of the iris, the ciliary processes, the unexcised remains of the vitreous base, and the whole inner retinal surface.

Unavoidably, after an extensive operation on a badly damaged eye, phthisis bulbi is bound to occur in some cases. Painful phthisis bulbi requiring enucleation has been surprisingly rare.

RESEARCH ASPECTS

Research aspects of subtotal open-sky vitrectomy cover two areas: globe incision, and new data on the pathogenesis of massive preretinal retraction.

GLOBE INCISION

Most of the present experience in globe incision for open-sky vitrectomy is through the cornea (Fig. 19). However, a corneal incision has certain disadvantages: Corneal transparency may be compromised, iridotomies and lens extraction are necessary, and peripheral anterior iris synechiae, sometimes with closure of the filtration angle, may occur. Additionally, the distance between the narrowest portion of the wound (pupillary plane) and the posterior pole is relatively great—about 21 mm.

A 170-degree scleral incision (Fig. 19) can be made over the pars plana ciliaris, 3.5 to 4 mm from the corneoscleral limbus.[17-19] The advantages of this technique are that the cornea and iris remain intact, peripheral anterior synechiae are less likely to form, there is no tendency to closure of the filtration angle, the wound opening is larger, and the distance between the wound opening and the posterior pole is shortened by more than 4 mm. However, the lens must often be removed at the end of the operation because of subluxation or damage to its posterior capsule.

In monkey eyes, mild postoperative necrosis of the ciliary processes facing the incision and a certain degree of anterior segment ischemia without corneal damage are evident postoperatively.[17] These changes may prove to be more severe in older human patients whose eyes have been previously subjected to several operations than in young experimental monkeys with intact eyes. So far, human eyes in which a 170-degree incision of the pars plana ciliaris was made have shown some ischemia of the iris and ciliary body but no massive necrosis. Another concern is the possibility of postoperative downgrowth of connective tissue through the extensive sclerochoroidal incision. This has not happened in monkeys observed for three years. However, in human eyes with severe pathologic changes and lowered tissue vitality, this danger may be real. The experience with monkey eyes and a few human eyes has been encouraging but more information is needed before this procedure can be recommended.

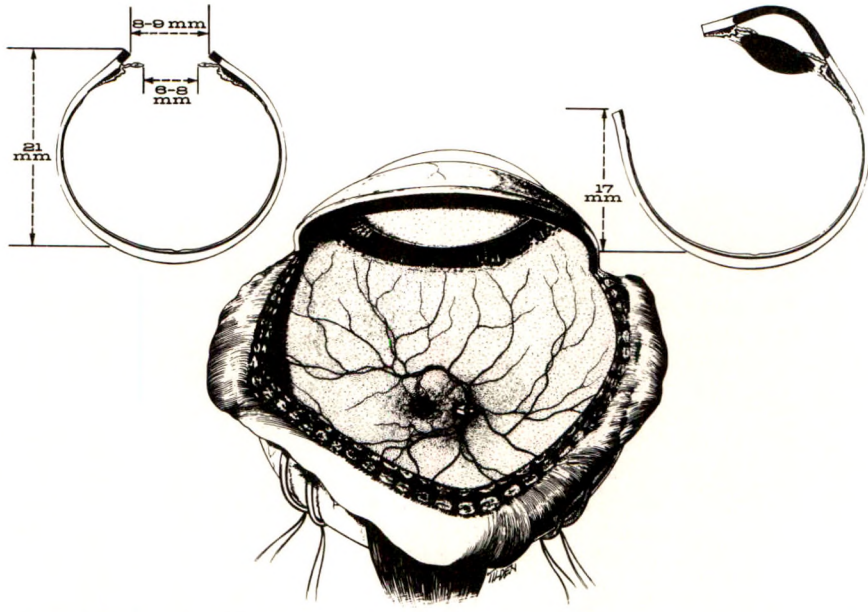


Fig. 19 (Schepens). Two approaches to vitreous cavity in open-sky vitreous surgery. Schema at top left shows that corneal button creates a corneal opening of 8 to 9 mm and an effective pupillary opening of 6 to 8 mm, located about 21 mm from posterior pole. Schema at top right shows that a 170-degree incision through the pars plana ciliaris gives a much wider effective opening and distance of about 17 mm from posterior pole. Large drawing shows full view of 170-degree incision made through the pars plana ciliaris.

New data on the pathogenesis of massive preretinal retraction

Massive preretinal retraction has generally been described as resulting from the development and proliferation of preretinal cellular membranes.[20] In certain cases the proliferation is both preretinal and retroretinal.[21] It has long been known that, in unoperated eyes, vitreous organization and preretinal membranes tend to occur mainly when a retinal detachment has been present for a long time.[22] The spontaneous development of massive preretinal retraction in unoperated eyes, a short time after the onset of retinal detachment, rarely occurs. In contrast, one or several unsuccessful operations have precipitated the development of vitreous organization and massive preretinal retraction in many eyes, although the retinal detachment was relatively recent. Therefore, retinal surgery seems to be an important precipitating factor of massive preretinal retraction.

Preretinal membranes are composed of both cellular and acellular elements. The cellular components have attracted attention for many years. Early authors, particularly Gonin,[22] studied the role of epithelial cells, some of which are pigmented while others are not; he thought that these cells proliferated from the ciliary epithelium into the anterior vitreous and on the inner retinal surface. More recently, other authors thought that pigment epithelial cells migrated through the retinal breaks and proliferated over the inner and outer retinal surfaces.[23] It is also well established that preretinal and retroretinal membranes often contain glial cells[24,25] and fibroblasts.[25] In a number of cases, the cellular elements in these membranes include fibroblasts, glial cells, macrophage-like

cells, lymphocytes, histiocytes laden with pigment granules, and new-formed vessels.[26]

In some preretinal and vitreous membranes, cells are scanty and the membranes are composed mainly of two acellular elements: vitrosin (vitreous collagen), which forms densely packed fibrils 10 nm in diameter and 22 nm in periodicity, and adult collagen, with a periodicity of 64 nm, which forms thick fibers.

Fibrin membranes—A novel aspect of preretinal membrane formation is the occurrence of both newly formed and long-standing fibrin membranes (E. Hultsch and C. L. Schepens, unpublished data). During an open-sky operation, a newly formed membrane often develops on the anterior surface of the iris. It is thin, transparent, sturdy yet very elastic, and adheres strongly to the underlying tissue. Therefore, its removal (Fig. 20) must be performed gently to avoid causing bleeding or tearing of the tissue to which it adheres; it must be cut with scissors rather than torn off. A similar membrane may form over all the intraocular vascularized surfaces. During open-sky surgery, these fibrin-like membranes can form within minutes and, after their surgical removal, may reform rapidly. Postoperatively, such membranes often disappear within a few days. They seem to be more frequent and more prominent in young than in old patients.

The impression given by the above observations is that the formation of fibrin-like membranes is a phenomenon peculiar to open-sky vitreous surgery. The following case report demonstrates that this is an incorrect assumption.

Case 6—A 7-year-old boy with emmetropia was found to have a retinal detachment in the left eye, of unknown duration. The right eye appeared normal. Two scleral buckling operations were performed elsewhere without success. Then an uncomplicated closed vitrectomy was done to no avail. I examined the patient one month later. Best corrected visual acuity was reduced to light perception. The fundus showed a total retinal detachment with massive preretinal retraction that was hiding the optic disk (Fig. 21). In the pars plana ciliaris, there were three areas from which strands of connective tissue grew into the vitreous cavity. Two of the areas were sites used for the injection of air or saline, six and eight weeks previously. The third area, located at the 4:30 o'clock position, was the entrance site of the nibbler and contained sizable new vessels. One horseshoe-shaped tear was found near the equator at the 5 o'clock position.

An open-sky vitrectomy was performed uneventfully five weeks after the closed vitrectomy. Samples of membranes from various preretinal sites were collected for study. The postoperative course was smooth but the retina remained highly detached. Three weeks after the open-sky vitrectomy the retina was still detached, with massive preretinal retraction that appeared much more severe than it was before the operation. Dense and easily visible preretinal

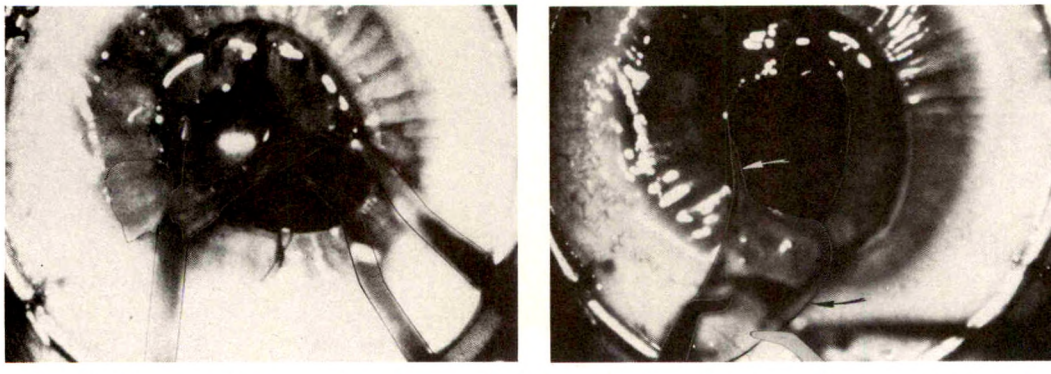


Fig. 20 (Schepens). Fibrin membrane being peeled off anterior iris surface at end of operation. Left, Peeling has started at the iridotomy at the 6 o'clock position (above) and extends to the 9 o'clock position. There is considerable stretching of peeled membrane. Right, Removal is continued (arrows) by combining gentle pulling with blunt membrane dissection.

membranes were present that were partly pigmented and maintained the retinal folds fixed around the optic disk.

Two scleral bucklings with silicone implant and circling silicone band were then performed without success. When last seen, three months after the last operation, the retina was highly detached and completely fixed. It was adherent to the pupillary edge at the 6 o'clock position, and marked rubeosis was present.

Nature of excised tissue—Three kinds of pathologic tissue were removed: (1) preretinal membranes that had been present before the open-sky vitrectomy and which caused the massive preretinal retraction; (2) membranes, formed during the operation, in front of and behind the iris and over the ciliary processes; and (3) a membrane extending into the vitreous from the site of the previous nibbler insertion. Unexpectedly, all preretinal material, excised from several locations in the fundus, was practically identical with the membrane that had formed during the surgery in front of and behind the iris and over the ciliary processes. Figure 22 is a representative electron micrograph of the preretinal membrane, demonstrating fibrin-like material of aggregated fibers interspersed with cellular debris and free pigment granules. Further identification as fibrin was achieved by specific degradation of the membrane with plasmin. Morphologically, the fibrin membrane is very different from vitrosin (Fig. 23). The membrane that extended into the vitreous from the site of nibbler insertion was cellular, in contrast to other membranes that were composed mostly of new-formed scar collagen and seen in eyes that had undergone open-sky vitrectomy (Fig. 24).

Interpretation of findings—During an open-sky vitrectomy, one frequently observes the formation of a fibrin membrane on the anterior surface of the iris. Sometimes a similar membrane is noted over the posterior iris surface or the ciliary processes. It is rare to observe such membranes forming over the inner retinal surface during an operation.

In the above case, the preretinal membranes that were excised were definitely present before the globe was opened because they are the only membranes found that could explain the presence of the massive preretinal retraction. The latter condition developed or became worse after the closed vitrectomy. Consequently, the massive preretinal retraction observed upon the patient's admission was produced by an acellular fibrin membrane. It is reasonable to assume that a cellular preretinal membrane was removed at the time of the closed vitrectomy.

It is likely that the preretinal fibrin membrane was formed after the uncomplicated closed vitrectomy, which had been done five weeks before the open-sky procedure. A precipitating factor may have been the patient's young age.

A fibrin membrane may fail to disappear in the days that follow an open-sky vitrectomy, either because the tissues are incapable of degrading it or because it keeps reforming faster than it can be degraded. These are probably the cases in which one observes postoperative persistence and even aggravation of massive preretinal retraction that existed preoperatively, with later appearance of pigment in the preretinal membranes and attraction of the detached retina toward the ciliary processes and iris. Sometimes this process even leads to the formation of an iridoretinal synechia. Anteriorly, changes vary. An iris fibrin membrane may cause ectropion uveae, which may be followed by rubeosis. A fibrin membrane on the posterior iris surface may produce pupillary rigidity, later followed by a cyclitic membrane. Sometimes the iris is attracted into the vitreous cavity by a fibrin membrane and becomes invisible because it covers the ciliary processes.

Post-vitrectomy syndrome—The above

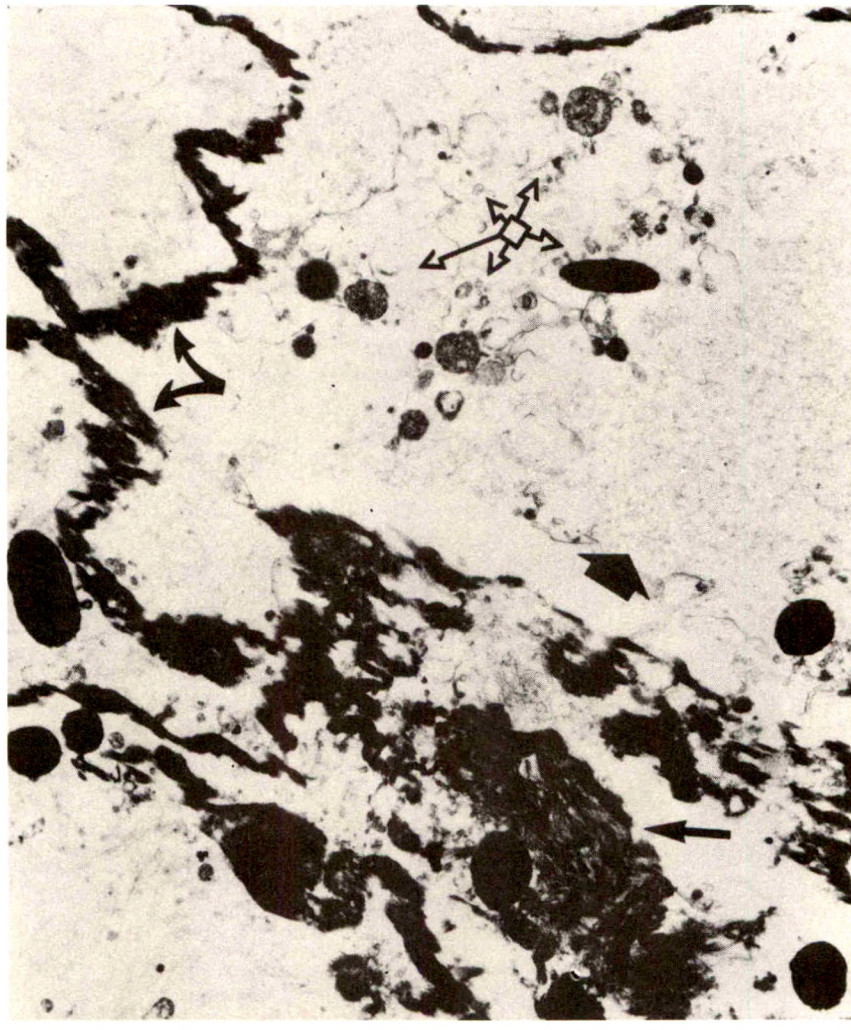


Fig. 22 (Schepens). Electron microscopic section of fibrin membrane. Dense fibrous material (thin solid arrows) is fibrin. Vitrosin (thick arrow) is in clearer areas. Cell debris and free round and oval pigment granules (five arrows starting from square) are interspersed between fibrin and vitrosin (× 20,412).

observations indicate that the intraocular formation of fibrin membranes may result from the surgical trauma of vitreous surgery, either open-sky vitrectomy or closed vitrectomy. The essential cause of the formation of a fibrin membrane is a profound disturbance of the barrier between circulating blood and the intraocular cavity, caused by hypotony and resulting in an outpouring of blood proteins.

When a fibrin membrane is not completely degraded postoperatively, it may be used as a scaffold for the florid growth of easily visible cellular membranes that are often pigmented and may become vascularized. These further changes may result from uncontrolled cellular stimulation.

Finally, it is likely that the absence of vitreous gel facilitates the formation of fibrin membranes and the proliferation



Fig. 23 (Schepens). Top, Fibrin shows fine, smooth filaments (×85,400). Bottom, Vitrosin filaments, showing typical fine periodicity (× 85,400).

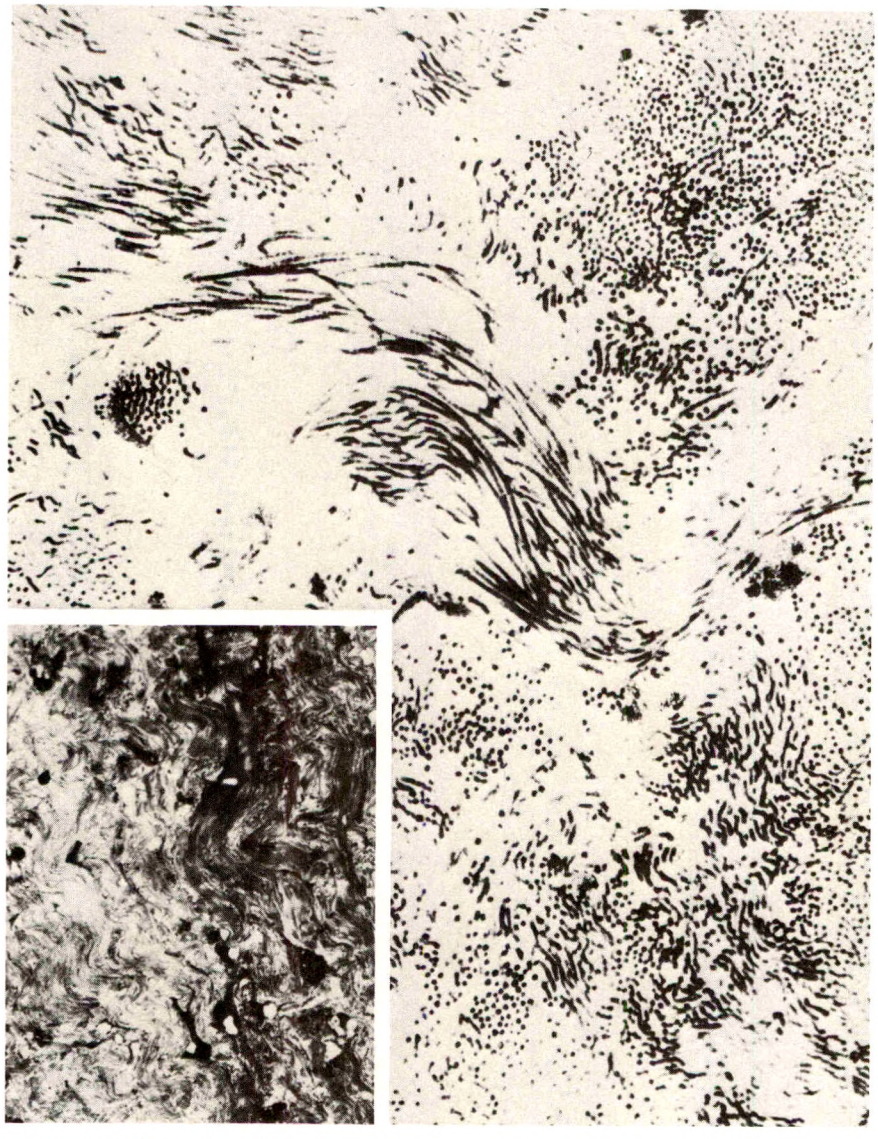


Fig. 24 (Schepens). Adult collagen from preretinal membrane removed by open-sky vitrectomy. Fibers are coarser in diameter and periodicity than vitrosin (× 31,000). Inset, Light microscopic section of same tissue, showing a few fibrocytic cells embedded in dense collagen fibers (× 500).

of cells in these membranes. The vitreous gel has been shown to act as a modulator of intraocular inflammation[8] and cellular proliferation.[27,28]

The three phenomena—disturbance of the blood-intraocular cavity barrier leading to the formation of fibrin membranes, loss of control on intraocular cells causing cellular proliferation over or in the fibrin, and absence of the modulating influence of the vitreous gel—constitute the post-vitrectomy syndrome. In its most severe manifestation the syndrome leads to the formation of extensive and permanent preretinal membranes, which become cellular and shrink, causing a severe and

often incurable retinal detachment with massive preretinal retraction.

Future research—The importance of the discovery of post-vitrectomy fibrin membranes cannot be fully evaluated at this time. Such estimation requires further detailed research in cases of open-sky and closed vitrectomy. The formation of fibrin membranes may be responsible for many or all cases where adequate removal of preretinal membranes is followed by a severe and often incurable recurrence of massive preretinal retraction. If this is proven to be the case, the prevention of formation or the elimination of fibrin membranes may make the treatment of severe massive preretinal retraction by either closed or open-sky vitrectomy considerably more effective.

Since the main element in the pathogenesis of the post-vitrectomy syndrome is severe or prolonged hypotony, similar pathologic changes may possibly occur under other circumstances. For instance, it is worth investigating whether such changes may result from a scleral buckling operation. Marked variations of intraocular pressure nearly always occur in this operation, and cellular stimulation is often very marked as a result of the use of diathermy, cryoapplication, or photocoagulation. The syndrome would be more intense if a vitrectomy had been performed prior to the scleral buckling procedure or in cases where the vitreous is liquefied, as in severe myopia and in Wagner's syndrome. One would expect that an athermal buckle,[29] as first practiced by Zauberman and Rosell,[30] would decrease the incidence and intensity of the syndrome and the risk of later development of massive preretinal retraction.

In the case of accidental globe perforation, the presence of extensive intraocular fibrin membranes may explain why it is difficult to perform a closed vitrectomy safely soon after the accident.

One may wonder why the formation of fibrin membranes has escaped the notice of clinicians for so long. The main reason is that the membranes are clinically invisible until a later stage, when their cellularity and increased thickness give them the typical appearance of preretinal membranes as they appear through the slit-lamp microscope. Additionally, an unsuspecting observer would have trouble identifying fibrin membranes removed by closed vitrectomy when examining the ground material under the microscope.

The degree to which vitreous removal can influence the frequency and intensity of the syndrome deserves further investigation. Another open question is whether a fibrin membrane that is not degraded always becomes cellular. Since a fibrin membrane may serve as a scaffold for cellular growth, one may also wonder whether a cellular membrane that forms before ocular surgery and leads to retinal detachment can sometimes be preceded by the formation of a fibrin membrane. If such a membrane can form spontaneously in the fundus, the presence of an initial severe disturbance of the blood-vitreous barrier should be demonstrable by fluorescein angiography.

The development of postoperative massive preretinal retraction is the main cause of failure in the treatment of retinal detachment. At this time, there are no preventive measures and therapy is still unsatisfactory. I hope that additional research as outlined here will lead to a better understanding of the pathogenesis of postoperative massive preretinal retraction. This, in turn, may help to uncover better methods of prevention and therapy.

SUMMARY

Subtotal open-sky vitrectomy can be used to improve visual function in some eyes for which closed vitrectomy is useless. Open-sky vitrectomy allows easier

access for surgical instruments, especially to anterior fundus structures. The technique also improves the visibility of intraocular structures.

The surgical procedure, which uses special instruments and high-molecular-weight hyaluronic acid, is divided into four stages: preparation, operating inside the vitreous cavity, wound closure, and retinal reattachment.

In 290 consecutive operations for which complete follow-up data are available, retinal reattachment was achieved in 61 eyes (21%). The greatest number of successes (16 of 30 cases [53%]) was achieved in severe tractional retinal detachments. Such results are encouraging because these eyes were considered inoperable by other techniques.

Open-sky vitreous surgery is performed through a corneal incision, but a scleral incision over the pars plana may become the method of choice in selected cases.

The development of a preretinal fibrin membrane, as a result of either a closed vitrectomy or an open-sky procedure, may be a major cause of postoperative failure. Hypotony during surgery is probably the main precipitating factor of intraocular fibrin membrane formation.

ACKNOWLEDGMENT

E. Hultsch, M.D., contributed substantially to the concept of post-vitrectomy syndrome and provided Figures 22 to 24.

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TREATMENT OF MACULAR SUBRETINAL NEOVASCULARIZATION WITH THE RED-LIGHT KRYPTON LASER IN PRESUMED OCULAR HISTOPLASMOSIS SYNDROME

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The presumed ocular histoplasmosis syndrome causes the development of subretinal neovascularization, which in turn leads to visual impairment. The results of medical therapy for this condition have been disappointing. Photocoagulation treatment, therefore, has been the only therapy recommended during the past decade, especially after reports of good results with xenon and argon lasers in the destruction of neovascular membranes.[1-4]

However, Klein, Fine, and Patz,[5] who examined 31 eyes with presumed ocular histoplasmosis syndrome treated by argon laser, showed that the improvement in visual acuity was not statistically significant when compared with the final visual acuities of an untreated group of patients with presumed ocular histoplasmosis syndrome and subretinal neovascularization. Croandijk,[6] who recently described focal macular choroidopathy, a condition with features similar to those of presumed ocular histoplasmosis syndrome, reported unfavorable results with argon-laser treatment in this disease.

Those authors who used the argon laser to treat subretinal neovascularization in presumed ocular histoplasmosis recommended this procedure only if the neovascular membrane was more than 0.25 disk diameters away from the fovea.[4]

Sheffer and associates[7] described the histologic appearance of argon-laser scars on treated subretinal neovascular membranes in presumed ocular histoplasmosis.

We believed one of our patients had macular subretinal neovascularization (caused by presumed ocular histoplasmosis) well suited to treatment with krypton-laser photocoagulation because of the special characteristics of red light.[8]

CASE REPORT

The patient was a 50-year-old woman, who, during the previous 20 years, had lived in various cities in the eastern United States and near the Mississippi river, spending a few months in each place. Visual acuity in her right eye had abruptly deteriorated a few months before we examined her. The patient reported being otherwise healthy.

At ocular examination, visual acuity was R.E.: 6/120 (20/400) and L.E.: 6/6 (20/20). Intraocular pressure was 14 mm Hg by applanation tonometry in both eyes and the media were normal. We found a 1,500-μ, elevated, grayish macular lesion in the fundus of the right eye. It was partially hemorrhagic and partially pigmented, suggestive of subretinal neovascularization, with elevation of the sensory retina above it. The upper part of the hemorrhage was light red; the lower part, which was much darker, extended into the fovea (Fig. 1). We also found another, smaller, subretinal lesion, with elevation of the sensory retina above it, 2 disk diameters beneath and temporal to the fovea. There were no drusen in the macula. There was a round atrophic choroidal scar about 200 μ in size in the area of the upper temporal equator. There were some peripapillary choroidal scars. There were two round atrophic choroidal scars in the periphery of the left fundus. The macula of this eye was normal and there were no drusen.

. Fluorescein angiography of the macular lesion of the right eye demonstrated a choroidal neovascular membrane that leaked profusely during all phases of the angiography (Figs. 2 and 3). The neovascular membrane reached the macula, and the retinal hemorrhage extended beyond the subretinal neovascular membrane. The lower temporal lesion also showed increasing dye leakage during the angiography, and was also suspected of being a subretinal neovascular membrane. We did not perform a skin

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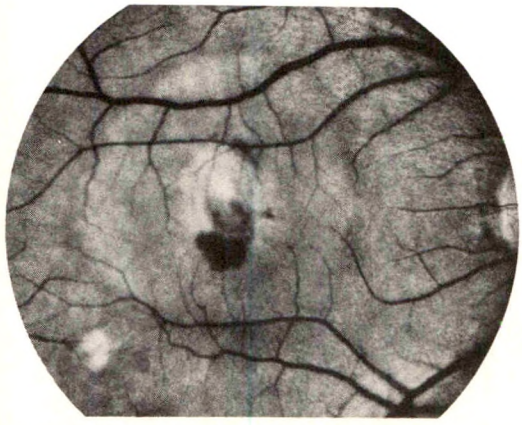


Fig. 1 (Yassur, Gilad, and Ben-Sira). The posterior pole of the right eye shows the macular subretinal neovascular lesion. The retinal hemorrhage covers the fovea. There is a second lesion 2 disk diameters inferotemporal to the macula.

test because presumed ocular histoplasmosis was easily diagnosed.[9]

The patient underwent laser photocoagulation with the continuous red-light krypton laser (647 nm) in order to destroy the two lesions. Laser applications, 50 to 200 μ in size, were done (0.05 to 0.3 seconds of exposure; 50 to 700 mW) until the whole area of identified neovascularization became moderately white (Fig. 4). The treatment included all the subretinal neovascular membrane demonstrated by fluorescein angiography, although it was less than 0.25 disk diameter from the fovea.

Within a few weeks, atrophic chorioretinal scars were observed in the treated area and the hemorrhage regressed (Fig. 5) until it completely disappeared. Six months later, we found that the treated area showed coarse pigmentation and depigmentation and reached the macula, but did not include the fovea, in which the fine pigmentary changes were caused by the long-standing hemorrhage (Fig. 6).

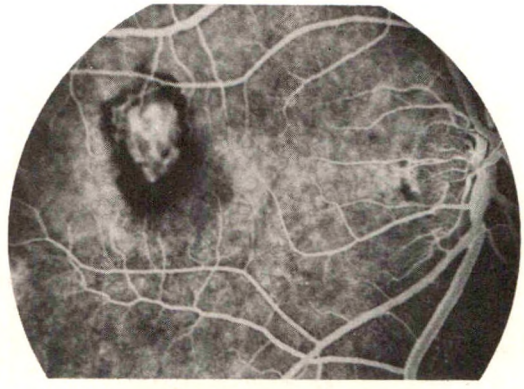


Fig. 2 (Yassur, Gilad, and Ben-Sira). Fluorescein angiography, venous phase, shows early leakage of lesions and staining of the fine peripapillary scars.

Fluorescein angiography demonstrated that the destruction of the neovascular membrane was complete, and that the fluorescein staining was merely that of a scar (Figs. 7 and 8).

Visual acuity six months after the treatment was 6/60 (20/200) and the patient could read J7 with a

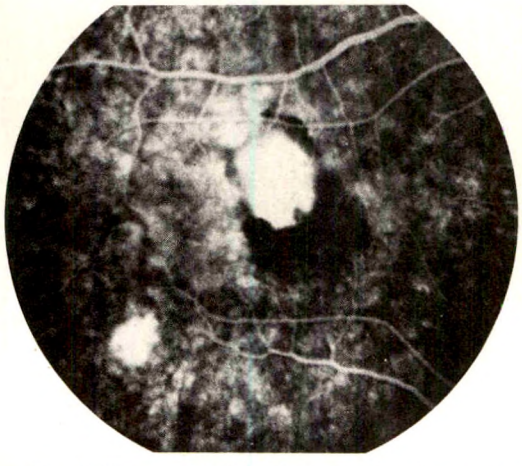


Fig. 3 (Yassur, Gilad, and Ben-Sira). Fluorescein angiography at two minutes, showing late leakage of membrane.

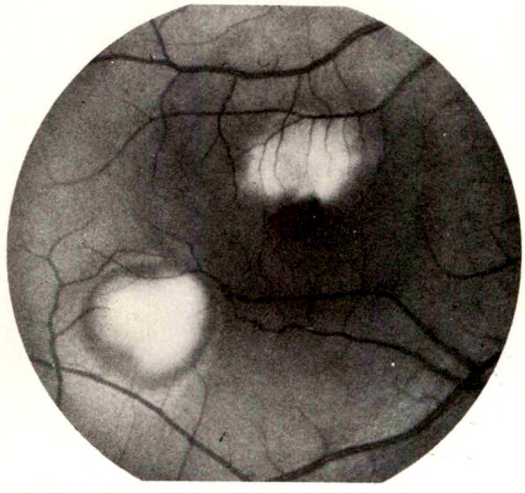


Fig. 4 (Yassur, Gilad, and Ben-Sira). Immediate results of medium-intensity krypton-laser photocoagulation of the macular lesion and strong-intensity treatment of the second lesion, which is farther away from the macula. Note that the krypton laser was applied only to the lighter hemorrhage, and did not extend into the dark foveal hemorrhage.



Fig. 5 (Yassur, Gilad, and Ben-Sira). Six weeks after treatment there are two atrophic chorioretinal lesions. Most of the macular hemorrhage has already disappeared.

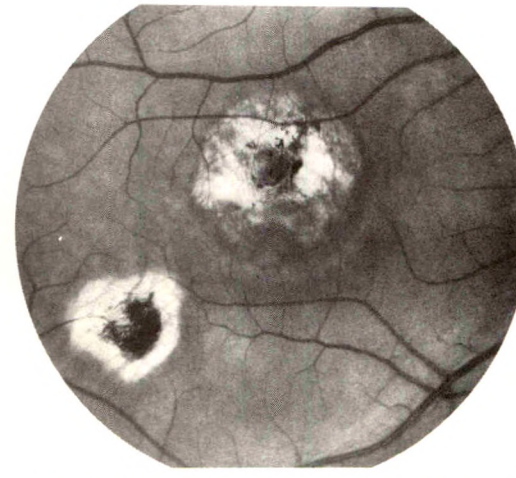


Fig. 6 (Yassur, Gilad, and Ben-Sira). Six months after treatment, there are atrophic coarse scars at the treated area and a fine scar in the foveal area where the hemorrhage existed but was not treated. Note the difference in the macular scar where medium-intensity treatment was used compared with the much coarser lower temporal scar where strong-intensity treatment was applied. The macular hemorrhage was completely absorbed.

low-power lens. The fixation point was below the fovea.

Discussion

Although the patient now lives in Israel, this disease has not yet been reported in this part of the world. Braunstein, Rosen, and Bird[10] described 15 patients in the United Kingdom with the ophthalmoscopic features of histoplasmosis and negative skin tests and Croandijk[6] found focal macular choroidopathy in Europe. However, we believed our patient had presumed ocular histoplasmosis syndrome because she showed the classical triad, as

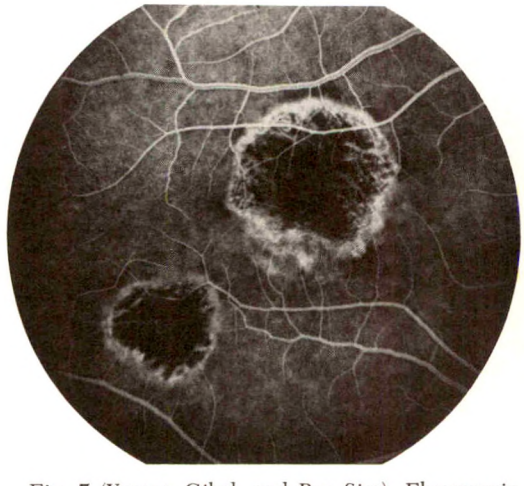


Fig. 7 (Yassur, Gilad, and Ben-Sira). Fluorescein angiography, early venous phase, six months after treatment. There is early scar staining of the treated sites.



Fig. 8 (Yassur, Gilad, and Ben-Sira). Fluorescein angiography at five minutes. There is late scar staining. There is no sign of the subretinal neovascular membrane.

described by Gass[11] and Schlagel and Kenny,[12] and because she had lived for long periods in histo-endemic areas in the United States. Any of these macular diseases would have raised the same question when the subretinal neovascular membrane appeared less than 0.25 disk diameter from the fovea.

The other problem which we faced was an obvious neovascular membrane in the macula, when visual acuity had been considerably reduced for more than a year. The patient probably would have had the same, or a very similar, visual acuity without treatment because the natural course of the disease would have led to macular scar formation. We did not expect photocoagulation to improve visual acuity significantly, and we considered krypton-laser therapy only to observe whether this treatment offered the theoretical properties of monochromatic 647-nm red light.

Xenon- and argon-laser photocoagulation are contraindicated when a macular lesion of any origin is less than 0.25 disk diameter from the fovea and when there is macular retinal hemorrhage, because the polychromatic light of the xenon laser and the blue light of the argon laser are absorbed by the macular xanthophyll and by blood. However, red light penetrates readily through hemoglobin (J. Cohen-Sabban, I. Ben-Sira, Y. Yassur, and C. Maroz, unpublished data), is not absorbed at all by the macular luteal pigment (xanthophyll), and is almost completely transmitted to the choroid.[13] The 647-nm red-light krypton laser is, therefore, very suitable for treating macular subretinal neovascular membranes. As L'Esperance[14] suggested, this laser is less dangerous to the internal retinal layers under which the neovascular membrane is located. The krypton laser also requires less power to destroy a subretinal neovascular membrane than the argon or xenon lasers whose energies are absorbed by intraretinal macular xanthophyll and blood before they reach the pigment epithelium and choroid.

Krypton-laser treatment in our case required less whitening of the retina at the macular lesion and less energy than is usually needed when such a subretinal neovascular membrane is destroyed by argon laser (Fig. 4). The second active lesion, which was farther away from the macula, was treated by strong retinal whitening, like that of an argon laser. Figures 7 and 8 demonstrate, however, that subretinal neovascular membranes of both lesions were eradicated even though less whitening of the retina was achieved. This means that, although less laser power was used, there was enough transmission of the red light into the choroid to destroy the subretinal neovascularization completely. Figure 6 clearly shows that the scar caused by the treatment, although it was within the macula, did not extend beyond the area at which the treatment was aimed, and that the fine pigmentary changes within the fovea were the result of the long-standing hemorrhage and not caused by the krypton-laser treatment.

In view of the anatomical results in this eye, perhaps krypton-laser treatment should be used before visual acuity has deteriorated irreversibly when a macular subretinal neovascular membrane occurs less than 0.25 disk diameter from the fovea.

Summary

A patient with presumed ocular histoplasmosis and decreased visual acuity in one eye had subretinal neovascularization and retinal hemorrhage in the macular area.

Argon-laser treatment to eradicate the subretinal neovascular membrane was contraindicated because of the proximity of the hemorrhagic lesion to the fovea. We used a red-light krypton laser

(647 nm) because this light is not absorbed by the hemoglobin in the retinal hemorrhage or by the macular luteal pigment (xanthophyll), and because it is transmitted into the choroid, allowing treatment within the macular zone with less damage to the internal retinal elements.

After treatment, the subretinal neovascular membrane completely disappeared and the macular hemorrhage cleared.

To the best of our knowledge, this is the first time that a histoplasmic macular subretinal neovascular membrane has been treated with the red-light krypton laser.

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PATHOLOGIC MYOPIA AND CHOROIDAL NEOVASCULARIZATION

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Pathologic myopia involves a progressive elongation of the globe accompanied by degenerative changes of the retina and choroid. This condition is the seventh leading cause of blindness in adults in the United States. It is especially common in the Orient and in the Middle East.[1]

Previous studies have suggested that 5% to 10% of eyes with an axial length of more than 26.5 mm develop choroidal neovascularization.[2,3] This study was designed to add new data on the clinical progression of eyes with pathologic myopia and choroidal neovascularization.

SUBJECTS AND METHODS

We reviewed clinical records, color photographs, and fluorescein angiograms of 81 patients with severe myopia who were examined here between 1970 and 1979. All but three patients had a refractive error of −6 diopters or more; three patients had a refractive error between −5 and −6 diopters with fundus changes typical of pathologic myopia.

Of the 81 patients with severe myopia, 33 had a choroidal neovascular membrane at the posterior pole (Group A). In four of these 33 patients, neovascularization had occurred in both eyes. None of these patients had any signs of other degenerative diseases of the posterior segment. Follow-up information was obtained on 23 of the 33 patients with choroidal neovascular membranes; 17 of these 23 patients were re-examined by us. Follow-up information for the other six patients was obtained from the original referring ophthalmologists. In 16 of the 23 follow-up cases, the fellow eye was also evaluated. All 16 eyes in this "contralateral group" had pathologic myopia but had not developed neovascular membranes (Group B).

From the group of 48 patients with severe myopia who did not have choroidal neovascularization, we selected 11 control patients who lived close to Baltimore, and re-examined them (Group C).

At the initial evaluation, we measured corrected visual acuity. A pinhole was used to improve visual acuity.

In the follow-up evaluations, a manifest refraction was performed in each case by one of us (M. L. H.). After dilating the pupils, we performed direct and indirect ophthalmoscopy and slit-lamp biomicroscopy with the Goldmann contact lens. Stereoscopic color photographs and fluorescein angiograms were obtained for all patients. We noted the following conditions whenever they were present: temporal crescent formation, diffuse or focal chorioretinal atrophy, posterior staphyloma, lacquer cracks, and retinal hemorrhage. In each patient re-examined here, we determined global axial length with an A-scan ultrasonoscope at a wave velocity of 1,545 m/sec. Three separate readings were taken for each globe and the values were averaged. This method is accurate to within 0.2 mm on either side of the averaged reading.[4,5]

RESULTS

Initial examination—Group A included a higher proportion of women than Group C did (Table 1). The difference may be clinically significant in some way not

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TABLE 1

AGE AND SEX OF PATIENTS AND CONTROLS

	Group A	Group C
No. of patients	33	11
Men	13	8
Women	20	3
Age at onset of visual symptoms (yrs)		
Mean	45	42
Range	19 to 73	10 to 64
Under age 50 yrs	21	9

TABLE 2

REFRACTIVE ERROR

Refractive Error	Group A	Group B	Group C
No. of eyes	37	21	22
Mean	−11.27	−12.10	−13.50
Median	−9.00	−12.25	−12.90
Range	−5.50 to −20.50	−5.50 to −20.00	−7.75 to −21.50

evaluated in this study. Patients in both groups first developed visual symptoms at similar ages.

Table 2 shows the mean, median, and range of refractive errors for all three groups. The mean refractive errors were similar for all three groups. However, the median refractive error in Group A was less severe than in Groups B and C.

The pathologic conditions of the three groups are shown in Table 3. Lacquer cracks were more common in Groups A and B. Retinal hemorrhages were found almost exclusively in Group A. The remaining conditions occurred with similar frequency in all the groups.

To analyze visual acuity data, we classified the eyes with neovascularization according to the location of the new vessels with respect to the foveal avascular zone. Using stereoscopic color photographs and fluorescein angiograms, we classified neovascular membranes as being outside the foveal avascular zone, extending to the edge of the foveal avascular zone, or within the foveal avascular zone. In Group A, the neovascular membrane was located inside the foveal avascular zone in 21 eyes, extended to the edge of the foveal avascular zone in six eyes, and was outside the foveal avascular zone in ten eyes.

Follow-up examination for Group A—We obtained follow-up information for 27 of the 37 eyes with neovascular membranes. Twenty-one of these eyes were re-examined by both of us. Referring ophthalmologists supplied clinical information, stereoscopic color photographs, and fluorescein angiograms for the remaining six eyes. The average

TABLE 3

RETINAL FINDINGS IN SEVERE MYOPIA

Finding	Group A	Group B	Group C
No. of eyes	37	21	22
Temporal crescent	20	11	10
Chorioretinal atrophy	28	16	19
Diffuse	23	16	10
Focal	11	4	8
Posterior staphyloma	16	10	8
Lacquer cracks	21	11	8
Retinal hemorrhage			
Foveal	16	1	0
Parafoveal	8	1	0

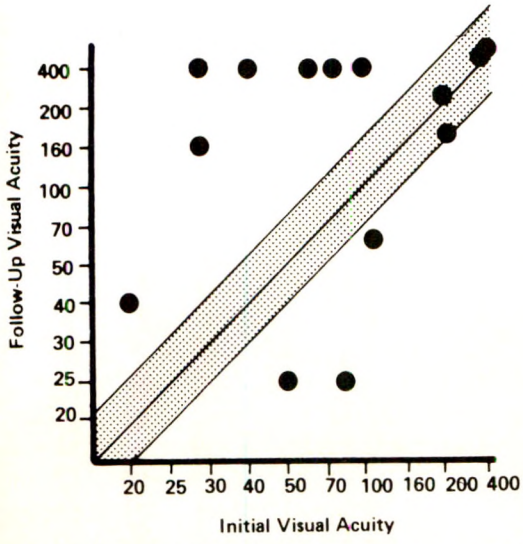


Fig. 1 (Hotchkiss and Fine). Change in visual acuity for the 14 eyes in Group A that had new vessels inside the foveal avascular zone.

follow-up interval was 25.5 months. The mean axial length of the eyes in Group A was 28.05 mm.

NEOVASCULAR MEMBRANES INSIDE THE FOVEAL AVASCULAR ZONE—We obtained follow-up information for 14 of the 21 eyes with neovascular membranes within the foveal avascular zone. These data are summarized in Figure 1. Three of the 14 eyes improved two or more lines on the Snellen chart; visual acuity remained the same in four eyes; and seven eyes lost two or more lines on the Snellen chart.

Although the presence of a new vessel membrane within the foveal avascular zone caused visual loss in most patients, the visual outcome was not uniformly poor. One patient, for example, had a seven-month history of visual loss in the left eye. The left eye had a refractive error of −9.50 diopters, and her visual acuity was 6/24 (20/80). New vessels were present beneath the fovea of the left eye (Fig. 2). Five years later, visual acuity in the left eye had improved to 6/9 (20/30) with no apparent change in the size of the neovascular membrane.

Photocoagulation was not used to treat any eye containing a neovascular membrane within the foveal avascular zone.

NEOVASCULAR MEMBRANES EXTENDING TO THE EDGE OF THE FOVEAL AVASCULAR ZONE—We obtained follow-up information for five of the six eyes that initially had a neovascular membrane extending to the edge of the foveal avascular zone. In all five cases, the new vessels had

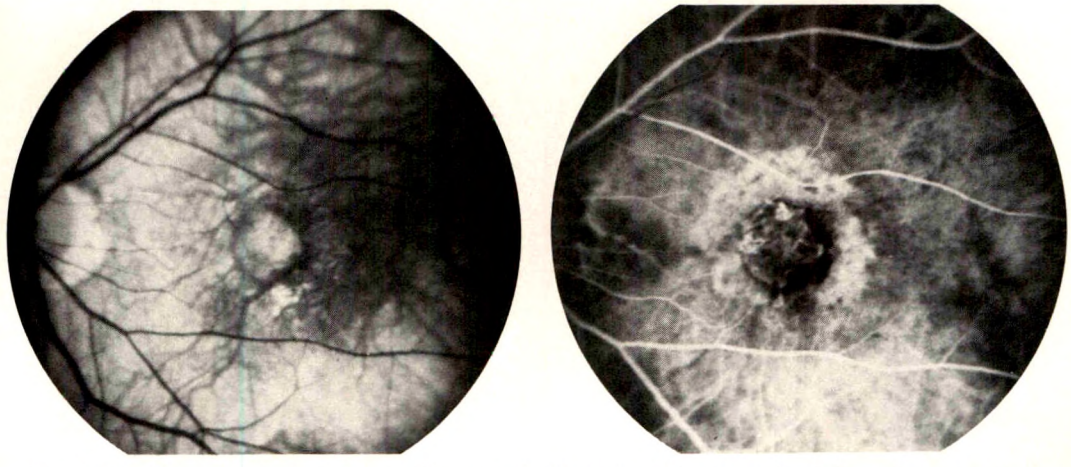


Fig. 2 (Hotchkiss and Fine). A 40-year-old woman had had visual blurring in her left eye for seven months. Visual acuity was 6/24 (20/80). Left, A neovascular membrane was present in the macula. Right, Fluorescein angiography showed the neovascular net beneath the fovea.

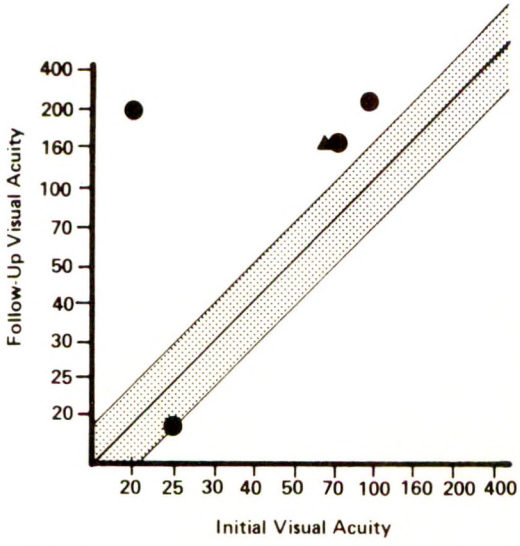


Fig. 3 (Hotchkiss and Fine). Change in visual acuity in the five eyes in Group A that had neovascular membranes extending to the edge of the foveal avascular zone. Triangular figure represents case in which the laser photocoagulation was used to treat the membrane.

advanced to within the avascular zone. Figure 3 summarizes the changes in visual acuity for these eyes. Visual acuity remained the same in one eye; the remaining four eyes lost two or more lines on the Snellen chart. One patient had received argon-laser photocoagulation to the new vessel at the time of initial examination. Pretreatment visual acuity was 6/22.5 (20/75) (Fig. 4, left). One year later, the membrane had advanced inside the avascular zone and visual acuity was 6/48 (20/160) (Fig. 4, right).

NEOVASCULAR MEMBRANES OUTSIDE THE FOVEAL AVASCULAR ZONE—We obtained follow-up information for eight of the ten eyes with neovascular membranes outside the foveal avascular zone. These data are summarized in Figure 5. Visual improvement occurred in only one eye and involved the use of eccentric fixation. Visual acuity remained the same in four eyes and decreased at least two Snellen lines in three eyes.

Five of the eight eyes for which follow-up data were obtained had been treated with argon-laser photocoagulation. In three of the five eyes, an initial visual acuity of 6/6 (20/20) was preserved. One eye retained a visual acuity of 6/60 (20/200). One treated eye progressed from 6/7.5 (20/25) to 6/60 (20/200) during an 18-month period.

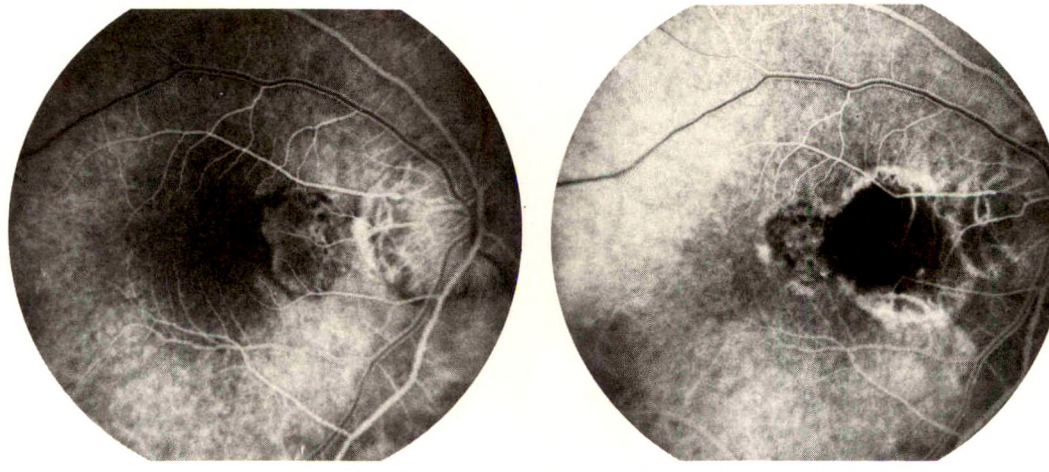


Fig. 4 (Hotchkiss and Fine). A 34-year-old man had a four-week history of decreased visual acuity in his right eye. Visual acuity was 6/22.5 (20/75). Left, A neovascular membrane extending to the edge of the avascular zone was treated with argon-laser photocoagulation. Right, Angiogram taken one year after photocoagulation of the membrane. Despite initially successful treatment, new vessels ultimately grew inside the avascular zone. Visual acuity was 6/48 (20/160).

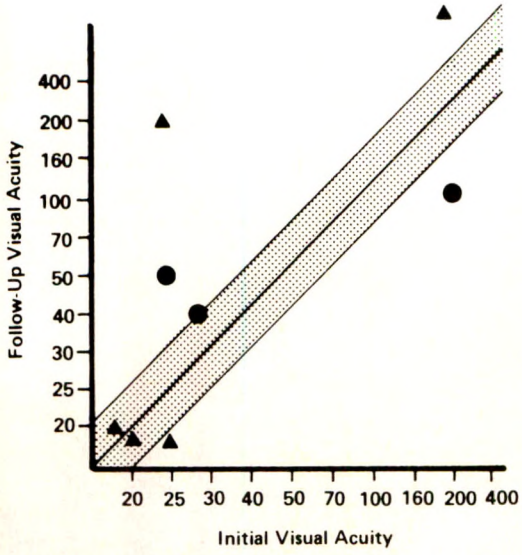


Fig. 5 (Hotchkiss and Fine). Change in visual acuity in the eight eyes in Group A that had new vessels outside of the foveal avascular zone. Triangular figures represent those cases treated with argon-laser photocoagulation.

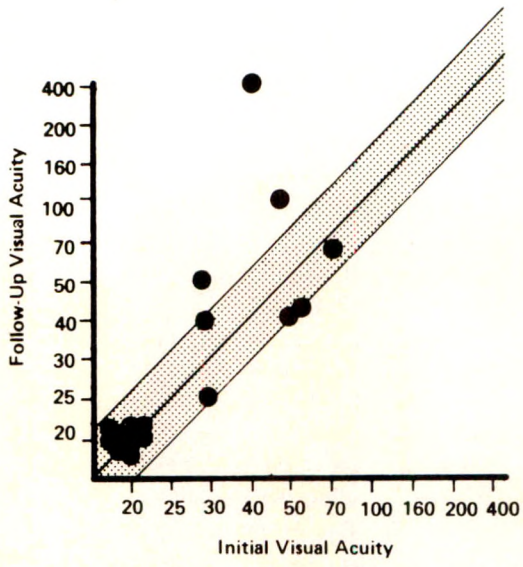


Fig. 6 (Hotchkiss and Fine). Change in visual acuity in the 16 eyes in Group B.

Follow-up examination for Groups B and C—We obtained adequate follow-up information for 16 eyes in Group B. The average axial length was 28.31 mm. Figure 6 summarizes the changes in visual acuity for these eyes. Three eyes lost two or more lines on the Snellen chart. In 13 eyes, initial visual acuity was unchanged.

We obtained follow-up information for 16 eyes in Group C. The average follow-up interval was 31.8 months. Axial length measurements averaged 28.92 mm. Visual acuity data are summarized in Figure 7. Five eyes improved two or more lines on the Snellen chart and visual acuity remained the same in 11 eyes.

Discussion

Choroidal neovascularization has been described in association with a number of conditions, including senile macular degeneration, ocular histoplasmosis, angioid streaks, and traumatic choroidal rupture.[6,7] A break in Bruch's membrane precedes the development of neovascularization in each of these conditions. Histologic changes of a degenerative, calcific, inflammatory, or neoplastic nature are usually present within the cho-

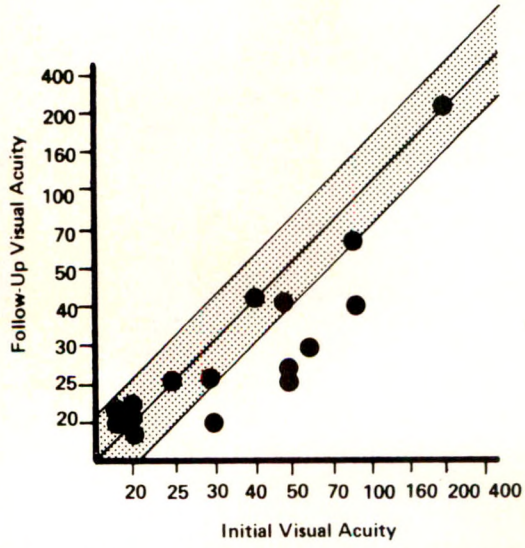


Fig. 7 (Hotchkiss and Fine). Change in visual acuity in the 16 eyes in Group C that were highly myopic but without neovascularization.

roid, adjacent to the defect in Bruch's membrane. Once established, the break in Bruch's membrane permits neovascular proliferation from the choriocapillaris into the subretinal pigment epithelial space.

A similar process has been proposed for severe myopia.[8,9] Fluorescein angiographic studies have shown that choroidal and retinal circulations are slowed in severe myopia. Conceivably, vascular compromise predisposes the eye to choroidal degeneration.[10,11] Continued stretching and degeneration of the choroid causes eyes with severe myopia to develop breaks in Bruch's membrane. Large breaks in Bruch's membrane, called lacquer cracks, allow choroidal neovascular growth. These lacquer cracks were present in 21 of the 37 eyes in Group A. However, in 16 eyes, choroidal neovascularization developed in the absence of lacquer cracks. Microscopic defects in Bruch's membrane might have been present in these cases. The other features of myopic degeneration, including temporal crescent formation, diffuse and focal chorioretinal atrophy, posterior staphylomas, and axial length, did not correlate with the development of choroidal neovascular membranes.

We evaluated eyes in Groups B and C separately to avoid masking a possible genetic predisposition towards choroidal degeneration and neovascularization. Indeed, lacquer cracks were more common in Groups A and B.

In five of the 16 eyes in Group C, visual acuity had increased two or more Snellen lines at the follow-up examination. Because manifest refraction had not been performed at the initial examination, we cannot exclude the possibility that refraction may account for this improvement. Results of fluorescein angiography in these eyes were also unchanged from the initial evaluation.

Summary

In 14 of 27 eyes with choroidal neovascular membranes associated with severe myopia, visual acuity deteriorated two or more lines on the Snellen chart. Twelve of the 27 eyes became legally blind.

Final visual acuity may be related to the position of the neovascular membrane within the posterior pole. Four of the eight eyes with new vessels outside of the foveal avascular zone had follow-up visual acuities of 6/12 (20/40) or better. Only one of five eyes with new vessels extending to the edge of the avascular zone had a final visual acuity of 6/12 (20/40) or better. Three of 14 eyes with a neovascular membrane beneath the foveal avascular zone had visual acuities of 6/12 (20/40) or better.

Neovascularization associated with severe myopia clearly jeopardizes central visual acuity. Laser photocoagulation therapy may be useful in treating neovascular membranes outside the foveal avascular zone or extending to the edge of the perifoveal capillary network. A larger prospective study is needed to determine the guidelines for and efficacy of such treatment for patients with severe myopia who develop choroidal neovascularization.

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Fifty years ago this month in The Journal:

We might say that the myopic eye of such a child is overgrown, and that if we put it at rest by means of atropin for such a period, while the body continues to grow and the child lives an outdoor life, the body can in a measure catch up with the eye. My experience in many such cases is that the myopia remains stationary or improves a little from June to September, and that most myopias increase about three quarters of a diopter during the school year. The special value of continued attention to the earliest stages of myopia in the young child should be repeatedly stated to the mother and teacher, and the pediatrist should aid in this education.

De Wayne Hallett: The Prevention of Myopia
Am. J. Ophthalmol. 14:146, 1931

GLUCOCORTICOSTEROID INHIBITION OF INTRAOCULAR PROLIFERATION AFTER INJURY

YASUO TANO, M.D., DAVID B. CHANDLER, M.D., BROOKS W. MCCUEN, M.D., AND ROBERT MACHEMER, M.D.

Durham, North Carolina

Penetrating and perforating injuries are among the most serious problems that ophthalmologists manage. Both the immediate destruction and the delayed formation of fibrovascular proliferation along the vitreous scaffold[1,2] from the wound can lead to an untreatable condition.

Modern vitrectomy has made it possible to cope with many perforating injuries previously considered untreatable or treated unsuccessfully.[3-5] Opaque media can be removed and future scaffolds for fibrous proliferation can be properly treated. Nonetheless, immediate vitreous surgery is not always possible or desirable. We looked, therefore, for a means of reducing intraocular proliferation until a final decision about vitreous surgery can be made.

Glucocorticosteroids have an inhibitory effect on fibroblast growth.[6,7] Using a reproducible animal model of perforating injuries,[1] we studied the effect of intraocularly injected dexamethasone alcohol on proliferation after perforating injury of the posterior segment.

MATERIAL AND METHODS

We used a total of 23 female pigmented rabbits weighing between 2.5 and 3.5 kg each. We divided the rabbits into two groups: a treated group of 12 rabbits, each of which received a single intravitreal injection of dexamethasone alcohol, and a control group of 11 rabbits, each of which received 0.1 ml of physiologic saline.

Pupils were dilated with 1% atropine sulfate, 10% phenylephrine HCl, and 2% cyclopentolate HCl. We anesthetized the rabbits by slow intravenous injection of sodium pentobarbital, 20 to 30 mg/kg of body weight. We then gave each rabbit a retrobulbar injection of 1 ml of 1% lidocaine. After we performed a 180-degree inferior limbal peritomy, we placed 4-0 silk bridle sutures under the inferior and lateral recti muscles to hold the eye. We inserted the sharp point of a jagged, four-sided injury probe through bare sclera inferotemporally, 7.5 mm posterior to the corneoscleral limbus and just temporal to the vortex vein. Using indirect ophthalmoscopy, we directed the tip across the vitreous cavity and through the posterior wall of the eye 1 disk diameter inferior to the optic disk. We then removed the probe and inserted a 27-gauge needle, attached to a tuberculin syringe with 0.1 ml of dexamethasone alcohol (1 mg) suspended in physiologic saline for the treated group or the same amount of physiologic saline for the control group, inferonasally, 7.5 mm posterior to the corneoscleral limbus and just nasal to the vortex vein. We injected the contents of the syringe slowly and then withdrew the needle. We observed vitreous escaping through the previously created wounds. The fellow eye was treated in the same fashion; it could not be used for control

From the Duke University Eye Center, Duke University Medical Center, Durham, North Carolina. This study was supported by grant EY02903 from the National Institutes of Health and by grants from the Helena Rubinstein Foundation, Research to Prevent Blindness, Inc., Alcon Laboratories, and the David Sime Foundation (Dr. Machemer).

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because we could not exclude systemic corticosteroid effects. The scleral wounds were left untreated. The conjunctiva was repositioned but not sutured to the corneoscleral limbus.

We examined the animals before and immediately after the injection and on days 1, 3, 7, 14, and 28 with slit lamp and ophthalmoscope. Fundus photographs were taken at each examination. The animals were then killed by an intravenous overdose of sodium pentobarbital. We enucleated the globes immediately and placed them in cold phosphate-buffered 5% glutaraldehyde. We removed a corneal button to allow better infiltration of the fixative. After a minimum fixation time of 24 hours, we rinsed the eyes in phosphate-buffered solution and bisected them circumferentially 4.5 mm posterior to the corneoscleral limbus. The calottes were examined under a dissecting microscope and photographs were taken. After embedding the eye in JB-4 plastic, we obtained sections through both the entrance and exit wounds. We stained the sections with paragon in preparation for light microscopic examination. The clinical and gross pathologic findings were evaluated by each of us independently and in a masked fashion. We excluded from the study those eyes with retina torn between the wounds, eyes with endophthalmitis, and eyes with technical complications, such as cataract, making the visualization of the fundus impossible (three eyes in the treated group and two eyes in the untreated group).

We evaluated the proliferative changes as follows: Proliferations into the vitreous along the injury tract were scored on the continuity between entrance and exit wounds (continuous stalk = 2, partial stalk = 1), the thickness of the stalk (thick stalk = 2, thin stalk = 1), and strand opacity (opaque stalk = 2, translucent stalk = 1). We totaled these scores to produce a final score for proliferation along the injury tract. A score for wound sealing was determined by rating the extent of filling of each perforation site. A wound filled with fibrous tissue and having a central mound or stalk received a score of 2. Those wounds that were partially filled (concave) received a score of 1. We totaled the entrance and exit wound scores to produce a final score for wound sealing. Diverging strand formation was scored on size (large strand = 2, small strand = 1), and opacity (opaque strand = 2, translucent strand = 1). We totaled the scores to produce a final score for diverging strands. We analyzed the total scores by one-tailed Wilcoxon test of control score vs treatment score. P value was adjusted for three tests by multiplying the one-tailed P value by three.

Results

Immediately after injury, there was a small amount of hemorrhage at both the entrance and exit wounds on the retina, under the retina, and under the pigment epithelium just adjacent to the wounds. All of the eyes had fine, barely visible transvitreal tracts between the entrance and exit wounds (vitreous condensation[1]). In many cases a fine streak of blood had formed in this transvitreal tract. There was no massive hemorrhage to obscure fundus details. Retinal incarcerations into the wound were common and there were small serous or serosanguinous retinal detachments with slight concentric retinal folds as much as 0.5 disk diameter from the wound edge. The injection of the corticosteroid suspension or physiologic saline caused an increase in intraocular pressure. We observed vitreous incarceration into both the exit and entrance wounds. We found the corticosteroid suspension incarcerated into the exit wound along with the vitreous and occasionally into the entrance wound.

On day 1 the anterior segment showed

a mild inflammatory reaction with fibrin exudate. The vitreous was somewhat hazy and the transvitreal tracts were gray and semi-opaque. All corticosteroid-treated eyes had a large solid and white corticosteroid mass in the vitreous cavity and less inflammatory reaction.

On day 3, as the vitreous haze became clear, both exit and entrance wounds appeared as deep excavations. Transvitreal tracts were seen more easily. Treated eyes retained a corticosteroid mass that was slightly decreased from the original size.

On day 7, inflammatory signs were hardly apparent in either the anterior chamber or the vitreous cavity. Localized retinal detachments had subsided and only occasional slight retinal wrinklings were left. Healing of the wounds was initiated by the formation of a thin gray membrane deep in the excavation, which still allowed the visualization of underlying vessels (15 of 20 eyes in the control group). In the treated group, more than one half of the eyes (14 of 21 eyes) had no gray membrane in their wounds. We sometimes saw fine fibrous strands originating from the wounds and directed toward the midvitreous cavity diverging

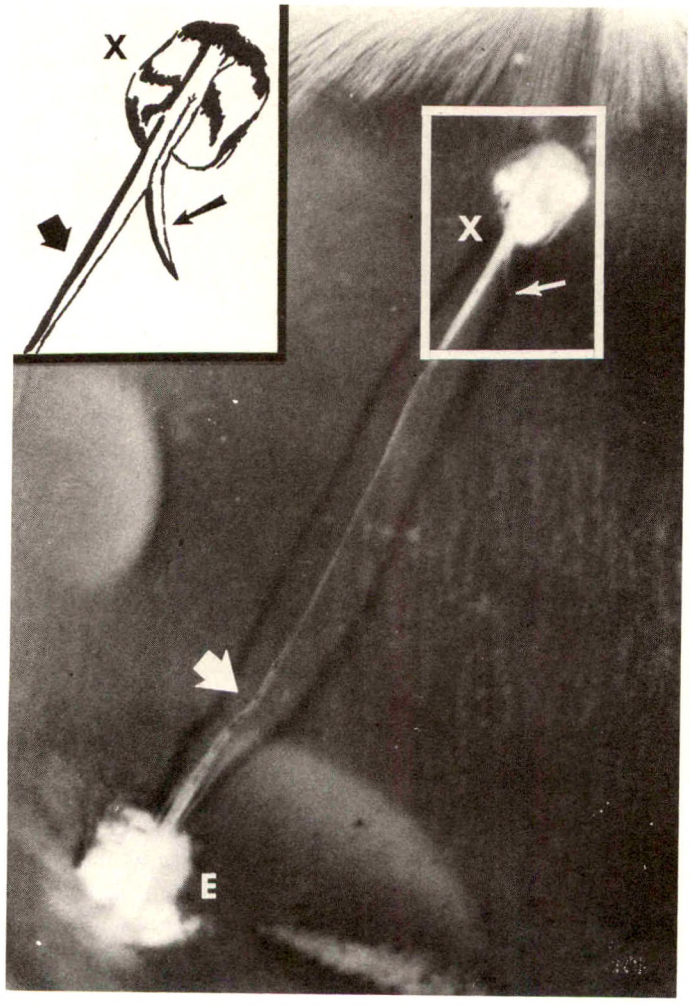


Fig. 1 (Tano and associates). A transvitreal continuous stalk (thick arrow) between entrance (E) and exit (X) wounds is completely filled with fibrous tissue. A small diverging strand (thin arrow) is barely visible at the exit wound. Inset, Schematic drawing of area within rectangle.

from the transvitreal wound tract. These were apparently growing along vitreous structures that had developed during the corticosteroid and saline injections where vitreous escaped through the perforation sites (ten of 20 eyes in the control group and 11 of 21 eyes in the treated group). The corticosteroid masses were now relatively small.

On day 14, fibrous proliferation had advanced. Both exit and entrance wounds were well sealed by gray-white tissue, although they were still concave. Some even had a mound of tissue at their centers. Many eyes showed diverging strands. Some were very thin and almost transparent. Others were thick but translucent. Fibrous proliferation advancing along the transvitreal injury tracts began to appear as tapered stalks (Figs. 1 and 2). The incidence of tapered stalks and strands at the wounds was lower in the treated group (six of 21 eyes) than in the control group (13 of 20 eyes). In both groups, there were now gray membranes at the bottom of entrance and exit wounds, representing scleral healing, in all eyes. Most of them appeared somewhat concave. Only six of 21 eyes in the treated group retained a small mass of corticosteroid.

On day 28, the wounds themselves showed no remarkable changes from the findings of day 14. Diverging strands had grown. Growth of the tapered stalks had advanced in such a way that in some eyes the two opposing stalks met within the transvitreal tract (Figs. 1 and 2). Corticosteroid was no longer visible.

Fibrous proliferation at both wounds was significantly less in the dexamethasone-treated group (mean, 2.29) than in the control group (mean, 3.05) ($P = .006$). Stalk formation along the transvitreal in-

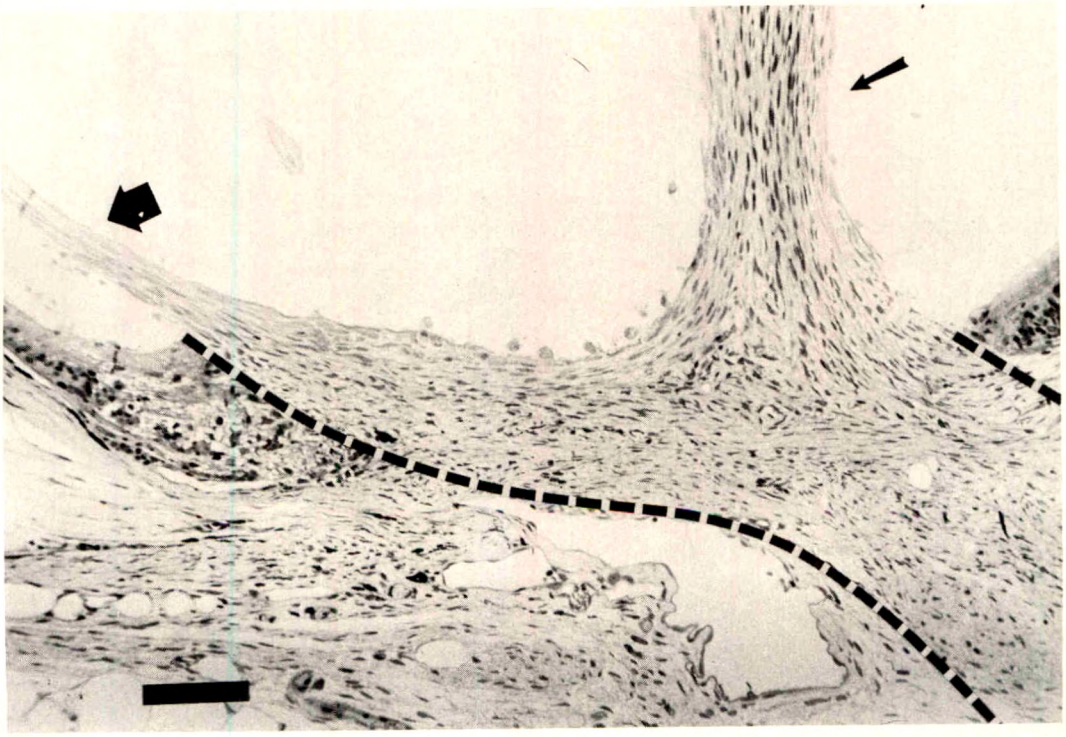


Fig. 2 (Tano and associates). Base of a transvitreal strand (thick arrow) and a diverging strand (thin arrow). The original wound (outlined by dotted lines) is entirely filled with fibrous tissue (bar gauge = 100 μ).

jury tract was less severe in the treated group (mean, 2.19) than in the control group (mean, 3.70) ($P = .021$). Diverging strand formation was not significantly different between the treated group (mean, 1.14) and the control group (mean, 1.35) ($P > .5$).

Discussion

The animal model employed provided highly reproducible intraocular proliferation after perforating injury to the posterior segment of the eye.[1] We measured the effect of dexamethasone alcohol on fibroblast proliferation by the extent of proliferations at both wounds and along the transvitreal injury tract. Eyes treated with dexamethasone alcohol clearly showed delayed and less filling of entrance and exit wounds, and less or no invasion of proliferative tissue into the transvitreal injury tract.

Corticosteroids have an inhibitory effect on fibroblast growth.[7,8] Ophthalmologists are aware of delayed corneal wound healing when the corticosteroid is applied topically.[9] This inhibitory effect on fibroblast growth has also been successfully used to treat keloids.[10] Dexamethasone alcohol also has an inhibitory effect on experimental intraocular proliferation by autologous tissue-cultured fibroblasts.[11]

Because this inhibitory effect is observed only with high corticosteroid levels, we selected intravitreal administration. To have an extended period of corticosteroid action without having to reinject the corticosteroid repeatedly, we selected dexamethasone alcohol or dexamethasone phosphate.[12] Dexamethasone remains in the intact rabbit eye for up to 28 days.[10] Perhaps because of the two large open wounds, dexamethasone disappeared earlier from the vitreous cavity (within 14 days) in this experiment.

The inhibitory effect of corticosteroid on fibroblast growth is attributed to the antimitotic activity in the cells.[13,14] Once the corticosteroid disappears from the eye, the inhibitory effect must decrease as well. The initial inhibition of fibroblast proliferation, however, is accompanied by suppression of inflammatory processes. This seems to affect proliferation because proliferation remains less in the treated eyes even after the disappearance of corticosteroid (14 days). Because our experiment spanned only a four-week period, the possibility of later proliferation could not be excluded.

Corticosteroids may be helpful in the treatment of penetrating and perforating injuries of the eye. High doses of corticosteroids injected intravitreally may help prevent the formation of intraocular scar tissue. In cases of severe injuries where vitreous surgery is needed, corticosteroids may help reduce early proliferation, thus allowing vitreous surgery to be delayed until one to two weeks after the injury.[15]

Summary

Perforating injuries were produced in the posterior segment of rabbit eyes. We studied the inhibitory effect of a slowly water-soluble corticosteroid, dexamethasone alcohol, on fibroblast growth. The treated group received a single intravitreal injection of 1 mg of the corticosteroid. The eyes were observed for four weeks. Transvitreal proliferation was significantly decreased and wound healing was delayed as compared to a control group. Inhibition of intraocular proliferation by high doses of intravitreally injected corticosteroid may be useful in the treatment of eyes with penetrating and perforating injuries.

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FUNDUS FLAVIMACULATUS WITH CYSTOID MACULAR CHANGES AND ABNORMAL STILES-CRAWFORD EFFECT

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Fundus flavimaculatus is usually an inherited autosomal recessive, progressive, dystrophic disease primarily affecting the retinal pigment epithelium with secondary effects on the retinal photoreceptors and choriocapillaris. There is no sex or race predilection. Fundus flavimaculatus and Stargardt's disease are commonly regarded as variants of the same dystrophy. In Stargardt's disease the age at onset is characteristically younger and the visual symptoms and atrophic lesion in the macula are more pronounced than the peripheral surrounding yellow flecks. In the fundus flavimaculatus variant the yellow flecks in the peripheral retina tend to be more prominent earlier during the course of the disease, whereas the macular atrophy occurs later.

Fundus flavimaculatus is characterized by bilateral, symmetrical, yellowish deposits that are irregularly sized, and are round, ovoid, pisiform, semilunar, or linear in shape. They appear in the retinal pigment epithelium, and occasionally have a visible hypopigmentation or hyperpigmentation at their borders. Later, as the disease progresses, there is a change in the macula, especially if yellow flecks have been present, that is indistinguishable from the change seen in Stargardt's disease. This change consists of mottling of the macular retinal pigment epithelium with hypopigmentation and hyperpigmentation, a snail slime or beaten metal appearance, and atrophy of the retinal pigment epithelium and choriocapillaris.[1-6] Less common manifestations associated with the disease include subretinal neovascular membranes, optic disk pallor, attenuated arterioles, bone corpuscular configuration of pigment clumping, extensive atrophy of the retinal pigment epithelium and choriocapillaris in the peripheral and central retina, macular cystoid changes, retinoschisis, and lamellar macular holes.[7-12]

We studied a patient with fundus flavimaculatus, photographically and clinically documenting nonfluorescent cystoid changes in both maculas and abnormalities of the Stiles-Crawford effect.

CASE REPORT

In June 1978, a 13-year-old boy without visual symptoms was found to have fundus flavimaculatus during a routine ocular examination. He was referred for further evaluation. Visual acuity with correction was R.E.: 6/6–4 (20/20–4) with -2.00 $+0.75 \times 100$ and L.E.: 6/7.5–3 (20/25–3) with -2.00 $+0.75 \times 85$. Results of an external examination and slit-lamp examination of the anterior segments were within normal limits. The irides were blue. Intraocular pressures by applanation tonometry were R.E.: 22 mm Hg and L.E.: 20 mm Hg. Direct and indirect ophthalmoscopy showed clear media. The optic disk and retinal vessels were normal. In the fundus midperiphery there were many yellow deposits showing early to late resorption of the more opaque yellow material with increasing translucency at the level of the retinal pigment epithelium. They were associated with some foci of hyperpigmentation (Fig. 1). We found many clear microcystoid spaces, verified by Goldmann biomicroscopic examination, in both maculas (Figs. 2 and 3).

Results of ocular examinations of the patient's only available blood relatives, a maternal aunt and the maternal grandmother, were within normal limits. There was no definite family history of a similar disease or loss of vision.

A fluorescein angiogram confirmed the disruption

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Fig. 1 (Morse and associates). Midperiphery of the right eye. Yellow flecks appear as lighter dots with other areas looking atrophied with minor specks of pigmentation.

of the integrity of the retinal pigment epithelium, or lack of uniform melanin granule distribution, after resorption of the yellow retinal deposits which showed many areas of increased transmission or hyperfluorescence. The retinal pigment epithelium, however, was intact centrally and beneath the foveolas (Fig. 4).

Retinal function testing showed dark adaptation to be essentially within normal limits. The electro-oculographic ratio was reduced to R.E.: 1.25 and L.E.: 1.20 (normal, 1.90). The electroretinogram was subnormal on photopic testing, with a normal implicit time, and subnormal on scotopic testing with an increased implicit time.

We tested color vision with the Nagel anomaloscope, using the testing procedure suggested by Linksz.[13] We administered the Ishihara screening plate and the Farnsworth-Munsell 100-hue tests under a Macbeth easel lamp according to the instructions accompanying the tests. The patient failed the Ishihara plates. The Rayleigh equation, measured with the Nagel anomaloscope, showed that the match midpoint was within normal limits. However, there was a wide matching range. Results of the Farnsworth-Munsell 100-hue test included a number of errors (R.E.: 113 errors and L.E.: 181 errors) but were within normal limits, showing no definite axis and poor patient attention.

Smith and associates[14,15] have described the design of the apparatus, calibrations, and clinical use of the Stiles-Crawford equipment. We measured the Stiles-Crawford effect by a brightness matching procedure using a circular 55-minute central target embedded in a 3-degree 38-minute annulus. Both fields were of 620-nm wavelength light.

The annulus provided a field luminance of 130 cd/m^2 centered on the optic axis of the patient's pupil. The central circle was illuminated by a beam that could be shifted along the horizontal meridian of the patient's pupil. We used ophthalmic lenses to maintain the appropriate dioptric correction for the movable beam of light and a tele-

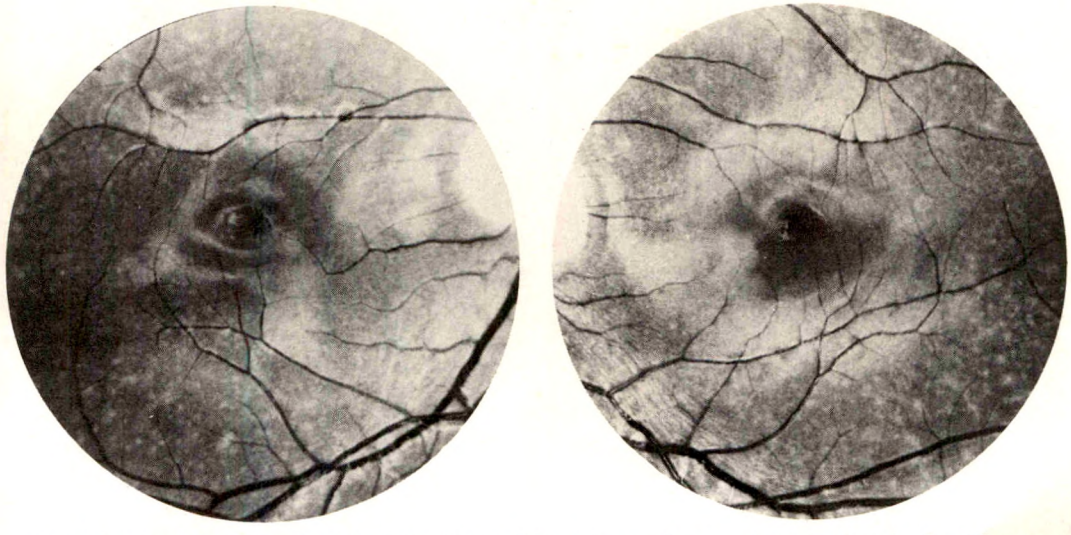


Fig. 2 (Morse and associates). Left, Central area of the right eye shows an irregular foveal reflex. There are also surrounding flecks of yellow deposits and small areas of retinal pigment epithelial atrophy in the posterior pole. Right, Left eye shows a similar irregularity of the foveal reflex with many areas of yellow deposits and disruption of the retinal pigment epithelium in the posterior pole.

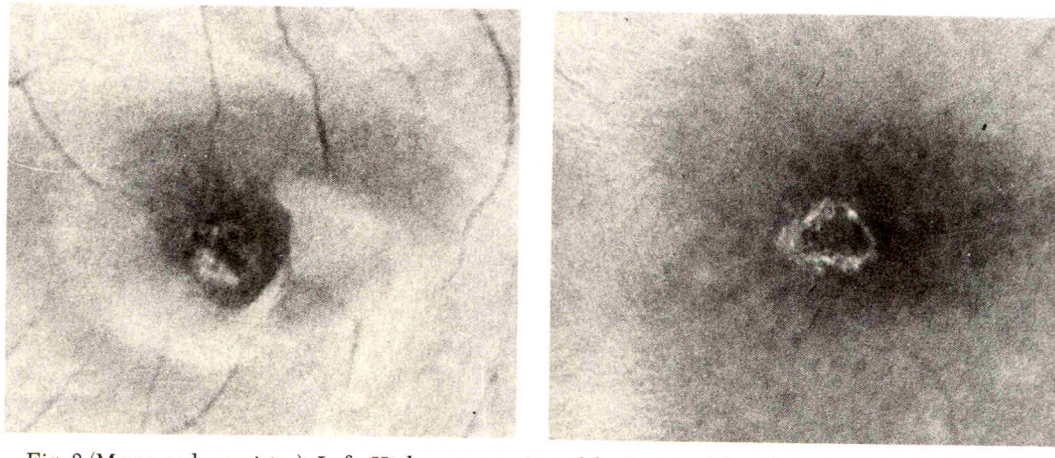


Fig. 3 (Morse and associates). Left, Higher-power view of the fovea and foveolar area in the right eye shows many microcystoid changes which appear as darker areas centrally. The light reflex accounts for the lighter coloration near the microcystoid spaces. Right, Higher-power view of the foveal and foveolar area in the left eye shows an even more striking number of microcystoid spaces which appear as darker round-to-oval areas centrally. The light reflex appears as an irregularly shaped oval centrally.

scopic viewing device to align and monitor the position of the patient's eye during the course of evaluation. The patient made three brightness matches at each pupil entry position. Data are reported as log eta (η) where eta is the relative efficiency of the central movable light beam for various pupillary entry positions. The Stiles-Crawford effect functions did not show the normal parabolic shaped peaking near the zero entry position.[16] In the right eye the function was broad with a suggestion of a peak near the nasal edge of the pupil. The left eye function was essentially flat (Figs. 5 and 6).

Discussion

There are four general distribution patterns of manifestations in patients with fundus flavimaculatus: (1) Atrophic macular degeneration without flecks; (2) macular degeneration with perifoveal flecks (Stargardt's disease); (3) macular degeneration with diffuse flecks (fundus flavimaculatus with macular degeneration); and (4) diffuse flecks without macular degeneration.[1] A similar classification by Fishman[3] relies more on the progressive nature and later atrophic stages of the disease.

The differential diagnosis of the macular degeneration, in the absence of systemic or central nervous system disease, involves central areolar choroidal dystrophy, vitelliruptive macular degeneration, progressive cone dystrophies, chloroquine ingestion, and the later changes seen in X-chromosome-linked congenital retinoschisis.

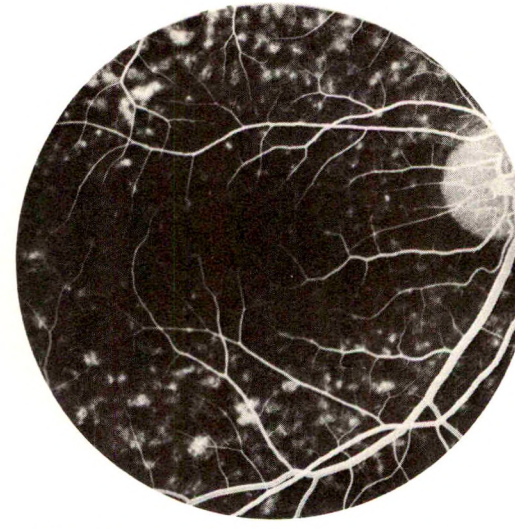


Fig. 4 (Morse and associates). Fluorescein angiogram of the right eye demonstrates hyperfluorescence and increased transmission characteristic of fundus flavimaculatus. Retinal pigment epithelium beneath the foveola is intact. A fluorescein angiogram of the left eye was similar.

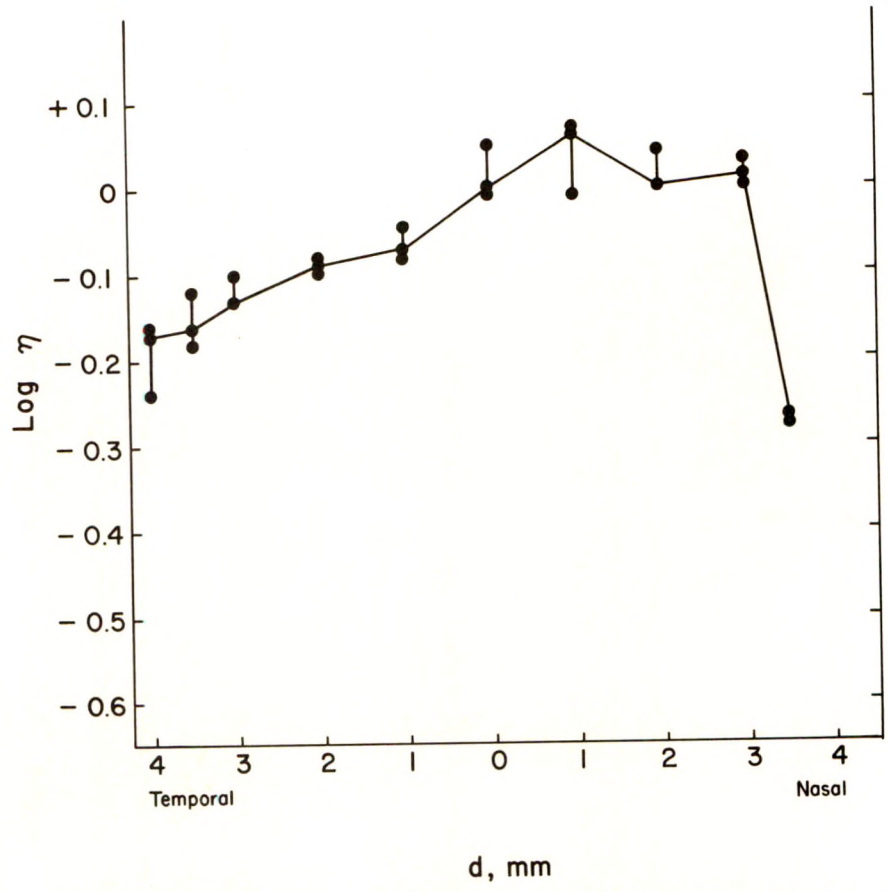


Fig. 5 (Morse and associates). Stiles-Crawford testing of the right eye discloses lack of the usual parabolic curve.

The yellow-white retinal flecks or areas of chorioretinal atrophy must be distinguished from those seen in fundus albipunctatus, retinitis punctata albescens, drusen, multiple vitelliruptive deposits, flecked retina syndrome of Kandori, healed disseminated choroiditis, and angioid streaks with more widespread retinal disease such as colloid excrescences and pigmentary change.

The reported results of psychophysical and electrophysiologic testing in patients with fundus flavimaculatus vary considerably. Generally, most patients have normal photopic and scotopic ERGs. Some patients show a slight reduction or prolonged time in reaching final photopic amplitudes. Dark adaptation is usually normal, although prolonged cone-rod breaks and prolonged periods required to reach the final thresholds have been noted. The EOG is usually normal but may be subnormal. Peripheral visual fields are usually within normal limits but may be constricted or show a central scotoma in patients with markedly impaired central visual acuity. Color vision testing is not a reliable diagnostic tool in early cases and normal values have been obtained in patients with a visual acuity of 6/60 (20/200). Some patients have a mild red-green defect or nondiagnostic color confusion.[1] Many variables must be considered when interpreting the tests;

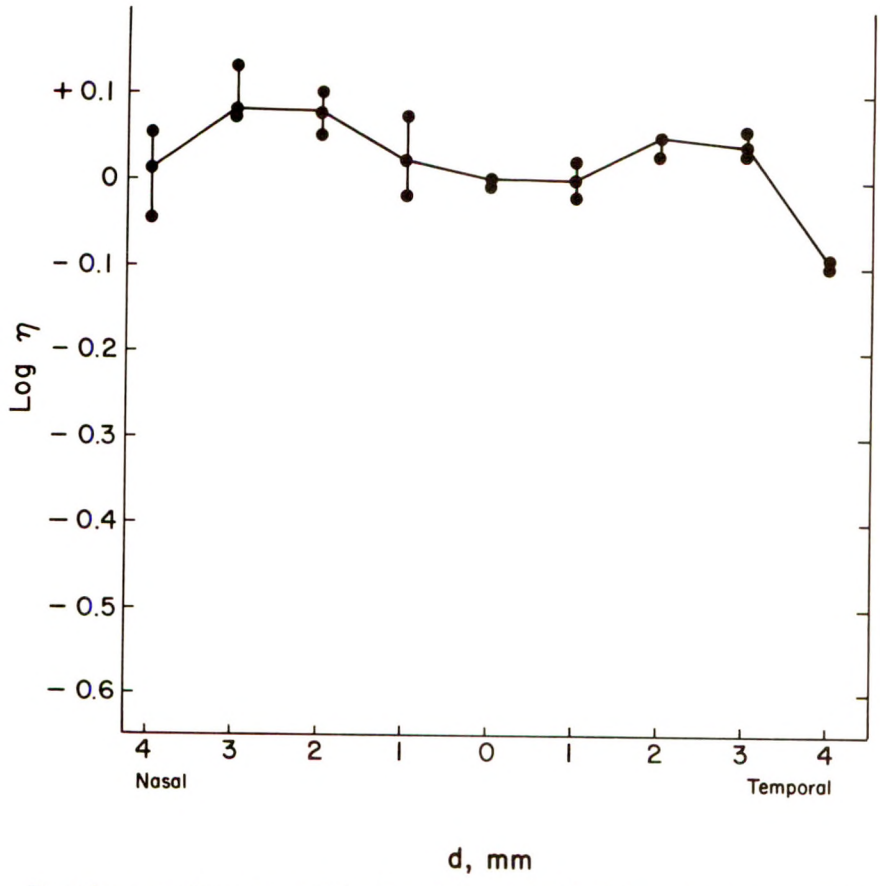


Fig. 6 (Morse and associates). Stiles-Crawford testing of the left eye shows an essentially flat determination.

among them are the patient's cooperation and comprehension, differences in equipment, standardization of stimuli applied, meticulousness of the technician performing the tests, and, of course, the extent or degree of progression of the disease. As Fishman[3] pointed out, the more severely afflicted patients are more likely to show detectable abnormalities on psychophysical and electrophysiologic testing.

Some patients described in published reports as having fundus flavimaculatus or Stargardt's disease may actually have had some other retinal disorder.

Our patient had unimpaired central visual acuity. Rayleigh match midpoints were within the normal range. Chromatic discriminative ability was partially reduced. Stiles-Crawford effect functions measured at the fovea, using a 55-minute target, were abnormal in both eyes, probably as a result of disorientation of the retinal photoreceptors. In diseases that cause severe disorientation of foveal photoreceptors, the foveal Rayleigh matches are also abnormal.[17] Therefore, we assumed that the ophthalmoscopically observed cystoid changes in the patient's maculas caused disorientation of the foveal retinal photoreceptors with orientation toward all regions of the pupil. In comparison, foveal photoreceptors are oriented to the center of the pupil in young,

normal eyes.[17] An alternative explanation is that morphologic changes in the cones might affect their directional sensitivity without modifying the optical density of the visual photopigment and hence the Rayleigh match.

There are only so many possible responses of ocular tissue to any disease process or injury. The slowly progressive nature of a disease may account for the variable ophthalmoscopic manifestations seen among members of the same family. A classification of the stages of a disease may be helpful only for a finite period of time as the appearance or location of the findings may change from examination to examination. Therefore, the extent of the disease should be documented by clinical notes and photography.

The fluorescein angiographic findings of increased transmission or hyperfluorescence also depend upon the amount of disruption of the melanin granules or the integrity of the retinal pigment epithelium. When the yellow deposits lie within the retinal pigment epithelial cell and have not yet disorganized melanin distribution or the cell, they appear hypofluorescent. A breakdown and resorption of the yellow deposits in the retinal pigment epithelium leads to a disruption or atrophy of the pigment epithelial cells with a varying amount of hypopigmentation and hyperpigmentation. If the disruption of the retinal pigmented epithelium occurs beneath the foveola, the patient will lose central visual acuity.

Any injury damaging the retinal pigment epithelium will also damage the photoreceptors and, possibly, the choriocapillaris. The choriocapillaris, Bruch's membrane, retinal pigment epithelium, and photoreceptors are metabolically, functionally, and anatomically interdependent. Any deleterious process involving one layer will almost certainly have some effect on the others.

Many diseases produce ophthalmoscopically visible fundus lesions which appear indistinguishable from one another and which are caused by the same tissue response to different injuries. For example, cotton-wool patches, histopathologic cytoid bodies, are present in many diseases with or without hypertension. Similarly, diseases or degenerations of the retinal pigment epithelium commonly appear as yellow deposits within the retinal pigment epithelium. Examples are vitelliruptive macular degeneration, deposits seen in senile macular degeneration, often mistaken for drusen although drusen may coexist, round yellow deposits seen in macular holes, and diseases whose causes are still unknown. These yellow deposits may be the result of the deposit of a substance foreign to the retinal pigment epithelium, an abnormal accumulation of metabolic or breakdown products that are not being adequately removed from the retinal pigment epithelium, or unknown mechanisms involving retinal pigment epithelial cell incompetence or death.

Summary

A 13-year-old boy with fundus flavimaculatus had a macular disease consisting of many transparent cystoid spaces which were not hyperfluorescent on fluorescein angiography. The Stiles-Crawford effect was abnormal, suggesting that the cystoid changes in the maculas created a disorientation of the patient's foveal retinal photoreceptors.

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OPHTHALMIC MINIATURE

. . . we are very thankful for light which enables us to enter on our way, to practice arts, to read, to distinguish each other, and yet sight is more excellent and beautiful than the various uses of light . . .

Sir Francis Bacon, *Novum Organum 1st Book*, 1620

MONOCULAR AXIAL MYOPIA ASSOCIATED WITH NEONATAL EYELID CLOSURE IN HUMAN INFANTS

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The refractive status of the human eye is determined by a number of factors, the most important of which are axial length, corneal curvature, and the dioptric power and position of the crystalline lens. Although the cause and pathogenesis of human myopia are not known, several investigators have reported that a significant degree of axial myopia follows both monocular and binocular eyelid fusion in neonatal animals.[1-6] Opacification of the cornea has been reported to have a similar, although less marked, effect on axial growth of the eye.[7]

These experimental studies suggest that early attenuation of pattern vision may be a relevant factor in the genesis of axial myopia. However, this thesis has been challenged by the work of von Noorden and Crawford[8] who were unable to demonstrate a consistent relationship between neonatal eyelid closure and axial myopia in monkeys studied in their laboratory. We report clinical and ultrasonic data for eight infants who developed monocular axial myopia associated with prolonged eyelid closure in early infancy.

SUBJECTS AND METHODS

Eight infants were examined either at the University of California, San Francisco, or the University of Sydney between February 1978 and August 1980. All the infants had unilateral axial myopia associated with eyelid closure in early infancy. These eight infants were selected from 64 patients with neonatal eyelid closure examined during that period. At the time of examination the patients ranged in age from 7 weeks to 19 months. A cycloplegic retinoscopic examination was completed after two drops of 1% cyclopentolate HCl had been instilled in both conjunctival sacs. All infants underwent an examination while anesthetized with ethrane or halothane. We inspected both eyes with a Zeiss operating microscope and used a Javal-Schiøtz keratometer to prove that the refractive errors were not corneal in origin. We performed A-scan ultrasonographic biometry with a transducer frequency of 8 MHz. We calculated the axial length of the ocular structures by the technique described by Jansson.[9]

RESULTS

In all cases unilateral axial myopia was associated with prolonged eyelid closure in infancy (Table 1). The eyelid occluded the visual axis of the involved eye in all eight infants. The causes of eyelid closure in these patients included congenital third nerve palsy, blepharoptosis from eyelid hemangioma, blepharoptosis from plexiform neuroma, and soft tissue swelling of the periorbital structures from obstetric trauma. The measured myopia ranged from −4.25 diopters to −7.00 diopters. In no case was there evidence of corneal damage that could have account-

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TABLE 1

CLINICAL DATA FOR INFANTS WITH AXIAL MYOPIA ASSOCIATED WITH NEONATAL EYELID CLOSURE

Case No.	Sex	Involved Eye	Nature of Closure	Duration of Closure	Refractive Error
1	M	R.E.	Congenital third nerve palsy	18 mos	R.E. −4.50 +.75 × 080 L.E. +.75 sphere
2	F	L.E.	Soft tissue swelling secondary to obstetric trauma	7 wks	R.E. +1.50 +.50 × 090 L.E. −4.50 +.25 × 180
3	F	L.E.	Soft tissue swelling secondary to obstetric trauma	11 wks	R.E. +.50 sphere L.E. −5.75 +1.25 × 070
4	M	L.E.	Blepharoptosis from eyelid hemangioma	13 mos	R.E. +1.25 sphere L.E. −4.75 + .50 × 010
5	F	R.E.	Soft tissue swelling secondary to obstetric trauma	10 wks	R.E. −6.50 × .50 × 090 L.E. +1.00 sphere
6	M	L.E.	Blepharoptosis from plexiform neuroma	19 mos	R.E. −.50 +1.00 × 075 L.E. −4.75 +.50 × 080
7	M	R.E.	Soft tissue swelling secondary to obstetric trauma	9 wks	R.E. −4.25 sphere L.E. Plano + .50 × 090
8	M	R.E.	Congenital third nerve palsy	14 mos	R.E. −7.00 +.50 × 090 L.E. −.50 sphere

ed for the myopia. Furthermore, keratometry disclosed that the corneal power was +54.1 ±0.7 diopters in both eyes with no significant asymmetry between the emmetropic and myopic eye. These data agree well with those from previous studies of infants.[10]

Ultrasonographic biometric measurements showed that there was no significant difference in the anterior segment depth or lens thickness between the emmetropic and myopic eyes (Table 2). In contrast, the vitreous length in the myopic eye was greater than that in the emmetropic eye in all cases. The total difference in axial length between the myopic and emmetropic eye ranged from 1.6 to 2.8 mm.

DISCUSSION

Experimental and clinical myopic conditions are a disturbance of the postnatal growth of the eye and concomitant development of the refractive state. A significant degree of axial myopia has been reported to result from monocular or binocular surgical eyelid fusion in neonatal *Macaca* monkeys.[1-3] Postmortem examination of the eyes of these monkeys disclosed marked axial elongation with thinning of the posterior sclera. The corneal curvature did not appear to be affected by eyelid occlusion.[1,2]

The mechanism by which eyelid fusion leads to axial myopia is not clear. Perhaps myopia is generated by thermal or mechanical factors associated with eyelid fusion. This thesis has been countered by the observation that eyelid fusion has no effect on the refractive state and axial length of the eyes in monkeys raised in the dark.[4] Myopia does develop and the eye elongates when these same animals are transferred to illuminated quarters.[4] The crucial factor in the pathogenesis of this experimental myopia may be the distorted representation of the visual world that the fused eyelids present to the visual system. Supporting evidence for the importance of anomalous visual input in the production of experimental myopia is provided by the observation that axial myopia also occurs after latex opacification of the corneal stroma of the eyes of *Macaca* monkeys.[7] The myopia after corneal opacification was less severe than that resulting from eyelid fusion.

Eyelid-suture myopia can also be gen-

TABLE 2

ULTRASONOGRAPHIC DATA FOR INFANTS WITH AXIAL MYOPIA ASSOCIATED WITH NEONATAL EYELID CLOSURE

Case No.	Anterior Segment Depth (mm)	Lens Thickness (mm)	Vitreous Length (mm)	Total Axial Length (mm)	Difference In Axial Length Between Two Eyes (mm)
1	R.E. 2.50	R.E. 3.5	R.E. 12.7	R.E. 18.1	1.6
	L.E. 2.50	L.E. 3.5	L.E. 10.5	L.E. 16.5	
2	R.E. 2.60	R.E. 3.9	R.E. 10.4	R.E. 16.9	1.7
	L.E. 2.60	L.E. 3.9	L.E. 12.1	L.E. 18.6	
3	R.E. 2.40	R.E. 3.8	R.E. 10.1	R.E. 16.3	2.1
	L.E. 2.40	L.E. 3.8	L.E. 12.2	L.E. 18.4	
4	R.E. 2.60	R.E. 3.6	R.E. 10.9	R.E. 17.1	1.6
	L.E. 2.60	L.E. 3.7	L.E. 12.4	L.E. 18.7	
5	R.E. 2.60	R.E. 3.6	R.E. 12.6	R.E. 18.8	2.5
	L.E. 2.60	L.E. 3.5	L.E. 10.2	L.E. 16.3	
6	R.E. 2.50	R.E. 3.6	R.E. 10.4	R.E. 16.5	2.0
	L.E. 2.50	L.E. 3.7	L.E. 12.3	L.E. 18.5	
7	R.E. 2.60	R.E. 3.6	R.E. 12.6	R.E. 18.8	2.6
	L.E. 2.60	L.E. 3.5	L.E. 10.1	L.E. 16.2	
8	R.E. 2.60	R.E. 3.7	R.E. 13.1	R.E. 19.4	2.8
	L.E. 2.60	L.E. 3.7	L.E. 10.3	L.E. 16.6	

erated in the tree shrew,[3] chicks,[6] and, perhaps, cats.[11-13] Significant myopia occurs in the neonatal tree shrew after eyelid closure.[3] However, although these eyes do demonstrate axial elongation, the elongation is not sufficient to account for the magnitude of induced myopia observed.[3] In a later study of myopia after eyelid occlusion in neonatal tree shrews, McKana and Cassagrande[14] demonstrated that ciliary zonular dysplasia and lens hypoplasia occur. These two observations suggest that eyelid-suture myopia in neonatal tree shrews results from excessive accommodation during the period of visual deprivation. In contrast, myopia produced by eyelid fusion in chicks is entirely axial in nature without any evidence that excessive accommodation plays any significant role.[6] Furthermore, eyelid fusion is not needed to produce the axial elongation because chicks reared with translucent occluders develop a comparable degree of myopia.[6]

Eyelid fusion studies in developing kittens have not yielded consistent findings.[11-13] Gollender, Erickson, and Thorn[12] did not report any significant myopia in kittens after eyelid fusion. Wilson and Sherman[11] observed only a minimal amount of myopia in their kittens. Although Kirby and Weiss[13] found significant axial elongation in kittens with sutured eyelids, decreased corneal curvature compensated for the elongation. Therefore, the refractive error in their animals was either hyperopic or emmetropic.

Our data suggest that unilateral axial myopia may occur in the human infant who has had significant eyelid occlusion in the neonatal period. In a recent study correlating anisometropia and congenital blepharoptosis, Merriam, Ellis, and Helveston[15] found that, although most of their patients were hyperopic, Patients 5 and 42 had significantly more myopia in the eye with blepharoptosis. Robb[16] reported an association between unilateral myopia and some hemangiomas of the eyelid and orbit.

We have not observed comparable axial myopia in any of our patients with congenital cataracts, vitreous hemorrhage, or corneal opacification. In light of this, we think that axial myopia in infants with

eyelid occlusion may result from mechanical or thermal effects rather than distortion of visual input. Abnormal ocular growth may have occurred because the intraocular pressure or orbital temperature increased as a consequence of eyelid occlusion. Whatever the mechanism, it seems that the development of axial myopia and subsequent anisometropic amblyopia may be associated with eyelid occlusion in the neonatal period. Therefore, early correction of the underlying cause of eyelid occlusion is indicated whenever possible.

Summary

We studied eight infants who developed unilateral axial myopia associated with neonatal eyelid closure. Clinical findings and ultrasonographic biometric measurements suggested that axial elongation of the eye may occur as a result of neonatal eyelid closure, a finding similar to that for experimental myopia produced in neonatal animals after eyelid fusion. Early treatment of eyelid occlusion in the neonate may prevent axial myopia and subsequent anisometropic amblyopia.

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RETINAL HEMORRHAGE CAUSED BY OVERDOSE OF METHAQUALONE (QUAALUDE)

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Methaqualone (Quaalude) is a common, frequently abused sedative hypnotic agent, often used in successful suicide attempts. I studied a patient with retinal hemorrhages presumably resulting from a methaqualone overdose during an unsuccessful suicide attempt.

CASE REPORT

On Aug. 20, 1979, a 23-year-old unconscious man was found in his home with his 20-year-old dead girl friend. They had made a suicide pact. Also found in the room were a recently dated empty bottle of 150-mg methaqualone (Quaalude) tablets, labeled to contain 50 pills, and two empty 750-ml vodka bottles.

On arrival at the emergency room the patient was unconscious and unresponsive to deep pain, but did breathe without assistance. He had purpura across his back; there was no evidence of facial or chest trauma. There were no conjunctival lesions. The corneas were dry and irregular. An ophthalmoscopic examination disclosed a retinal hemorrhage in the right eye. The pupils were small but reactive to light. The patient was admitted to the respiratory intensive-care unit. Medical history, family history, and systemic review were all noncontributory. The patient had no history of diabetes mellitus or hypertension. The patient later claimed that he had not taken methaqualone previously. His physician had prescribed the tablets as a sleeping aid. The patient did not require intubation. Studies done at the time of admission gave the following values: serum carbon dioxide combining power, 21.0 mEq/liter; serum chloride, 111 mEq/liter; serum glucose, 100 mg/100 ml; hemoglobin, 11 gm/100 ml; hematocrit, 33%; and platelets, 61,000/mm^3 (increasing to 150,000/mm^3 four days later). Serum sodium and serum potassium levels were normal. Arterial pH was never higher than 7.50. Screening tests for drugs (methaqualone, barbiturates, alcohol, and salicylates) disclosed only methaqualone.

The patient remained in a coma for four days. His blood pressure was never higher than 130/80 mm Hg. An ophthalmology consultation was requested on Aug. 28, 1979, because of inferior corneal drying. At that time, the patient still appeared moderately sedated. Visual acuity was 6/6 (20/20) in both eyes. Extraocular movements were full without diplopia. The pupils were each 4 mm and reacted well to light and accommodation. The slit-lamp examination disclosed minimal inferior corneal stippling in each eye secondary to incomplete eyelid closure. The anterior chamber and lens were clear in each eye. Applanation tensions were 15 mm Hg in each eye. Ophthalmoscopic examination with the pupils dilated showed intraretinal and preretinal (subinternal limiting lamina) hemorrhages in both eyes (Figs. 1 and 2). The optic disks were normal.

The patient was observed as a psychiatric inpatient after being discharged from the intensive-care unit. He was released from the psychiatric ward on Sept. 15, 1979. Indirect ophthalmoscopy and Goldmann contact lens examinations were performed at weekly intervals. Six weeks after the overdose (Oct. 2, 1979) the hemorrhages in both eyes had clinically resolved.

When I tried to determine why the patient had not returned for post-resolution photographs, I discovered that he had again taken an overdose of methaqualone and had died. The eyes were not available for histopathologic study.

DISCUSSION

Previous reports have noted that excessive amounts of methaqualone cause purpura and conjunctival hemorrhages without retinal hemorrhages.[1,2] No hematologic values were given. Fraunfelder[3] mentioned retinal hemorrhages as a possible complication of methaqualone. The present case demonstrated large retinal hemorrhages without conjunctival hemorrhage. Other investigators have reported transient loss of central vision and yellow vision,[4] perhaps a foveal hemorrhage which reabsorbed. Normal and abnormal pupillary reactions have been described in methaqualone abuse. Our patient had normal pupillary reactions.

We know that excessive amounts of methaqualone can cause aplastic anemia.[5] Such an aplastic anemia would take at least 12 days to manifest itself, because 12 days is the half-life of circulating platelets. My patient was observed only 48 hours after the overdose. The mecha-

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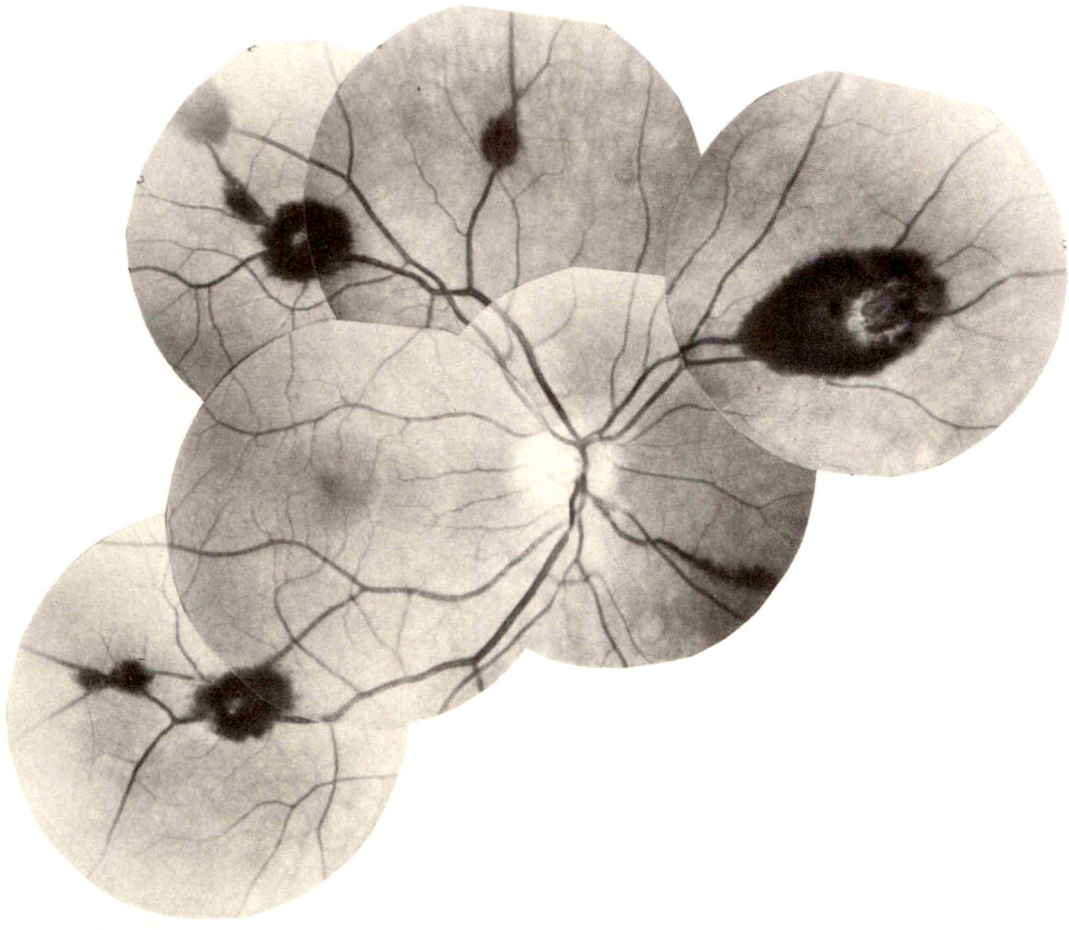


Fig. 1 (Trese). Right eye. Montage shows intraretinal and sublaminar hemorrhages predominantly involving the retinal veins.

nism of the hemorrhages was an acute toxic or consumptive affect on circulating platelets. This could have caused retinal hemorrhages and purpura. The rapid return to a normal platelet count also suggested a toxic affect. The alcohol ingestion probably had little to do with the thrombocytopenia. Another possible mechanism was generalized hypoxia leading to increased capillary permeability and hemorrhage.

The clinical impression was that these hemorrhages were intraretinal and preretinal (subinternal limiting lamina). This suspicion was supported by the rapid resolution of these hemorrhages. It is often difficult to distinguish preretinal hemorrhage (hemorrhage beneath the internal limiting lamina of the retina) from subhyaloid hemorrhage (hemorrhage between the internal limiting lamina and posterior hyaloid face). Attempts to distinguish between the two have been based on the shape of the hemorrhage (boat-shaped) or on the quality of the reflex overlying the hemorrhage. A more reliable method may be to determine the rate of reabsorption of the hemorrhage. The rapid resolution (six weeks) of these hemorrhages in this patient indicates

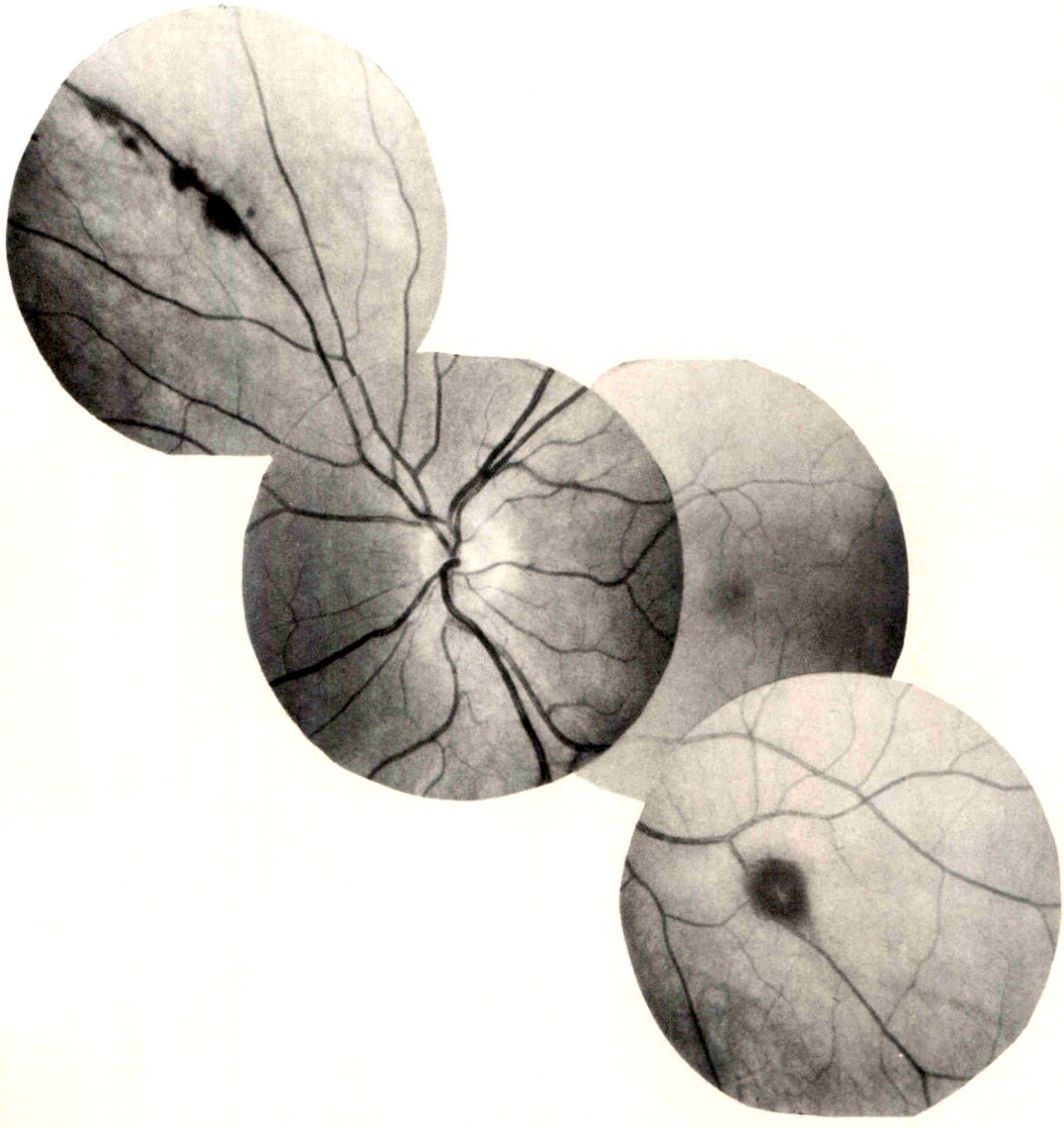


Fig. 2 (Trese). Left eye. Montage discloses the same pattern of involvement shown in Figure 1.

closer contact with the sensory retina and retinal circulation and, therefore, a sublaminar location.

Summary

A 23-year-old man who had taken an overdose of methaqualone (Quaalude) developed purpura and intraretinal and preretinal (sublaminar) hemorrhages. Methaqualone-induced thrombocytopenia may have been the mechanism of these hemorrhagic complications.

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HISTOLOGIC STUDY OF THE OCULAR LESIONS IN MULTIPLE ENDOCRINE NEOPLASIA SYNDROME TYPE IIb

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Three syndromes characterized by multiple neoplasms of the endocrine glands are currently recognized: multiple endocrine neoplasia type I (MEN type I), multiple endocrine neoplasia type IIa (MEN type IIa), and multiple endocrine neoplasia type IIb or III (MEN type IIb or III). Manifestations of multiple endocrine neoplasia type IIb include medullary thyroid carcinoma, multiple mucosal neuromas, pheochromocytoma, a marfanoid habitus, a characteristic facies, intestinal ganglioneuromatosis, skeletal anomalies, an abnormal response to the intradermal administration of histamine, prominent corneal nerves in a clear corneal stroma, and several other ocular abnormalities.[1-5] Recognition of these ocular lesions as components of the syndrome is of the utmost importance because prompt identification of individuals affected by multiple endocrine neoplasia type IIb facilitates early treatment of associated medullary thyroid carcinomas and pheochromocytomas.

Corneal tissues in individuals with multiple endocrine neoplasia type IIb have rarely been studied by light or electron microscopy, and, to the best of our knowledge, there is no published report of a histopathologic study of the various ocular lesions in the syndrome. We studied the ocular lesions in four eyes obtained post mortem from two patients. Clinical reports of these patients were previously published by Norton and associates.[3]

CASE REPORTS

Case 1—At the age of 12 years, this boy had a six-month history of bilateral neck masses. A total thyroidectomy and bilateral radical neck dissection were performed for a medullary thyroid carcinoma in both lobes of the thyroid gland. One of 32 resected cervical lymph nodes contained metastases. The patient was referred here when he was 13 years old for evaluation of recurrent disease.

His medical history disclosed that the patient experienced birth trauma and had delayed developmental milestones. Although severe constipation was noted when he was 6 months old, this resolved spontaneously; a watery diarrhea subsequently developed. The diarrhea stopped after the patient's thyroidectomy, but recurred as his metastatic disease progressed. There was no family history of thyroid or adrenal disease.

At the time he was referred here, the patient had a marfanoid habitus (Fig. 1) and was in the 75th percentile for height and 25th percentile for weight. Skeletal abnormalities included pectus excavatum, bilateral pes cavus, bilateral tapes equinovarus deformities, hyperlordosis, and thoracolumbar scoliosis. He had widely separated teeth and the characteristic facies of multiple endocrine neoplasia type IIb, with thickened bumpy lips, prominent base of the nose, and prominent jaw (Figs. 2 to 4). Additionally, there were several small tumors on his tongue. Enlarged corneal nerves were observed with the slit lamp (Figs. 5 and 6).

Despite treatment with various combinations of doxorubicin HCl, fluorouracil, streptozotocin, bleomycin, vinblastine, and cisplatin, the disease continued to progress. Chest roentgenograms and liver-spleen scans disclosed abnormalities consistent with metastases. The patient died during his final admission in July 1979 after receiving a course of mitomycin followed by an autologous bone marrow transplantation.

At autopsy medullary thyroid carcinoma was found in the lungs, liver, lymph nodes, and kidney. Both adrenal glands contained pheochromocytomas (not diagnosed before death). There was moderate ali-

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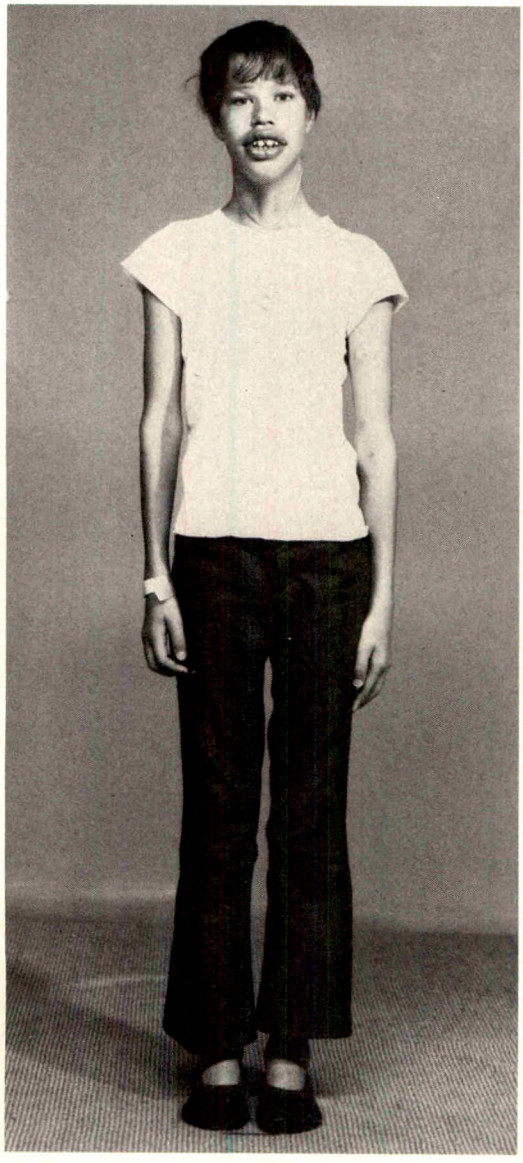


Fig. 1 (Spector, Klintworth, and Wells). Case 1. Individual with multiple endocrine neoplasia type IIb has marfanoid habitus and characteristic facies with large lips.

mentary tract ganglioneuromatosis and severe candidiasis of the colon.

Case 2—When she was 26 years of age, this woman noted a mass in her neck. This was treated with a partial thyroidectomy and a right radical neck dissection one year later for bilateral medullary carcinoma of the thyroid gland. Seven resected cervical lymph nodes contained metastases. Despite postoperative

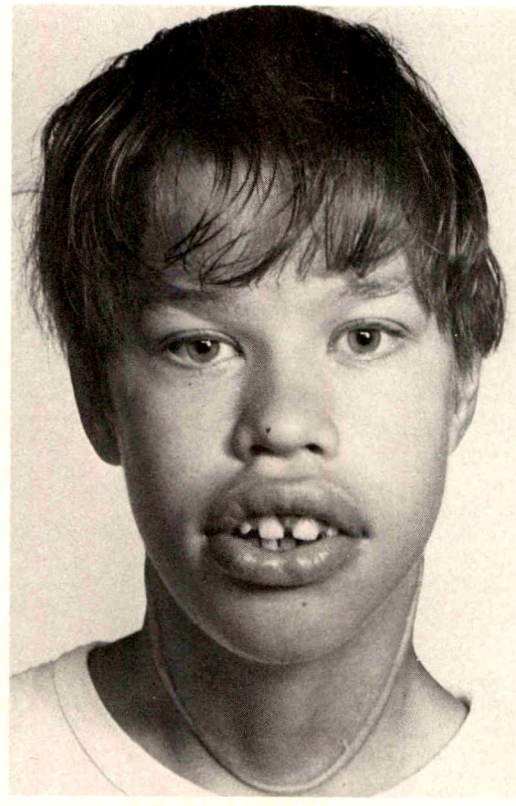


Fig. 2 (Spector, Klintworth, and Wells). Case 1. Characteristic facies of patient with multiple endocrine neoplasia type IIb includes thickened lips, prominent base of the nose, conspicuous jaw, and thickened eyelids. (Reprinted with permission from Norton and associates.[3])

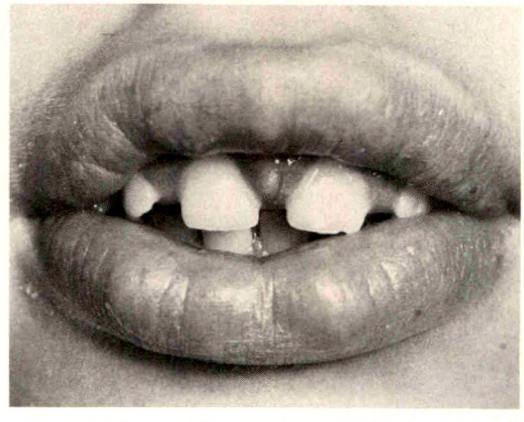


Fig. 3 (Spector, Klintworth, and Wells). Case 1. Thickened lips and widely separated teeth.



Fig. 4 (Spector, Klintworth, and Wells). Case 1. Numerous small neuromas on tongue.

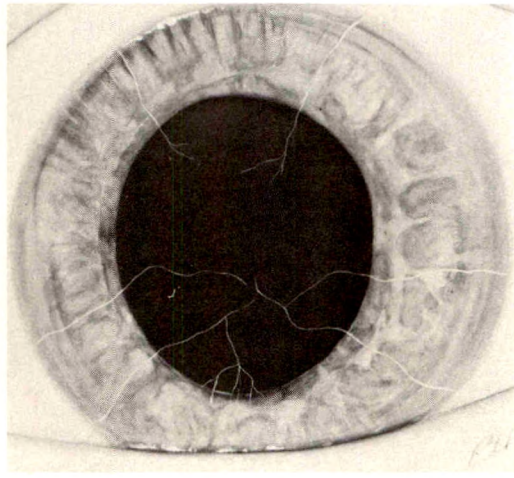


Fig. 5 (Spector, Klintworth, and Wells). Case 1. Drawing of prominent corneal nerves in a clear corneal stroma.

irradiation of the neck and superior mediastinum, roentgenograms of the chest during the next six months disclosed pulmonary nodules and a progressively enlarging mediastinal mass. Radionuclide scanning of the liver demonstrated filling defects.

As a child the patient had experienced chronic constipation, which required a weekly enema. At the age of 26 years the patient experienced episodes of tachycardia, pallor, diaphoresis, nervousness, and diarrhea. The diarrhea resolved after the partial thyroidectomy, but recurred as her metastatic disease progressed. The family history was noncontributory for thyroid and adrenal tumors, as well as for other symptoms of multiple endocrine neoplasia type IIb.

On physical examination the patient was tall and thin with a marfanoid habitus and the characteristic facies associated with multiple endocrine neoplasia type IIb (Fig. 7). Skeletal abnormalities included dorsal scoliosis and bilateral pes cavus. Ocular findings included prominent corneal nerves by slit-lamp examination and multiple neuromas of the eyelids. Neuromas were also present over the lips and tongue; a right submandibular and a left axillary lymph node were palpable.

Results of laboratory studies (including a urinary vanillylmandelic acid level of 17 mg/24 hr, an epinephrine level of 47 μg/24 hr, and a norepinephrine level of 118 μg/24 hr) were consistent with the presence of a pheochromocytoma, so the patient underwent a laparotomy. Both adrenal glands contained pheochromocytomas; the one in the right gland measured 4 × 6 cm and the one in the left measured 1 × 1 cm. The lesion in the right adrenal gland was resected. A liver biopsy confirmed the presence of metastatic medullary thyroid carcinoma and numerous diverticula were detected in the large bowel.

The patient's disease continued to progress. At the age of 31 years she developed spastic paraparesis as the result of a metastatic focus in the vertebral column for which she underwent laminectomy. A few months later her final hospital admission was prompted by the onset of a left-sided facial paralysis. She died of progressive respiratory failure caused by bronchopneumonia.

Autopsy disclosed medullary thyroid carcinoma in the lung, liver, ovary, and mediastinum. The wall of the trachea was completely eroded by the superior mediastinal mass and the petrous portion of the left temporal bone was destroyed by a large necrotic tumor mass. Metastatic carcinoma also involved the seventh thoracic vertebra at the site of the previous laminectomy. There was extensive intestinal ganglioneuromatosis.

Results

Both eyes were removed from the two patients at autopsy. These were obtained one and three hours after death. One eye from each patient was fixed in 10% buffered formalin or 4% cacodylate-buffered glutaraldehyde (pH 7.2). Central horizontal sections of each eye were processed by standard procedures for light microscopy. We stained representative sections with hematoxylin and eosin (with and without luxol fast blue) and Masson's trichrome and Holmes' stains. Representative pieces of the glutaraldehyde-fixed conjunctiva, cornea, iris, and choroid were processed for electron microscopy. These tissues were postfixed with 1% osmium tetroxide and embedded



Fig. 6 (Spector, Klintworth, and Wells). Case 1. Slit-lamp appearance of corneal nerves.



Fig. 7 (Spector, Klintworth, and Wells). Case 2. Characteristic facies of patient with multiple endocrine neoplasia. (Reprinted with permission from Norton and associates.[3])

in epoxy resin for transmission electron microscopy by the customary methods. We cut sections 500 nm thick and then stained them with toluidine blue to select appropriate areas for study by transmission electron microscopy. We studied sections 50 nm thick by transmission electron microscopy after staining them with uranyl acetate and lead citrate.

Examination of the eyes of both patients disclosed markedly thickened nerves in the cornea, conjunctiva, and iris (Figs. 8 to 12). Several discrete masses (400 to 600 μ) composed of aggregations of Schwann cells around poorly myelinated axons were evident within the conjunctiva (Fig. 10). There was also



Fig. 8 (Spector, Klintworth, and Wells). Prominent nerve in corneal stroma (arrow) (hematoxylin and eosin, ×600).

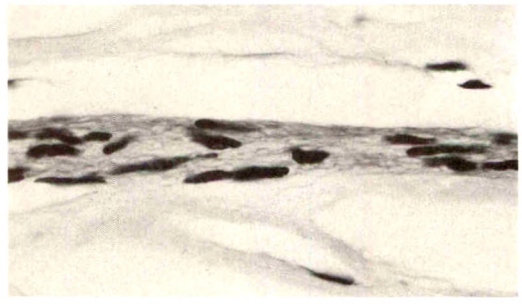


Fig. 9 (Spector, Klintworth, and Wells). Higher magnification of corneal nerve shown in Figure 8. Note the many Schwann cell nuclei (hematoxylin and eosin, ×400).

an excessive number of ganglion cells in the ciliary nerves within the choroid and ciliary body (Fig. 13). Abnormalities were not found in the optic nerve, sclera, retina, or lens.

Transmission electron microscopy showed that the thickened corneal nerves consisted of bundles of nonmyelinated axons in close association with Schwann cells (Fig. 14). Although the thickened nerves in the conjunctiva also contained numerous Schwann cells and axons the enlarged conjunctival nerves, in contrast

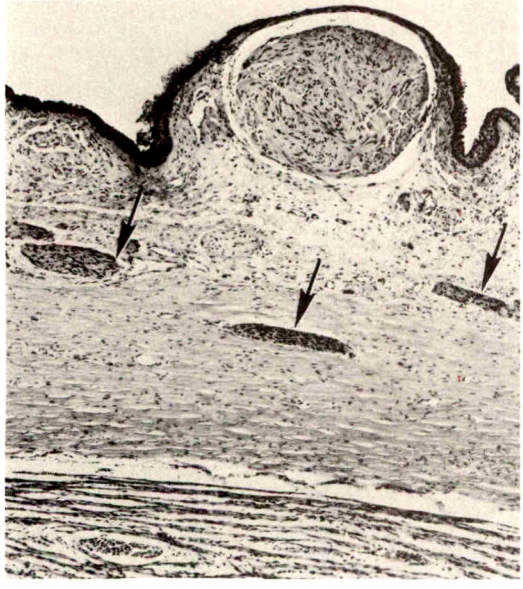


Fig. 10 (Spector, Klintworth, and Wells). Small neuroma and prominent nerves in conjunctiva (arrows)(hematoxylin and eosin, ×40).

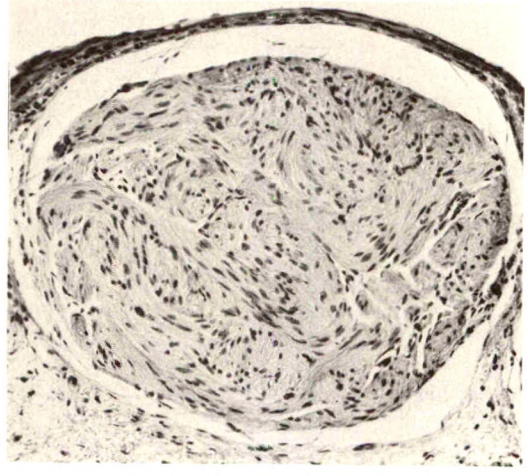


Fig. 11 (Spector, Klintworth, and Wells). Higher magnification of neuroma showing abundant Schwann cells (hematoxylin and eosin, ×100).

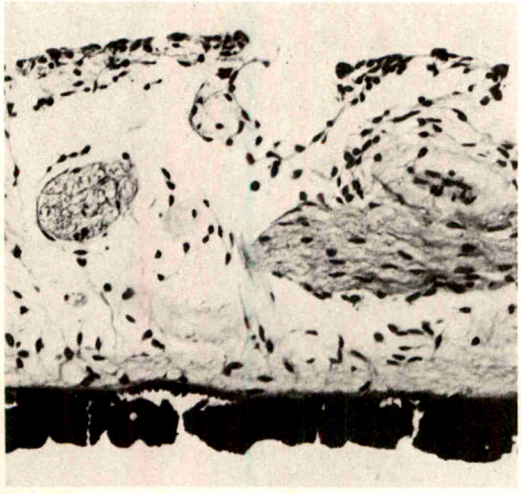


Fig. 12 (Spector, Klintworth, and Wells). Thickened nerves are present in the iris stroma (hematoxylin and eosin, ×170).

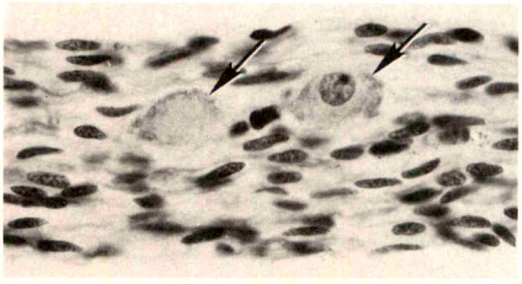


Fig. 13 (Spector, Klintworth, and Wells). Prominent ganglion cells within ciliary nerve (arrows) (hematoxylin and eosin, ×400).

to the cornea, were partially myelinated (Fig. 15).

Discussion

The three multiple endocrine neoplasia syndromes have a number of overlap-

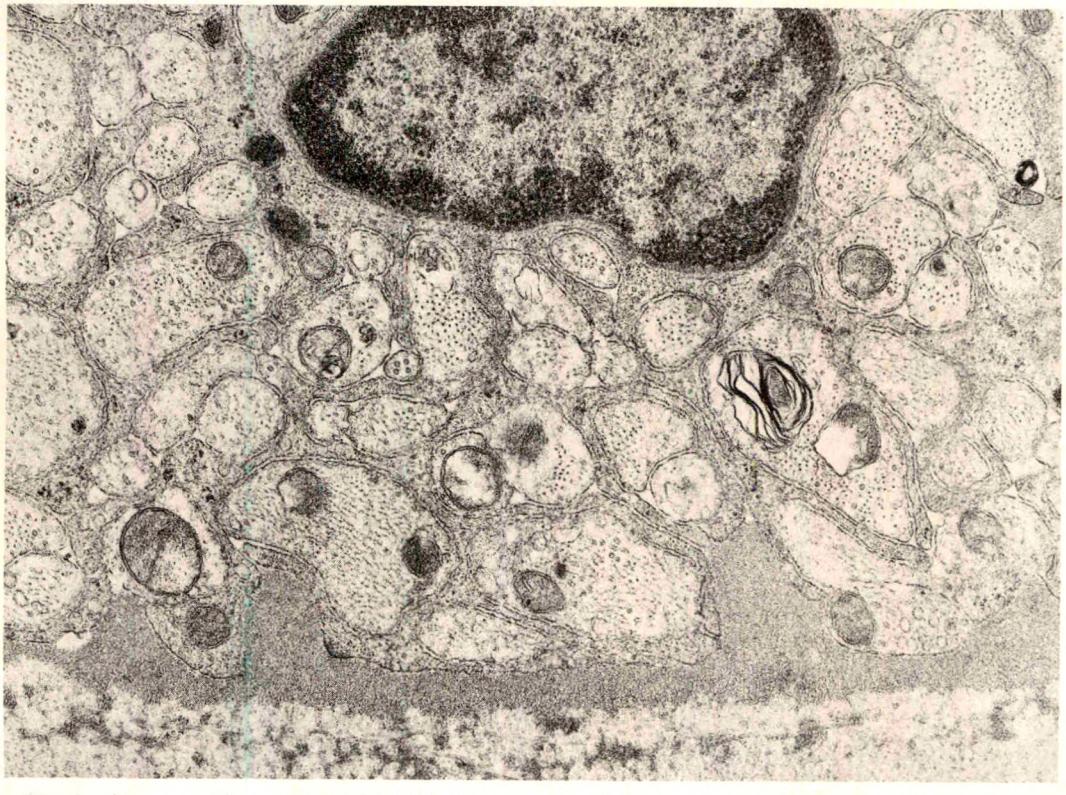


Fig. 14 (Spector, Klintworth, and Wells). Transmission electron micrograph of corneal nerve showing numerous axons surrounded by abundant basal lamina of Schwann cell. Note the absence of myelinated axons (×30,000).

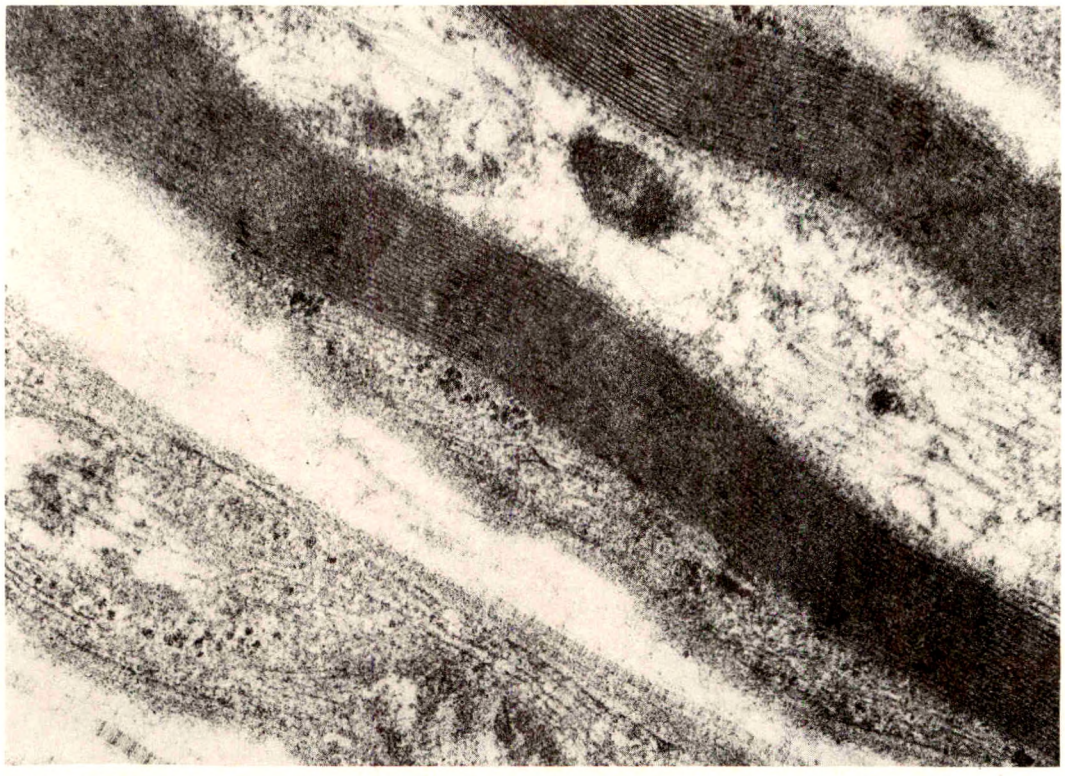


Fig. 15 (Spector, Klintworth, and Wells). Electron microscopic appearance of myelinated conjunctival nerve (×35,000).

ping characteristics (Table 1). Although it was first described in German by Wagenmann[6] in 1922, Williams and Pollock[5] were the first to delineate multiple endocrine neoplasia type IIb as a clinicopathologic entity. This condition is of particular importance to the ophthalmologist because ocular problems may be the first sign. Recognition of ocular abnormalities as components of the entity may facilitate diagnosis and thus bring about early treatment of the associated and potentially fatal medullary thyroid carcinomas and pheochromocytomas. Family members should also be examined because about 50% of the persons with multiple endocrine neoplasia type IIb appear to inherit the condition as an autosomal dominant trait. In addition to the ophthalmic manifestations, individuals with this condition almost always develop many small neuromas on the tongue (especially on the anterior dorsal surface) before the eighth year of life. They are sometimes evident at or within a few years of birth.[1] These neuromas are diagnostically important.

The most commonly noted ocular findings in multiple endocrine neoplasia type IIb are prominent corneal nerves, hamartomatous thickened nerves, and thickened everted eyelids. Other ophthalmic abnormalities that have been observed in affected patients are listed in Table 2. The incidences of the various manifestations are difficult to estimate, because most patients with this rare condition have not undergone thorough ocular examinations by ophthalmologists and many case reports are incomplete. However, the observations of Robertson, Sizemore, and Gordon[4] on a series of 15 patients provide an approximate frequen-

TABLE 1

CHARACTERISTICS OF THE THREE MULTIPLE ENDOCRINE NEOPLASIA SYNDROMES

Syndrome*	Characteristic
MEN type I (Wermer)	Parathyroid adenomas or hyperplasia
	Pancreatic adenomas (gastrinomas [Zollinger-Ellison syndrome], insulinomas, glucagonomas)
	Pituitary adenomas (all types)
	Thyroid adenomas
	Adrenocortical adenomas
	Subcutaneous lipomas
	Visual field defects secondary to pituitary adenoma
	Autosomal dominant inheritance
MEN type II or IIa (Sipple)	Medullary carcinoma of thyroid
	Pheochromocytomas
	Parathyroid adenomas or hyperplasia
	Multiple mucosal neuromas
	Marfanoid habitus
	Prominent corneal nerves (rare)
	Autosomal dominant inheritance
MEN type IIb or III (Froboese)	Medullary carcinoma of thyroid
	Pheochromocytomas
	Parathyroid adenomas or hyperplasia (rare)
	Characteristic facies (thickened lips, soft-tissue prognathism, broad-based nose, everted thickened eyelids)
	Skeletal anomalies
	Intestinal ganglioneuromatosis
	Abnormal intradermal histamine test
	Many ophthalmic manifestations (see Table 2)
	Inheritance 50% sporadic and 50% autosomal dominant

*MEN, multiple endocrine neoplasia.

cy for certain ophthalmic findings: prominent corneal nerves, 100%; conjunctival neuromas, 87%; neuromas of eyelids, 80%; dryness of the eyes, >67%; and prominent perilimbal conjunctival blood vessels, >40%. Prominent corneal nerves appear to be a constant feature of the syndrome. They have been found in all patients with proven multiple endocrine neoplasia type IIb who have undergone slit-lamp examinations. Because of the slit-lamp appearance, Koke and Braley[7] suggested that the thickened corneal nerves in multiple endocrine neoplasia type IIb resulted from myelinization. Since then several reports have assumed this to be so.[1,3] Although the corneal nerves with multiple endocrine neoplasia type IIb were readily identified by light microscopy in the four eyes we studied, we did not find a stainable myelin sheath around these nerves. Moreover, transmission electron microscopy also failed to disclose myelinated axons within the cornea. Instead, the prominent corneal nerves consisted of numerous closely packed axons and Schwann cells. The latter had abundant basal lamellar material but almost no tendency to ensheath the axons with myelin. Our findings are virtually identical to those of Riley and Robertson.[8] Two reports[9,10] described an

TABLE 2

OPHTHALMIC MANIFESTATIONS OF MULTIPLE ENDOCRINE NEOPLASIA TYPE IIb

Site	Manifestation
Cornea	Prominent nerves in clear stroma
Eyelids	Diffuse or nodular thickening
	Nasal displacement of lacrimal puncta
	Rostral displacement of eyelashes
	Eversion
Conjunctiva	Subconjuntival neuromas (up to 6 mm in diameter)
	Thickened nerves
Lacrimal glands	Decreased tear formation
Eyebrows	Prominent
Iris	Impaired pupillary dilation
	Thickened nerves
Intraocular pressure	Increased (rare)

apparent decrease in the number and size of the corneal nerves after surgical removal of the associated pheochromocytoma, but this observation has not been confirmed in other cases[4] and it is difficult to explain on the basis of the ultrastructural appearance of the corneal nerves.

Snell[11] also noted prominent ganglion cells in the ciliary body. The nodules of the conjunctiva and eyelids are composed of coiled myelinated nerves with a thickened perineurium[11] and, like other neural lesions in this disorder, some contain a few ganglion cells.[7,12] The thickened nerve fibers in the iris, so prominent in the histologic sections from the eyes in our study, have been seen extending through the iris stroma of some patients.[4]

Because of the characteristic phenotype of multiple endocrine neoplasia type IIb and the clear corneal stroma between the nerves, it is not difficult to distinguish this condition from other diseases that cause clinically detectable thickened corneal nerves or nerve-like structures.[4] An association of neurofibromas with enlarged corneal nerves in several reports published during the early part of this century[11-14] has led to the belief that hypertrophied corneal nerves are a manifestation of von Recklinghausen's neurofibromatosis. However, these reports preceded the recognition of multiple endocrine neoplasia type IIb and the distinction between its nerve lesions and neurofibromas. Thickened corneal nerves rarely occur in typical cases of von Recklinghausen's disease and the association between prominent corneal nerves and neurofibromatosis has only been documented histologically in one patient with congenital buphthalmos and bullous keratopathy.[15] Because prominent corneal nerves sometimes develop in persons with underlying corneal diseases,[16,17] the association in that particular instance may have been fortuitous, and early case reports of thickened corneal nerves in subjects with neurofibromatosis may have been examples of multiple endocrine neoplasia type IIb.

Both multiple endocrine neoplasia type IIb and multiple neurofibromatosis are genetically determined disorders of neural crest derivatives, and it is necessary to distinguish between these conditions, which share several attributes (Table 3). Alimentary tract involvement, skeletal anomalies, and an increased incidence of pheochromocytoma are associated with both of these neurocristopathies. Café-au-lait spots have been noted in a few patients with multiple endocrine neoplasia type IIb,[4,18] and lip hypertrophy and eyelid and lingual neurofibromas are among the manifestations of von Recklinghausen's disease.[5] The lesions of the mucous membranes in multiple endocrine neoplasia type IIb are sometimes so diffuse that they simulate those in von Recklinghausen's disease.[19] However, the characteristics of multiple endocrine neoplasia type IIb are usually distinct. Although lesions of the eyelid, tongue, and

TABLE 3

COMPARISON OF MULTIPLE ENDOCRINE NEOPLASIA TYPE IIb AND NEUROFIBROMATOSIS

Multiple Endocrine Neoplasia Type IIb		Von Recklinghausen's Neurofibromatosis
	Basic Defect	
Disorder of neural crest derivatives		Disorder of neural crest derivatives
	Mode of Inheritance	
Sporadic or autosomal dominant		Autosomal dominant
	General Appearance	
Marfanoid habitus		Hemihypertrophy of face or extremity (occasional)
Characteristic facies		Lip hypertrophy (occasional)
	Skeleton	
Various anomalies		Various anomalies
	Mucocutaneous Symptoms	
Multiple mucosal neuromas		Multiple neurofibromas
Café-au-lait spots		Café-au-lait spots
		Molluscum fibrosum
		Freckling, especially in armpits
		General hyperpigmentation
	Alimentary Tract Symptoms	
Ganglioneuromatosis (diffuse hyperplasia of nerves)		Neurofibromas (specific localized tumors)
	Neural Symptoms	
Thickened peripheral nerves		Increased incidence of tumors of the central and peripheral nervous systems (neurofibromas, gliomas, meningiomas, ependymomas)
	Other Tumors	
Increased incidence of pheochromocytomas (56%)*		Increased incidence of pheochromocytomas (1–5%)
	Ophthalmic Symptoms	
Prominent corneal nerves		Prominent corneal nerves (questionable)
Ocular neuromas		Ocular neurofibromas
Thickened eyelids		Thickened eyelids
Increased intraocular pressure (rare)		Congenital glaucoma
Impaired pupillary dilation		Melanocytic hamartomas of iris
Thickened conjunctiva or white lines in the area of the corneoscleral limbus		Hamartomas of retina and choroid
		Melanocytosis of choroid
Decreased tear formation		Retinal detachment (secondary to choroidal neurofibromatosis)
Nasal displacement of lacrimal puncta		Optic nerve gliomas and meningiomas
Prominent orbital ridges		Ectopic neurons in optic nerve
Heavy eyebrows		Orbital bone defects
		Nonexpansile pulsating exophthalmos
		Myelinated retinal nerve fibers

*The pheochromocytomas appear to develop relatively late and are found in 90% of patients more than 20 years old.[2]

lip occur in both von Recklinghausen's disease and multiple endocrine neoplasia type IIb, it is uncommon for this distribution of lesions to be seen in a particular subject with von Recklinghausen's disease.[2] Medullary carcinoma of the thyroid is not a feature of multiple neurofibromatosis. Constipation and diarrhea often occur in multiple endocrine neoplasia type IIb and von Recklinghausen's neurofibromatosis, but the nature of the gastrointestinal lesions are different in the two

disorders. In multiple endocrine neoplasia type IIb, there is typically a diffuse thickening of the nerves with many ganglion cells within the enlarged nerves (ganglioneuromatosis), whereas discrete neurofibromas or schwannomas sometimes develop in the gastrointestinal tract in von Recklinghausen's disease. The diarrhea in multiple endocrine neoplasia type IIb may result from products of the associated medullary thyroid carcinoma rather than from the mechanical effects of the neuronal lesions in the bowel.

Medullary thyroid carcinoma, which usually becomes evident during the second decade of life,[11] secretes the hormone calcitonin which serves as a tumor marker. Increased levels of plasma calcitonin (>300 pg/ml), either basally or after the intravenous administration of calcium or pentagastrin, suggest the presence of medullary thyroid carcinoma even when the tumor is not apparent clinically.[20,21] Basal or stimulated levels of plasma calcitonin exceeding 1,000 pg/ml are generally diagnostic of medullary thyroid carcinoma and serve as an indication for neck exploration and total thyroidectomy. The pheochromocytomas are usually bilateral and develop in older individuals than the medullary thyroid carcinoma (average age, 25 years).

Medullary thyroid carcinomas also produce prostaglandins, serotonin, and ACTH,[1,2] and some of these substances or other products of these tumors may be responsible for the diarrhea which so commonly accompanies multiple endocrine neoplasia type IIb. Normal patients respond to intradermal histamine by forming a wheal surrounded by a flare, but persons with multiple endocrine neoplasia type IIb frequently develop the wheal without the flare. Although increased amounts of histaminase have been detected in the serum of some persons with thyroid medullary carcinoma,[22] this abnormal response to intradermal histamine probably does not result from increased levels of this enzyme in the circulation. An abnormality in the cutaneous nerves is the more likely cause, because the flare still fails to form after surgical removal of the medullary thyroid carcinoma[4,11] and even occurs when the tumor is not associated with an increased serum level of histaminase.[11]

Summary

We studied the clinical and histopathologic findings in the eyes of two patients with multiple endocrine neoplasia type IIb. A prominent clinical feature of this condition is enlarged nonmyelinated corneal nerves composed of bundles of axons associated with Schwann cells. Prominent corneal nerves within an otherwise normal corneal stroma are an important clinical feature of multiple endocrine neoplasia type IIb. Because of this, ophthalmologists are in a position to make an early diagnosis and hence to recommend diagnostic procedures and treatment for the associated potentially fatal medullary carcinoma of the thyroid and pheochromocytomas.

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CONJUNCTIVAL INFLAMMATION AND AMYLOIDOSIS IN ALLERGIC GRANULOMATOSIS AND ANGIITIS (CHURG-STRAUSS SYNDROME)

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Allergic granulomatosis and angiitis (Churg-Strauss syndrome) is a severe, multisystem vasculitis characterized clinically by bronchial asthma, fever, and eosinophilia. Histopathologically there is a necrotizing vasculitis of small arteries and veins with an infiltration of vessels and perivascular tissues by eosinophils. Extravascular granulomas have been noted. We report a case of conjunctival inflammation and amyloidosis in the Churg-Strauss syndrome.

CASE REPORT

In 1975 a 32-year-old woman came here with asthma, eosinophilia, purpura, and mononeuritis multiplex. Churg-Strauss syndrome was diagnosed from these clinical findings and because a muscle-biopsy specimen demonstrated a granulomatous reaction consisting of epithelioid cells, eosinophils, plasma cells, and lymphocytes about an arteriovenous complex (Fig. 1). The disease was controlled with oral corticosteroids and azathioprine until the patient was lost to follow-up in October 1979. She was later examined by another physician who diagnosed corticosteroid-induced diabetes and tapered off the corticosteroid therapy.

In late December 1979, the patient's vasculitis and asthma worsened and she was admitted here for further tests and treatment. She had had chills, fever, wheezing, and general malaise for four weeks.

Her temperature was 38.5° C and her respiratory rate was 24 breaths per minute. There were purpura on her buttocks, extremities, and chin, and the nasal mucosa was friable and edematous.

Laboratory findings included the following values: sedimentation rate (Westergren), 97 mm/hr; white blood cell count, 17,000/mm^3, with 56% eosinophils; and IgE level, 16,677 ng/ml. A chest X-ray showed bilateral upper and lower lobe interstitial infiltrates.

Two of us (R. W. and M. K.) examined the patient approximately one week after admission. The patient said that she had had bilateral upper eyelid swelling and occasional bloody, ocular discharges during the previous two weeks. She complained of a continuous sensation of fullness and itchiness around her eyes. She had been treated topically with chloramphenicol drops and neomycin-dexamethasone ointment by another ophthalmologist just before her admission without obtaining relief. Except for these symptoms, there was no personal or family history of ocular disease.

Best corrected visual acuity was 6/7.5 (20/25) in both eyes. External examination demonstrated a fullness of her upper eyelids, which had a hard, irregular consistency. Both upper tarsal conjunctivas were yellow, waxy, and nodular (Fig. 2). These lesions were hyperemic with dilated surface vessels and superficial hemorrhages. A small pedunculated lesion of the conjunctiva in the right inferior fornix was also noted. There were no corneal, anterior chamber, or lenticular abnormalities. An ophthalmoscopic examination gave normal findings.

Biopsy specimens were taken from the left upper tarsal conjunctiva and from a purpuric skin lesion on the chin. Hematoxylin and eosin demonstrated thickened stroma with pale-staining amorphous matrices heavily infiltrated with eosinophils, plasma cells, and

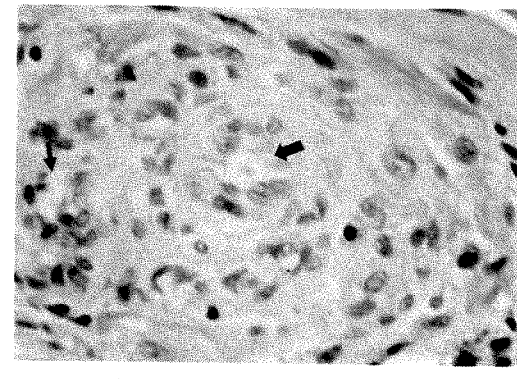


Fig. 1 (Meisler and associates). Granulomatous reaction around an arteriovenous complex on muscle biopsy. Large arrow, lumen of artery; small arrow, lumen of vein (hematoxylin and eosin, ×200).

From the Departments of Ophthalmology (Drs. Meisler, Stock, Wertz, Khadem, and O'Grady) and Pathology (Dr. Chaudhuri), Northwestern University Medical School, and the Veterans Administration Lakeside Medical Center, Chicago, Illinois.

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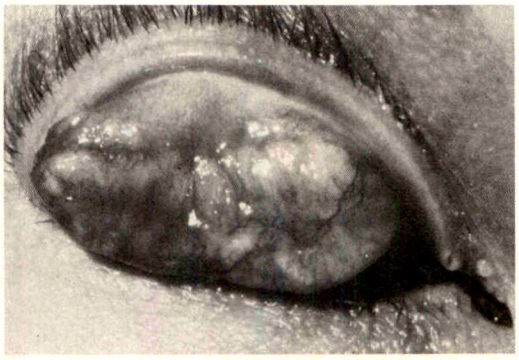


Fig. 2 (Meisler and associates). Right upper tarsal conjunctiva during exacerbation of the Churg-Strauss syndrome.

lymphocytes (Fig. 3). We found focal areas of histiocytic infiltration. Sections prepared with Congo red stain disclosed amyloid scattered throughout the thickened stroma, which exhibited dichroism under polarized light. This was confirmed by electron microscopic studies, which demonstrated the characteristic fibrillar structure of amyloid (Fig. 4).

High-dosage corticosteroid therapy was resumed and the patient showed dramatic clinical improvement. At her most recent follow-up visit in June 1980, her vasculitis had resolved and her asthma was controlled with prednisone, 100 mg every other day. The lesion in the right inferior fornix was no longer present and the lesions of her tarsal conjunctivas had substantially regressed.

Discussion

In 1951, Churg and Strauss described a series of patients with asthma and a rapidly progressive multisystem syndrome characterized by fever and eosinophilia.[1] The most common symptoms were the result of cardiac failure, renal damage, and peripheral neuropathy. Since the original report, the disease has been recognized as a distinct entity belonging to the polyarteritis group of systemic necrotizing vasculitides.[2]

The Churg-Strauss syndrome is distinguished from classical polyarteritis nodosa by clinical and histologic criteria. Asthma is always present in the Churg-Strauss syndrome, whereas it is infrequently associated with polyarteritis nodosa. Polyarteritis nodosa affects small and medium-sized arteries and is characterized by a neutrophilic infiltrate. The Churg-Strauss syndrome is characterized by a vasculitis with fibrinoid necrosis of capillaries and veins in addition to small arteries. There is an infiltration of blood vessels and extravascular tissues, predominantly by eosinophils. Plasma cells and lymphocytes are also present in varying numbers. Histiocytes appear in the chronic stages. Allergic granulomas develop in the extravascular tissues in the Churg-Strauss syndrome and are characterized by a central eosinophilic core consisting of necrotic eosinophilic leuko-

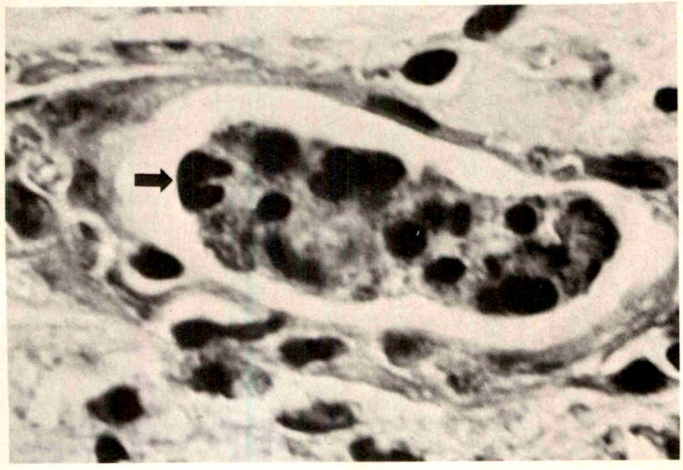


Fig. 3 (Meisler and associates). Accumulation of eosinophils (arrow) within an epithelial nest in the conjunctiva (hematoxylin and eosin, ×400).

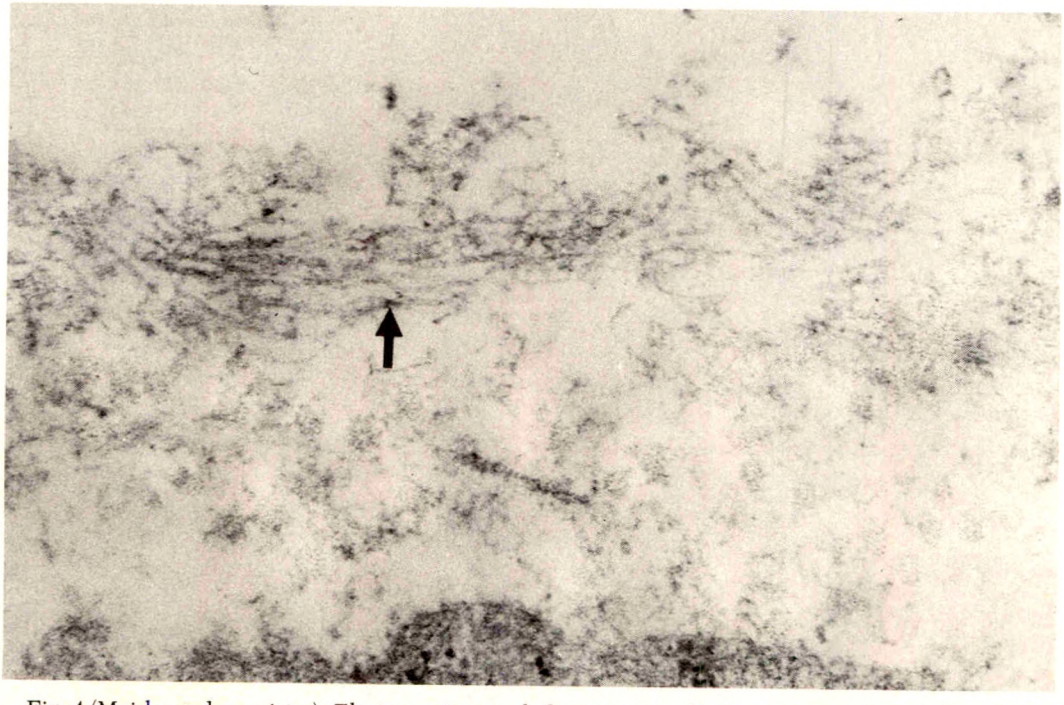


Fig. 4 (Meisler and associates). Electron micrograph demonstrating fibrillar structure (arrow) of amyloid of upper tarsal conjunctiva (×54,000).

cytes and fibrinoid swelling of collagen fibers. This central core is surrounded by radially arranged histiocytes and epithelioid cells.[1] These granulomas are not present in polyarteritis nodosa.

Previous reports of ocular involvement in the Churg-Strauss syndrome are vague and sparse. In 1956 Cury, Breakey, and Payne[3] reported a case of allergic granulomatosis and angiitis associated with uveoscleritis and papilledema. Although they did not describe the general clinical features of the Churg-Strauss syndrome in their report, the histopathologic findings of allergic granulomas in the sclera and uvea are consistent with this diagnosis. Chumbley, Harrison, and DeRemee[4] briefly alluded to the development of a marginal corneal ulcer in one of their 30 patients with Churg-Strauss syndrome. Ashton and Cook[5] described allergic granulomas of the conjunctiva in an asthmatic patient (their Case 12). However, the benign course of the illness and the lack of multisystem involvement makes the diagnosis of Churg-Strauss syndrome unlikely.[5]

The clinical pattern of asthma, leukocytosis with prominent eosinophilia, fever, mononeuritis multiplex, purpura, and extremely high IgE levels in our patient was consistent with the diagnosis of Churg-Strauss syndrome. The muscle-biopsy specimen demonstrating allergic angiitis supported the diagnosis.

The histopathologic findings in the conjunctiva showed the diffuse extravascular inflammatory infiltrate described by Churg and Strauss.[1] The absence of allergic granulomas in our tissue sample may have reflected the acute nature of the exacerbating process. Resolution of the conjunctival lesions at the same time the patient improved clinically suggested

that the conjunctival changes were associated with the underlying inflammatory process.

The firm, nodular, waxy appearance of the conjunctivas was similar to that in other reported cases of conjunctival amyloidosis. Amyloidosis of the conjunctiva is considered to be a primary localized disease when there are no antecedent or systemic factors.[6] It is considered to be a secondary localized disease in cases associated with local inflammatory processes such as trachoma, vernal disease, pinguecula, and pterygium.[7] Amyloidosis of the conjunctiva as part of a systemic disease is rare; however, cases associated with hypothyroidism,[8] rheumatoid arthritis,[9] and hyperglobulinemia[10] have been reported.

According to Brownstein, Elliott, and Helwig,[6] ophthalmologic signs attributable to amyloid infiltrate are virtually unknown in secondary systemic amyloidosis. The finding of amyloid deposits in the skin and conjunctiva of our patient suggested that her amyloidosis was of the secondary systemic variety.

Summary

A 37-year-old woman developed nodular, waxy conjunctival lesions associated with the exacerbation of Churg-Strauss syndrome. Conjunctival and skin biopsy specimens demonstrated a diffuse inflammatory process composed of eosinophils, plasma cells, lymphocytes, and focal collections of histiocytes. There were amyloid deposits in the conjunctiva and skin. To our knowledge, this is the first report of conjunctival changes and amyloidosis associated with the Churg-Strauss syndrome.

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IDOXURIDINE-LIPOSOME THERAPY FOR HERPES SIMPLEX KERATITIS

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An important prerequisite for therapeutic success with a pharmacologically active drug is the drug's ability to reach the target area and maintain its presence there at a therapeutic level. The use of a liposome as a drug carrier can make this possible.[1-3] Liposomes are microscopic structures consisting of one or more concentric lipid bilayers enclosing an equal number of aqueous-filled compartments (Fig. 1). As drug carriers, they can transfer water-soluble, normally impermeable molecules into cells.

The mechanisms by which liposome enhances penetration into the cell have not been clearly defined. Liposomes may adhere to cell walls and release their drug into the cell, or they may be taken into the cell as phagosomes. In order for a drug to be incorporated into the aqueous space within the vesicle, the primary solubility characteristics of the drug must be of a polar nature. Nonpolar drugs may be captured by adherence to the lipid membrane. Drugs equally soluble in lipid and aqueous phases of the liposome may not be effectively captured.

We hoped that adding liposome to idoxuridine before introducing it into cells containing herpes simplex virus would increase idoxuridine's penetration of the cornea and its entrance into the virus-laden cells, substantially enhancing its therapeutic value.

MATERIAL AND METHODS

The liposome was prepared by combining phosphatidylglycerol, phosphatidylcholine, and cholesterol in the molar ratio of 1:4:5 and dissolving them in chloroform. This lipid mixture was placed in a 50-ml round-bottomed flask with a long extension neck. The chloroform was removed by a rotary evaporator under reduced pressure. We purged the lipids with nitrogen and redissolved them in diethyl ether. We added 6 μmole of the lipids first to 3 ml of diethyl ether and then to 1 ml of aqueous-phase idoxuridine. The preparation was kept continuously under nitrogen, and the resulting two-phase system was sonicated for two to five minutes at 0 to 5° C. We removed the ether phase by rotary evaporation under reduced pressure (water aspirator) at 20 to 25° C, rotating the preparation at about 200 rpm. We then dialyzed it overnight against phosphate-buffered saline solution at 40° C. Finally, we forced it through a 200-nm unipore filter under positive pressure.[4] Radioactive labeling of the drug showed that 35% of it had been captured by the liposome vesicles.

The experiment was performed in two parts.

Part 1—We used 18 New Zealand white adult rabbits of both sexes, weighing 2.2 to 2.7 kg each. All of the animals were examined with a biomicroscope to be sure that they were free of external ocular disease.

On day 0 we anesthetized both eyes of each rabbit with 0.5% proparacaine HCl

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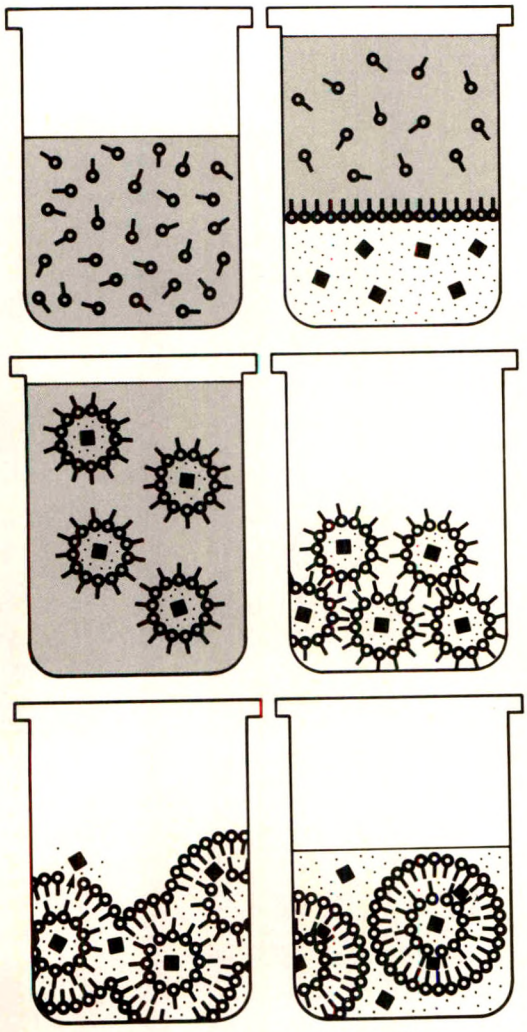


Fig. 1 (Smolin and associates). Use of liposome as a drug carrier. Top left, Lipid solution. Top right, Drug (solid squares) is added. Middle left and right and bottom right, Evaporation and sonication causes vesicles to collapse into gel form. Bottom right, Reconstitution results in multilamellar vesicles. Drug (solid squares) is captured in aqueous phase or on lipid membrane. Some drug may escape into surrounding fluid.

and traumatized the eyes with a platinum spatula to obtain deep penetration of the virus. Ten horizontal and ten vertical scratches were made in the corneal epithelium and superficial stroma. We dropped about 0.01 ml of the pH strain, type 1 herpes simplex virus (lethal dose, 10^{-6}/0.03 ml) onto the rabbit corneas. (The viral pool was tested by diluting it tenfold and injecting eight mice intracerebrally with each dilution. The animals were observed for two weeks and the deaths recorded. The Reed-Muench technique was used to calculate the lethal dose.)

The 18 rabbits were divided randomly into three groups. Group A (rabbits 1 to 6) received 0.1% idoxuridine (35% solution). Group B (rabbits 7 to 12) received the 0.1% idoxuridine-liposome solution (with 35% capture of the idoxuridine). Group C (rabbits 13 to 18) received a liposome-saline solution.

All medications were placed three times each day into the inferior cul-de-sacs of both eyes of all rabbits on days 3, 4, and 5. The clinical epithelial responses were recorded in a single-masked fashion by two observers and were scored according to the area of the epithelial defect as determined by fluorescein staining (Fig. 2). No viral assays were performed.

We also recorded the severity of the conjunctival and anterior chamber responses and the amount of exudate produced. We shall not present these data



Fig. 2 (Smolin and associates). Epithelial defect.

because all of the responses correlated well with the severity of the corneal lesions. The most severe of the corneal lesions, for example, were associated with the severest conjunctival hyperemia, edema, and white purulent exudate, and with the most intense anterior chamber reaction.

Part 2—In the second part of the experiment, we used 21 New Zealand white adult rabbits of both sexes. On day 0, all animals were inoculated as in Part 1. In Part 2, however, we injected 2 mg of triamcinolone acetonide suspension subconjunctivally in both eyes of all rabbits on the day before corneal inoculation with herpes simplex virus and on days 2, 9, and 16 after inoculation.

These animals were divided randomly into three groups of seven animals each. Each group received the same medication as the groups in Part 1 but on days 8, 9, and 10.

In addition to recording the epithelial defect, we noted the degree of stromal opacification and scored it as follows: 0, no stromal opacity; 1, a nebulous opacity of most of the stromal tissue; 2, a macular opacity of most of the stromal tissue; and 3, a leukomatous opacity of most of the stromal tissue (Fig. 3).



Fig. 3 (Smolin and associates). Stromal opacification.

Results

On day 2, all eyes in Part 1 had essentially the same corneal epithelial defect. By day 4 there were still no significant differences among the three groups (Table 1). Thereafter, the group treated with liposome-saline (the control) ran the usual unaltered course of herpetic keratitis, the epithelial defect being at its worst on day 8. The group treated with idoxuridine alone responded favorably to the application of the antiviral drug and had a significantly milder disease than the control group treated with liposome-saline on days 6 and 8 ($P = .05$ on both days). (The Mann-Whitney test for non-parametric values was used for the statistical analysis throughout the experiments.)

The group treated with idoxuridine-lipsome had a significantly milder epithelial disease (smaller defects) than either of the above groups ($P = .05$ on days 6 and 8 when compared with the idoxuridine-treated group, and $P = .01$ on day 6 and .005 on day 8 when compared with the group treated with liposome-saline).

In Part 2 there were no significant differences among any of the three treatment groups on day 7 (Table 2). By day 12, however, the group treated with idoxuridine-liposome had significantly smaller epithelial defects than the other groups. When they were compared with the idoxuridine-treated group, $P = .01$; when they were compared with the group treated with liposome-saline, $P = .001$. On day 12 the idoxuridine-treated group also had smaller epithelial lesions than the control group treated with liposome-saline ($P = .05$). The stromal lesions were evaluated on day 18 (Table 3). The stroma was the least affected in the group treated with idoxuridine-liposome; $P = .01$ when compared with the idoxuridine-treated group. Only one of the 14 eyes in the group treated with idoxuridine-liposome had any stromal disease. This was in striking contrast to 11 eyes in the idoxuridine-

TABLE 1

PERCENTAGE OF CORNEAL DEFECT IN PART 1 OF EXPERIMENT

Rabbit No.	Day 4		Day 6		Day 8	
	R.E.	L.E.	R.E.	L.E.	R.E.	L.E.
			Group A: Idoxuridine			
1	5	5	5	5	25	25
2	10	10	10	20	15	50
3	5	15	5	20	15	45
4	30	15	65	35	35	40
5	10	45	55	65	65	35
6	0	5	5	5	15	25
			Group B: Idoxuridine-Liposome			
7	15	20	30	5	35	5
8	15	10	20	0	35	0
9	30	10	35	35	45	45
10	15	10	0	0	0	0
11	10	5	10	5	10	5
12	5	10	5	5	15	15
			Group C: Liposome-Saline			
13	5	10	35	10	45	10
14	10	10	40	30	55	60
15	5	15	55	65	85	70
16	30	5	60	60	80	85
17	10	15	65	60	65	65
18	5	10	45	40	55	45

TABLE 2

PERCENTAGE OF CORNEAL DEFECT IN PART 2 OF EXPERIMENT

Rabbit No.	Day 7		Day 12	
	R.E.	L.E.	R.E.	L.E.
		Group A: Idoxuridine		
1	75	60	15	10
2	65	60	25	10
3	35	55	10	30
4	45	65	15	10
5	25	35	15	20
6	75	80	20	35
7	80	80	60	50
		Group B: Idoxuridine-Liposome		
8	35	20	0	0
9	85	80	5	0
10	80	75	10	0
11	60	30	15	0
12	20	40	15	0
13	45	85	0	0
14	70	70	0	0
		Group C: Liposome-Saline		
15	35	50	25	30
16	35	65	50	65
17	80	80	55	70
18	60	70	80	80
19	60	50	65	50
20	50	30	55	50
21	55	50	60	50

TABLE 3

CLINICAL SCORES ON DAY 18

Rabbit No.	R.E.	L.E.
	Group A: Idoxuridine	
1	1	0
2	2	1
3	1	3
4	0	1
5	0	1
6	1	1
7	3	4
	Group B: Idoxuridine-Liposome	
8	0	0
9	0	0
10	0	0
11	2	0
12	0	0
13	0	0
14	0	0
	Group C: Saline-Liposome	
15	1	1
16	2	3
17	2	3
18	3	4
19	2	2
20	2	2
21	1	3

treated group and 14 eyes in the group treated with liposome-saline.

DISCUSSION

The local treatment of herpes simplex virus epithelial keratitis has consisted of topically applied chemotherapeutic agents that attack viral synthesis (for example, idoxuridine), debridement of the epithelial lesion, or observation because the majority of acute epithelial attacks resolve within several days without treatment, usually leaving no visible sequelae. The first two methods (antiviral therapy and debridement) are reasonably effective for acute epithelial herpes simplex virus disease in an immunocompetent host. It is in the chronic epithelial, stromal, or uveal-tract disease that therapy can be a problem. Debridement is then usually ineffective, and chemotherapy has been limited by the drugs' inadequate penetration of the cornea, the difficulty of maintaining the drugs long enough at a therapeutic level at the site of infection, and the side effects resulting from their use.

The use of corticosteroids in these chronic cases further complicates the situation. The anti-inflammatory and immunosuppressive actions of the corticosteroids are certainly welcome when one is attempting to maintain the integrity of the tissue in the visual axis, but there is also delayed and impaired wound healing and enhanced viral growth as a result of suppression of vital elements in the body's immune antiviral defense.[5,6]

We hope that the use of corticosteroids can be eliminated, or at least minimized, by an improvement in the therapeutic effect of the antiviral agents. The development of new agents that can penetrate the ocular tissues (for example, acycloguanosine and bromovinyl deoxyuridine)[7,8] and the use of liposome carriers may lead to the effective treatment of deep, chronic herpetic disease.

Liposomes should improve the penetration of the drugs and maintain them at higher therapeutic levels in the target areas (that is, in the cells containing virus). Our results indicate that combining idoxuridine with a liposome carrier significantly enhances its efficacy against epithelial and stromal herpes simplex virus keratitis in rabbits. The liposome-idoxuridine combination was most effective in preventing stromal lesions. It may have accomplished this by destroying the virus early (thus reducing the antigenic load of viruses contained in the stromal tissue) or by reducing the amount of direct cell death by decreased invasion of smaller numbers of surviving viruses, or by both means.

The combination of effective chemotherapeutic antiviral preparations with liposomes can be used in treating stromal and uveal-tract herpetic disease caused

by virus-induced cell death. This combination should be most effective when corticosteroids must be used to preserve visually important tissues. The liposome-antiviral agent may also be helpful when the patient is subjected to the various herpetic triggers such as fever, stress, and sunburn.

Summary

In the treatment of acute and chronic herpetic keratitis, an idoxuridine-liposome preparation was more effective than a comparable therapeutic regimen of idoxuridine alone. Both antiviral agents were more effective than a control combination of liposome and saline.

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ANIMAL AND HUMAN OCULAR SURFACE RESPONSE TO A TOPICAL NONIMMUNE MAST-CELL DEGRANULATING AGENT (COMPOUND 48/80)

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Ocular allergy, a common clinical problem, encompasses a broad range of diseases involving the eyelids, conjunctiva, cornea, sclera, and other ocular tissues. The frequent association of vasodilation, itching, mucous discharge, and conjunctival edema suggests that anaphylactic mechanisms play a role in acute and chronic ocular allergy. The central role of the mast cell in the inflammatory response is well accepted but less well understood. Local anaphylaxis occurs when a previously sensitized individual is re-exposed to a specific allergen, thereby activating IgE-induced mast cell mediator release.[1-3] Mast cells can be activated to release intrinsic preformed and secondary mediators by both immunologic and nonimmunologic means. The presence of mast-cell-derived mediators in the subconjunctival tissue and tear film probably accounts for the inflammatory changes noted in ocular allergies.[4]

Each year millions of people use over-the-counter and prescription drugs to treat this condition.[5] The medications used (decongestant-antihistamine preparations and cromolyn) are relatively innocuous but frequently ineffective. Occasionally, the clinician finds corticosteroid therapy to be essential, despite the well-known risks. Since little is known about the immunologic mechanisms involved in presumed allergic ocular disease, it is useful to study animal and human models. Although a number of immunologically induced animal models of immediate conjunctival hypersensitivity have been described,[6,7] a nonimmunologic mast-cell degranulation model allows one to study the effects of mediators in a controlled clinical setting without the need for immunologic sensitization. Compound 48/80, a condensation product of formaldehyde with paramethoxyphenylethylamine, represents the prototype nonimmune mast-cell degranulating agent.[8] It reliably induces mast-cell degranulation in vitro,[9] and, when injected intracutaneously in humans, produces the triple response of itching, injection, and swelling.[10,11] We describe herein the external ocular effects induced by topical compound 48/80 in the guinea pig, rabbit, and human.

MATERIAL AND METHODS

Preparation—We prepared compound 48/80 in phosphate-buffered saline at concentrations of 7.5 and 50 mg/ml. We prepared antazoline phosphate in phosphate-buffered saline at a concentration of 5 mg/ml. Preliminary studies showed that the dosages used were well located on the dose-response curve. The solutions were adjusted to pH 7.0 to 7.4 with 0.1N sodium hydroxide. Phosphate-buffered saline alone served as the placebo control. The osmolarity of the solutions was checked to ensure physiologic tonicity.

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Experimental procedure—Eight male Hartley guinea pigs weighing approximately 500 g each and eight male New Zealand albino rabbits weighing approximately 2,000 g each were treated topically with 20 μl of compound 48/80 (50 mg/ml) in the right cul-de-sac and 20 μl of phosphate-buffered saline in the left cul-de-sac.

Nine normal, healthy laboratory volunteers received 20 μl of compound 48/80 (7.5 mg/ml) in the right cul-de-sac and 20 μl of phosphate-buffered saline in the left cul-de-sac.

Both animal and human participants were examined with the slit lamp before treatment and at periodic intervals after treatment for evidence of injection, chemosis, and mucous discharge, and were photographed to document the observed effects. We assumed that blepharospasm and scratching with the hindpaw indicated itching in the animals. The presence and intensity of itching in the human subjects was determined by subjective evaluation and was graded on a scale of 0 to 3+ (0 = none, 1+ = mild itching, 2+ = moderate itching, and 3+ = severe itching that required scratching). Injection intensity was also graded on a scale of 0 to 3+ (0 = none, 1+ = mild redness, 2+ = moderate redness, and 3+ = severe redness). We noted the presence or absence of chemosis and mucous discharge.

We performed an additional study to determine the effect of antazoline phosphate, a histamine H_1 receptor antagonist,[12] on human eyes pretreated with compound 48/80. Three weeks after the initial study, five of the nine volunteers who had experienced itching for at least 45 seconds received 20 μl of compound 48/80 (7.5 mg/ml) in the cul-de-sac of both eyes. Five minutes later, 20 μl of antazoline phosphate (5 mg/ml) was instilled in the right cul-de-sac. Slit-lamp examinations were performed immediately before and ten minutes after treatment with antazoline phosphate to evaluate external signs of inflammation; itching was determined as previously described.

RESULTS

All species studied responded to treatment with compound 48/80 (Table). The predominant signs were bulbar chemosis in the guinea pigs, palpebral chemosis, injection, and mucous discharge in the rabbits, and injection in the humans. With the concentration used, one human experienced gross chemosis. Itching or blepharospasm was noted in all species. In the human, itching began 15 to 30 seconds after treatment; the peak effect was reported at two minutes; maximum duration was five minutes. In all three species, signs appeared within two minutes. Maximum injection, chemosis, and mucous discharge occurred at 30 minutes, followed by gradual resolution of signs over a two- to four-hour period.

TABLE

SIGNS AND SYMPTOMS AFTER TOPICAL APPLICATION OF COMPOUND 48/80

Species	No.	Dosage	Itching or Blepharospasm	Injection	Chemosis	Mucous Discharge
Guinea pig	8	50 mg/ml	Present in 100%	1+ in 38%	Present in 75%	Absent
Rabbit	8	50 mg/ml	Present in 100%	≥ 1+ in 100%	Present in 100%	Present in 100%
Human	9	7.5 mg/ml	≥ 1+ in 89%	≥ 1+ in 100%	Present in 11%	Absent

In the antazoline phosphate-compound 48/80 study, all subjects experienced itching after treatment with compound 48/80. After the initial sting, antazoline phosphate treatment induced complete resolution of itching within 15 seconds in all subjects. However, the external ocular signs produced by compound 48/80 were unaffected by antazoline phosphate.

Discussion

The itching, injection, chemosis, and mucous discharge caused by topical application of compound 48/80 resembled the signs and symptoms of ocular allergy. Although pathobiologic effects were evident in the species tested, we noted differences at the concentrations used. Mucous discharge, a conspicuous and uniform finding in rabbits, was not detected in guinea pigs or humans. Chemosis, a major sign noted in the bulbar conjunctivas of guinea pigs and the palpebral conjunctivas of rabbits, was less apparent in humans. All species experienced itching or blepharospasm. The differential response may be related to the relative densities of mast cells, reactivity of the vascular system, properties of the connective tissue permitting edema formation, goblet cell population, and type of inflammatory mediators released into the extracellular space. These differences may be useful in determining the effectiveness of particular agents in inhibiting itching, injection, chemosis, and mucous discharge.

Compound 48/80 has been used extensively as a mast-cell degranulating agent both in vitro and in vivo. It is frequently used in studies comparing the pharmacologic effectiveness of various agents in inhibiting immunologic and nonimmunologic mast-cell degranulation.[13-16] Although the exact mechanism of compound 48/80-induced degranulation is not understood, glycolytic substrates and calcium are essential to both compound 48/80- and IgE-mediated degranulation.[17] Unless very high concentrations of the agent are used, compound 48/80-induced degranulation is noncytolytic.[9]

Mast cells are storehouses of inflammatory mediators.[18] Membrane activation initiates the release of primary preformed mediators (for example, histamine and eosinophilic chemotactic factor of anaphylaxis) followed by the synthesis of secondary mediators (for example, prostaglandins, bradykinin, and slow-reacting substance of anaphylaxis) which modulate and amplify aspects of the inflammatory response. Because of the large numbers of mast cells in the human conjunctiva and in guinea pig and rabbit eyelids, significant inflammatory activity is likely.[19-21] Knowledge about mast-cell-derived mediators in tears is limited. We previously demonstrated the presence of histamine, a preformed primary mast-cell constituent, in tears of normal humans, and increased levels in patients with active vernal conjunctivitis.[22] The extensive mast-cell degranulation noted in vernal conjunctivitis probably accounts for the abnormally high level of histamine in tears.[23,24] Topical instillation of histamine in humans reliably produces the in vivo effects of itching and injection in a dose-dependent fashion.[25] Antazoline phosphate, a histamine H_1 receptor antagonist, effectively blocks itching in the histamine model but has no effect on vasodilation.[26] Similarly, in the compound 48/80 model, antazoline phosphate blocks itching but not vasodilation, suggesting that histamine was released and its effects on the histamine H_1 receptor were blocked by antazoline. Other studies have detected prostaglandin F, a secondary mediator, in the tears of normal subjects; these levels are increased in patients with vernal conjunctivitis.[27]

We conclude that compound 48/80 reliably produces the signs and symptoms of external ocular inflammation

without using immunologic sensitization. This may prove to be a useful model with which to study mast-cell mediators and the effectiveness of agents that prevent mast-cell degranulation.

SUMMARY

Topical application of the nonimmune selective mast-cell degranulating agent, compound 48/80, produced the signs and symptoms of ocular allergy (itching, injection, chemosis, and mucous discharge) in eight guinea pigs, eight rabbits, and nine humans. The histamine H_1 receptor antagonist, antazoline phosphate, blocked itching but not vasodilation in five humans pretreated with compound 48/80. This suggests that histamine was one of the mediators released by compound 48/80-induced degranulation. Compound 48/80 may be helpful in evaluating the effects of therapeutic agents capable of modifying mast-cell degranulation and in the study of mediators involved in external ocular inflammation.

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OPHTHALMIC MINIATURE

Whilst finishing *Jeptha* in 1751, Handel became increasingly blind from cataract, but "His faculties remained in their full vigour almost to the hour of his dissolution." He became totally blind in 1753 at which time John Christopher Smith looked after him and continued his music business. There is no truth that Handel wrote "Total Eclipse" in *Samson* at this time since this oratorio was composed in 1741. From Coxe, we learn that: "When Smith played the Organ at the Theatre, during the first year of Handel's blindness, Samson was performed, and Beard sang with great feeling:

Total eclipse—no sun, no moon,
All dark, amid the blaze of noon.

"The recollection that Handel had set this air to Music, with the view of the blind Composer then sitting by the Organ, affected the audience so forcibly, that many persons present were moved to tears."

Milo Keynes, Handel's Illnesses
Lancet 2:1355, 1980

KERATOCONUS AND ACUTE HYDROPS IN MENTALLY RETARDED PATIENTS WITH CONGENITAL RUBELLA SYNDROME

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Postnatal ocular manifestations of the congenital rubella syndrome may appear years or even decades after birth.[1,2] The study of ocular abnormalities appearing in the second decade of life is particularly relevant because children born in the United States in the early 1960s during the rubella epidemic are now reaching their late teens. One unusual occurrence that has appeared in mentally retarded teen-aged and young-adult patients with congenital rubella syndrome is acute corneal hydrops.

CASE REPORTS

The four patients with keratoconus described here were all seen at the Children's Hospital Medical Center where a cumulative diagnostic index has been maintained for the past 14 years. During this period 33,296 patients have been included in the index. Of these, 27 had keratoconus. During this same 14-year period, 170 patients with the congenital rubella syndrome were included in the index. Four of the 170 had keratoconus with evidence of acute or past corneal hydrops.

Case 1 (born 1958)—The diagnosis of congenital rubella syndrome was based on the maternal history of rash and fever diagnosed as rubella at two months' gestation, unilateral cataract and microphthalmia, pigmentary changes in the fundus, deafness, microcephaly, severe retardation, and congenital heart disease. Additionally, the patient had undescended testicles bilaterally.

Available medical records date from the time he was 9 years of age. There was a long-standing cataract in the left eye. When he was 10 years old, his family noticed that he brought lights very close to his right eye. Later, he brought the lights into contact with his right eye in a violent and alarming fashion.

An ocular examination when he was 10 years old documented rotatory and horizontal nystagmus of both eyes. He could fixate a light with the right eye but not with the left. Corneal diameters were 9 mm in the right eye, and 7 mm in the left. The right lens appeared clear but the left lens was opaque and appeared to have undergone partial spontaneous absorption. The left eye was exotropic and hypertropic. There was pigment mottling in the fundus of the right eye, especially in the macular region. It was impossible to visualize the fundus of the left eye. There was no evidence of glaucoma in either eye.

An ocular examination when he was 11 years old gave similar results except for the left lens. The previously opaque cataract in the left eye had become a clear membrane with a few opacities in it. The fundus in the left eye could now be visualized; pigmentary retinopathy and a healthy optic nerve were present.

We re-examined the patient when he was 15 years old after the family had noted a dramatic change in the right eye associated with a marked decrease in his visual functioning. Results of an ocular examination of the left eye were not significantly different from those of earlier examinations. The right cornea, however, had marked stromal thickening and opacification (corneal hydrops). A large opacified cone, with microcystic epithelial edema and bullae, protruded inferiorly about 3 mm above the curvature of the clear normal cornea near the superior corneoscleral limbus. The conjunctiva was moderately injected in the right eye. The intraocular pressure in the right eye was 14 mm Hg.

The patient was treated with topically applied atropine and erythromycin. The corneal edema cleared slowly.

At the age of 20 years, he still sought out lights and continued to jab them into his right eye.

Case 2 (born 1969)—The diagnosis of congenital rubella syndrome in this boy was based on maternal rubella in the first trimester of pregnancy, cataracts, deafness, and severe retardation. Medical records currently available date from the time he was 8 years of age. At that time, the family stated that his right eye had been operated on when he was very young. The eye had then turned white. The left eye had been operated on three times.

On ocular examination his right cornea was completely opacified and obscured any view of the anterior segment. The cornea was clear in the left eye and there were lens remnants. The pupil was small and would not dilate enough to allow examination of the fundus.

When the patient was examined while anesthetized, the corneal diameter in the right eye was 11 mm and intraocular pressure was increased. Corneal leukoma precluded further ocular examina-

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tion in the right eye. Corneal diameter in the left eye was 9 mm and intraocular pressure was 9 mm Hg. At gonioscopy there were broad sheet-like peripheral anterior synechiae between which normal trabecular meshwork could be seen. The optic nerve was healthy and pigmentary changes were not prominent.

Neurologic and behavioral reports described the child as "extremely active and aggressive and destructive. . . . He often rolled on the floor, threw his legs up in the air, and pressed and picked at his left eye."

When he was 10 years old, he developed cloudiness of the left cornea and injection of the left eye. An ocular examination while he was anesthetized, done two weeks after the onset of the corneal cloudiness, demonstrated diffuse corneal stromal and epithelial edema with several large breaks in Descemet's membrane. The intraocular pressure was 12 mm Hg. Ultrasound indicated an unremarkable posterior segment without retinal detachment. He was treated with topically applied atropine and a corticosteroid ointment. One month later his conjunctiva was less injected, but the central cornea was still cloudy and some peripheral corneal vascularization was noted. Seven months later the conjunctiva was no longer injected and the cornea was scarred rather than edematous. At slit-lamp examination, an oblique rent in Descemet's membrane was clearly evident with corneal scarring anterior to the break. The corneal stroma was vascularized. He continued to poke himself in the eye, despite all attempts to discourage this activity.

Case 3 (born 1964)—The diagnosis of congenital rubella syndrome in this boy was based on a maternal history of rubella during the first month of pregnancy, bilateral cataracts, bilateral microphthalmia, retinopathy, congenital heart disease, microcephaly, and severe mental retardation. He also had undescended testes bilaterally. Cataract surgery had been performed on the left eye when he was 6 months old and extracapsular cataract extraction on the right eye when he was 4 years old.

He held new objects against his eyes in an apparent attempt to see them. The people working with him on a daily basis recorded vigorous rubbing and digital manipulation of his eyes. An ocular examination when he was 13 years old documented microphthalmia, sensory nystagmus, esotropia, and rubella retinopathy. There was no evidence of glaucoma and the optic disks were normal.

When he was 15 years old, he was examined because he kept rubbing his left eye and appeared to be uncomfortable. The corneal stroma was edematous and swollen (hydrops). The conjunctiva was mildly injected. The edema and swelling subsided, but the corneal opacity and stromal edema still persisted four months after the examination. The left eye was no longer injected and he appeared to be comfortable although he continued to rub the eye vigorously. The right eye had a mild keratoconus without hydrops.

Case 4 (born 1964)—The diagnosis of congenital rubella syndrome in this girl was based on bilateral microphthalmia, bilateral cataracts, and deafness.

Available ophthalmic records date from the time she was 5 years of age. Extracapsular cataract surgery had been performed in both eyes in the first year of life. The patient had esotropia in the left eye and poor fixation. Attempts at patching the right eye in the past had been unsuccessful. Visual acuity in the right eye was 6/30 (20/100). Both eyes had similar residual pupillary membranes but there was a good axial opening in both eyes. The optic disks were flat and pink. There was a mild pigmentary retinopathy in both eyes. Nystagmus was present, with some variability from time to time. She had an enlarged thyroid when she was 10 years of age. This was treated with propylthiouracil.

The ocular symptoms had not changed but, during a routine follow-up visit when she was 12 years of age, keratic precipitates were noted in the right eye. Despite the keratic precipitates the anterior chamber was deep and clear with no evidence of cells or flare. The right eye was treated with topically applied corticosteroids. The keratic precipitates disappeared and did not recur as long as the patient continued using the medication at least once a day.

On another routine follow-up visit when she was 14 years old, intraocular pressure was 32 mm Hg in the left eye. Interestingly, glaucoma occurred in the left eye and the keratic precipitates in the right eye. Intraocular pressure remained high in the left eye until it was successfully treated with timolol. Particularly noteworthy was the finding at slit-lamp examination of an old break in Descemet's membrane of the left cornea. The right eye had some thinning of the central corneal stroma and mild keratoconus.

Discussion

The term acute hydrops was introduced by Rychener and Kirby[3] in 1948 although the phenomenon had been described much earlier.[4] The role of trauma or chronic rubbing in the development of keratoconus has been controversial.[5] Some investigators noted an association between atopy and keratoconus[6-9] and wondered whether chronic rubbing was the linking factor.

All of the individuals described here were retarded and rubbed their eyes vigorously. Excessive trauma may cause a break in Descemet's membrane in an otherwise normal cornea. The presence of keratoconus without hydrops or breaks in Descemet's membrane in the fellow eyes of two of these patients, however,

suggests that the acute hydrops developed in the context of preexisting keratoconus. The presence of transient keratic precipitates in one of these patients, as well as in other individuals with congenital rubella syndrome,[1] suggests that there may be an association between these two corneal manifestations, but such a relationship is still highly speculative. In the four patients described here, chronic vigorous rubbing of the eyes appears to be the common factor accounting for their keratoconus and the occurrence of acute corneal hydrops. Franceschetti[10] noted that acute keratoconus seems to be relatively frequent in mental retardation and several authors have reported the occurrence of keratoconus or acute keratoconus in patients with Down's syndrome.[11-13] The keratoconus and acute corneal hydrops in these four patients probably resulted from the chronic traumatizing mannerisms common in other patients with mental retardation and are not specific for the congenital rubella syndrome.

Summary

Four patients with the congenital rubella syndrome had keratoconus and evidence of acute or previous corneal hydrops. All four of the patients vigorously rubbed and poked their eyes. The keratoconus and acute corneal hydrops in these patients probably resulted from chronic traumatizing mannerisms common in other patients with mental retardation and are not specific for the congenital rubella syndrome.

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GRANULAR CELL TUMOR (MYOBLASTOMA) OF THE PALPEBRAL CONJUNCTIVA CAUSING PSEUDOEPITHELIOMATOUS HYPERPLASIA OF THE CONJUNCTIVAL EPITHELIUM

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Granular cell tumors occur most commonly in the tongue, but the skin and subcutaneous tissues are also frequent sites of involvement. These tumors rarely occur in the ocular adnexal structures and, to my knowledge, only three cases have been reported in which the eyelid alone was involved.[1-3] The patient described herein was, I believe, the first in whom a granular cell tumor appeared clinically as a conjunctival lesion. She was also the first patient with a granular cell tumor of the ocular adnexa to exhibit pseudoepitheliomatous hyperplasia of the overlying epithelium, a pathologic feature associated with granular cell tumors originating elsewhere in the body.

CASE REPORT

A 48-year-old woman, known to have had scleroderma for six years, consulted an ophthalmologist because of a lesion involving the palpebral conjunctiva of her right lower eyelid. She had been aware of the lesion's presence for about eight months.

Examination disclosed an elevated, vascular tumor with papillary features in the nasal aspect of the palpebral conjunctiva. The clinical diagnosis was "conjunctival papilloma" and the ophthalmologist removed the lesion by "shaving it down."

The specimen received in the ophthalmic pathology laboratory measured 3 × 2 × 2 mm. Examination of sections of the lesion disclosed the presence of a mass in the conjunctival stroma. The tumor consisted of nests of large (30 to 60 μ in diameter) cells with rather indistinct cell borders (Fig. 1). Most of the cells were polyhedral and many of them were elongated. The nuclei were small compared with the overall cell size and they did not contain prominent nucleoli. The cytoplasm had a granular appearance (Fig. 1) and was strongly eosinophilic. The granules stained deeply with PAS, but were resistant to the action of diastase, indicating that the intense PAS-positivity did not result from the presence of glycogen. Only occasional inflammatory cells, chiefly neutrophils, were present in the tumor. These were confined almost entirely to the more superficial layers of the lesion.

The conjunctival epithelium overlaying the tumor exhibited marked acanthosis (Figs. 2 and 3). The hyperplastic epithelium demonstrated prominent mitotic activity (Fig. 3) and extended downward far into the conjunctival stroma (Fig. 2). Foci of dyskeratosis were scattered throughout the proliferating epithelium (Fig. 3).

The pathologic diagnosis was "granular cell tumor of conjunctiva with pseudoepitheliomatous hyperplasia of the conjunctival epithelium."

I recalled the patient for follow-up examination six months postoperatively. The conjunctival tumor had not recurred. There was residual nodular thickening of the eyelid margin (Fig. 4), but the patient reported that any increase in size of the residual tumor since the time of surgery had been minimal. The patient was recalled again for examination six years postoperatively. The lesion had not increased in size and her general health remained excellent except for some progression of her scleroderma.

DISCUSSION

Although they have been found in virtually every tissue in the body, granular cell tumors occur most commonly in the tongue (where 40% of them develop), in the skin, and in the subcutaneous tissues.[4] They occur most often in patients from 30 to 50 years old and are usually solitary. Granular cell tumors are almost always benign. There have been several reports of tumors described as "malignant granular cell tumors"; in one case a granular cell tumor of the eyelid was considered metastatic from a primary site in the ovary.[5]

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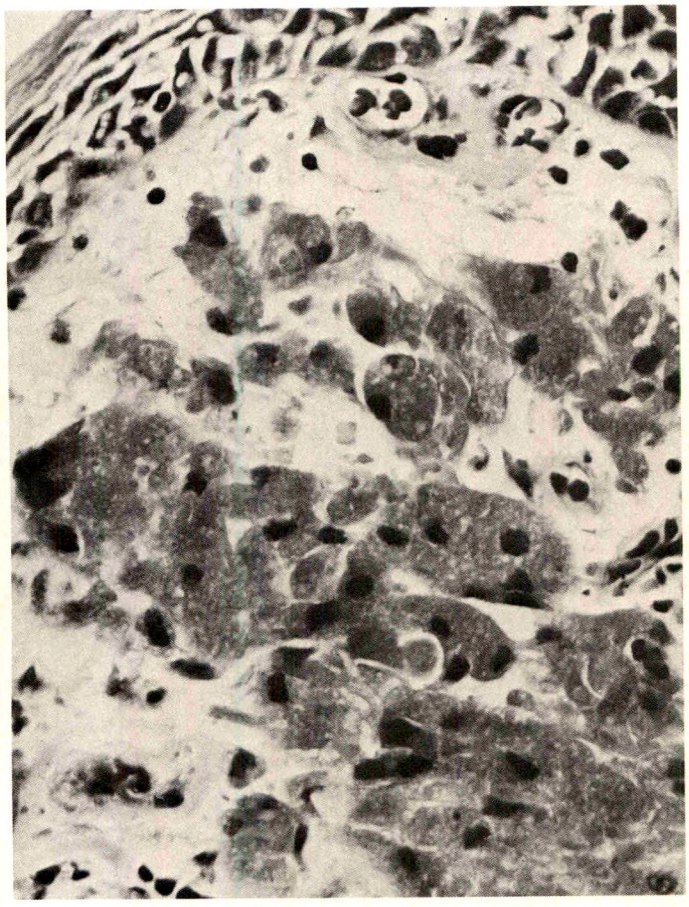


Fig. 1 (Ferry). Nests of large cells with deeply eosinophilic, granular cytoplasm in the conjunctival stroma (hematoxylin and eosin, ×525

Previous reports of ocular adnexal involvement—Granular cell tumors in the eyelid, orbit, or caruncle are rare. Orbital involvement has been reviewed in some detail by Morgan[6] and by Drummond and associates.[7] In a series of 112 caruncular lesions examined pathologically, one was a granular cell tumor.[8] (Cunha and Lobo[9] reported a case of intraocular involvement in which a mass interpreted as a granular cell tumor developed in the ciliary body.)

Although the proximal portion of the eyelid has been involved in some patients with orbital granular cell tumors, the only patients whose tumors originated in the eyelid (without orbital involvement) were the ones described by Timm and Timmel[1] (their Case 2), by Friedman, Eden, and Neumann,[2] and by Rubenzik and Tenzel.[3] (Although the case reported by Cristini[10] is always included in lists of granular cell tumors involving the eyelid, I believe that the lesion he described was not a granular cell myoblastoma.)

The patients described by Friedman, Eden, and Neumann[2] and by Rubenzik and Tenzel[3] had lesions involving the palpebral margin. The recurrent tumor in one case[2] and the preoperative appearance of the tumor in the other[3] resemble the residual tumor at the eyelid margin of this patient (Fig. 4). In all three previously described patients with granular cell tumors originating in the eyelid,[1-3] the lesion appeared to be essentially a

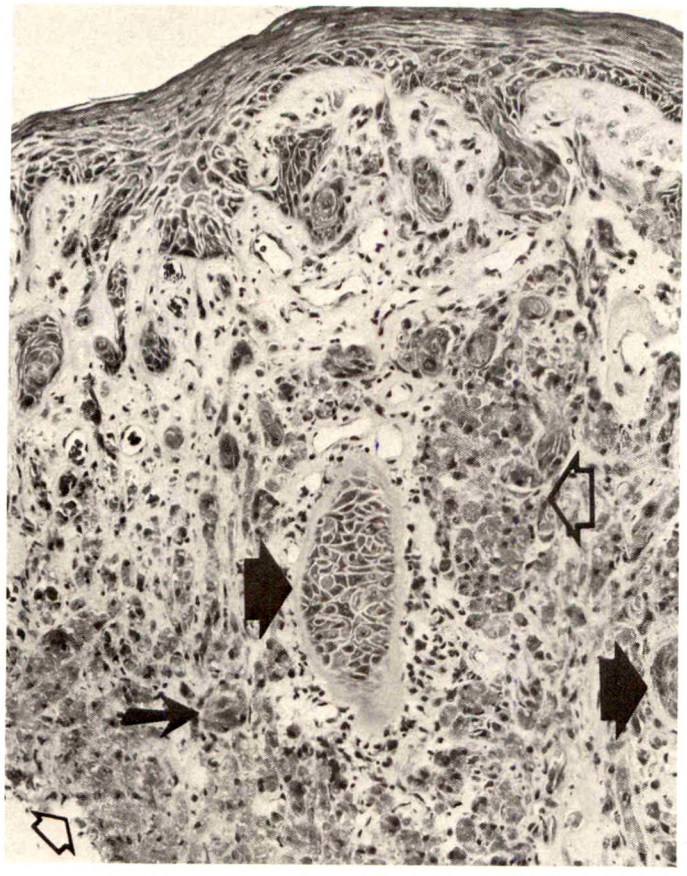


Fig. 2 (Ferry). The conjunctival epithelium exhibits marked acanthosis and extends far down (solid arrows) into the conjunctival stroma. Several prominent nests of granular cells (open arrows) are present in this field (hematoxylin and eosin, × 150).

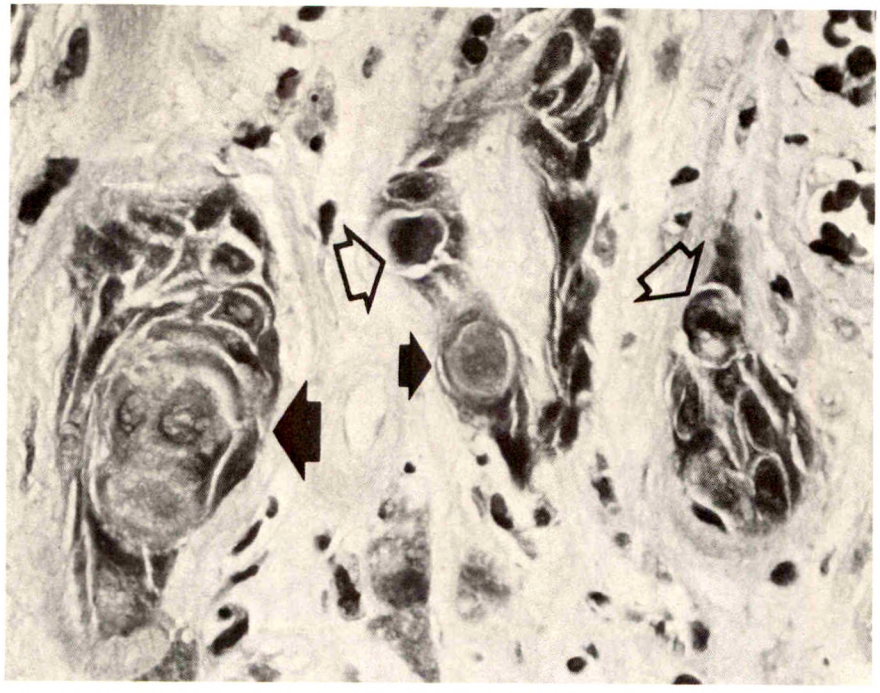


Fig. 3 (Ferry). Foci of dyskeratosis (solid arrows) and mitotic figures (open arrows) are present in the acanthotic epithelium invading the conjunctival stroma (hematoxylin and eosin, × 530).

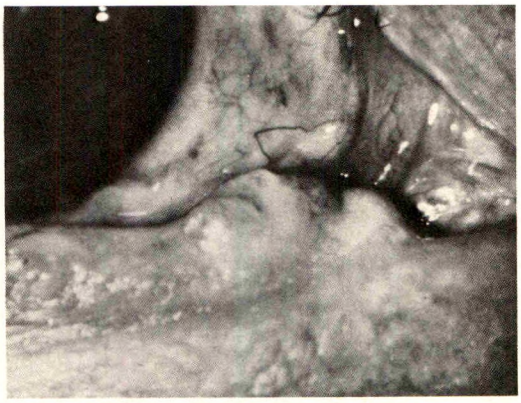


Fig. 4 (Ferry). Six months after the papillary tumor of the nasal palpebral conjunctiva had been "shaved down," there was residual nodular thickening of the corresponding portion of the eyelid margin.

cutaneous one. In this patient the lesion appeared as a papillary tumor of the palpebral conjunctiva.

Histogenesis—The histologic and electron microscopic features of granular cell tumors have been discussed in some detail by several authors.[4,6,7,11] The histogenesis of granular cell tumors remains in doubt. For many years after its recognition in 1926, this tumor was thought to be composed of immature striated muscle cells, hence the designation "granular cell myoblastoma." This view arose because of the frequent close association between the tumor cells and striated lingual muscle fibers, suggesting transitions between them.[4]

Although electron microscopic and histologic studies have eliminated myoblasts as the source of the tumor cells, there is no agreement about their origin. Alternative views are that the tumor cells originate from Schwann cells (Duke-Elder and MacFaul[12] discuss granular cell tumors under the heading of "Nervous Tissue Tumours"), from histiocytes, or from primitive mesenchymal cells resembling fibroblasts.[6,11] In any event, the term myoblastoma is a misnomer because granular cells, like histiocytes, may be merely a morphologic and metabolic condition that a variety of cells can attain, and because neither striated muscle nor smooth muscle features are in evidence.[11]

Pseudoepitheliomatous hyperplasia of overlaying epithelium—Pseudocarcinomatous proliferation of the surface epithelium overlaying granular cell tumors is a well-known histopathologic feature. (As a rule, dermal neoplasms either do not affect the epidermis or they cause epidermal atrophy by compression. Granular cell tumors, by an unknown mechanism, are able to provoke a degree of acanthosis that may simulate squamous cell carcinoma.) This hyperplasia may be seen both in epidermis overlaying dermal or subcutaneous granular cell tumors and in nonkeratinized, mucosal epithelium of the tongue, gastrointestinal tract, or upper respiratory tract.[4] Lever and Schaumburg-Lever[4] emphasized that the pathologist may overlook the few granular cells present in the subepithelial tissue, particularly if a biopsy specimen is superficial, and may diagnose carcinoma.

This case was, to the best of my knowledge, the first in which pseudoepitheliomatous hyperplasia of the overlaying epithelium was present in a lesion involving the ocular adnexa. Timm and Timmel[1] searched for, but did not find, proliferation of the epidermis overlaying the palpebral tumor they reported. There is no mention of pseudoepitheliomatous hyperplasia, or of a search for it, in the brief accounts of histopathologic examinations provided by Friedman, Eden, and Neumann[2] and by Rubenzik and Tenzel.[3]

In this case, the conjunctival lesion had been regarded as a papilloma on clinical examination. The acanthotic epithelium contributed to this clinical picture by having grown down into the subjacent

stroma, thereby subdividing it into loculues of fibrovascular tissue (Fig. 2).

Although granular cell tumors are admittedly rare, the clinician, and particularly the pathologist, should be alert to the possibility that a disturbing epithelial lesion may be caused by pseudoepitheliomatous hyperplasia of the epithelium overlaying a granular cell tumor. Failure of the pathologist to recognize that the epithelial proliferation is benign and reactive may lead to unnecessary further surgery for supposed carcinoma.[4]

SUMMARY

Granular cell tumors rarely occur in the ocular adnexal structures and only three cases have been described in which the eyelid alone was involved. I examined a patient who was, I believe, the first in whom a granular cell tumor appeared clinically as a conjunctival lesion. She was also the first patient with a granular cell tumor of the ocular adnexa to exhibit pseudoepitheliomatous hyperplasia of the overlaying epithelium, a pathologic feature associated with granular cell tumors originating elsewhere in the body.

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GIANT PAPILLARY CONJUNCTIVITIS AFTER KERATOPLASTY

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Giant papillary conjunctivitis occurs in individuals who wear soft and hard contact lenses.[1,2] It is characterized by the presence of giant papillae (1 mm or larger) of the upper tarsal conjunctiva, increased mucus production, mild itching, and decreased contact lens tolerance.[3] It is clinically and histologically similar to palpebral vernal conjunctivitis.[4] Meisler, Zaret, and Stock[5] have described a corneoscleral limbal form similar to corneoscleral limbal vernal conjunctivitis.

Histologic findings showing mast cells, eosinophils, and basophils in the conjunctivas of patients with giant papillary conjunctivitis[2-4] are consistent with an immunologic basis for the condition. Allansmith, Korb, and Greiner[3] noted similar findings, however, in one patient with a protruding suture from a cataract wound and in two others with ocular prostheses. Allansmith and associates[3,4] suggested that giant papillary conjunctivitis is a nonspecific response of the upper tarsal conjunctiva. We found a similar syndrome, unrelated to contact lens wear, in 11 patients who underwent penetrating keratoplasties with interrupted nylon sutures.

SUBJECTS AND METHODS

Each patient had undergone a penetrating keratoplasty performed by one of us (A.S. or R.F.M.). Giant papillary conjunctivitis was discovered during routine postoperative examinations with a Haag-Streit slit lamp. We examined the upper tarsus of both eyes after eyelid eversion. Scrapings from the tarsal conjunctiva were taken with a platinum spatula in selected cases and examined with a light microscope after Giemsa staining. When indicated, loose sutures were removed and the patients were reexamined at monthly intervals.

RESULTS

The patients ranged in age from 14 to 76 years (mean, 51 years) (Table). Six were female and five male. The time of diagnosis ranged from six weeks to nine months after keratoplasty (mean, 4.5 months). The indications for transplantation had varied, but had not differed from those of other patients receiving routine grafts.

All transplants were penetrating grafts; ten were homografts and one was a rotating autograft. In all cases, the surgeon had used interrupted 10-0 nylon sutures, trimmed the knots with a razor knife, and pulled the sutures to the recipient side. In nine patients some sutures had been removed because of adequate healing, looseness, or exposed ends. Nine grafts were clear at the time giant papillary conjunctivitis was diagnosed; two had failed. Two patients had had previous episodes of rejection.

Four patients had worn contact lenses previously (two hard lenses and two soft lenses). None had worn contact lenses in the surgically treated eye since the corneal transplant. One patient had worn a hard contact lens for keratoconus in his fellow eye for 23 years. The upper tarsal conjunctiva in all fellow eyes was normal, as was the lower tarsal conjunctiva in

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TABLE

SUMMARY OF DATA FOR 11 PATIENTS WITH GIANT PAPILLARY CONJUNCTIVITIS

Patient, Age (yrs), Sex	Diagnosis	Time Between Surgery and Diagnosis	Symptoms		
			Mucus	Itching	Blepharoptosis
1, 48, M	Failed graft	3 mos	Yes	No	Yes
2, 42, F	Herpes simplex	3 mos	Yes	Yes	Yes
3, 59, M	Keratoconus	4 mos	Yes	Yes	Yes
4, 64, F	Keratoconus	1½ mos	No	No	Yes
5, 31, M	Trauma	8 mos	Yes	No	No
6, 58, F	Interstitial keratitis	2 mos	Yes	No	No
7, 72, F	Herpes simplex	9 mos	No	No	No
8, 27, M	Perforated ulcer	4 mos	No	No	No
9, 70, F	Aphakic bullous keratopathy	4 mos	No	Yes	Yes
10, 14, M	Sympathetic ophthalmia	5 mos	No	No	No
11, 76, F	Pseudophakic bullous keratopathy	7 mos	No	No	Yes

both eyes of all patients. All patients were using topically applied corticosteroids at the time giant papillary conjunctivitis was diagnosed. Six patients had cardiovascular disease. Only one patient, the individual with keratoconus, had a history of atopy.

Symptoms in most patients were limited to mild irritation. Only one patient spontaneously complained of itching; on direct questioning, two others admitted to mild itching. Five patients noted mucoid discharge; six noted mild-to-moderate blepharoptosis.

All patients had papillae 1 mm or more in diameter in the upper tarsal conjunctiva. In several patients, the papillae increased in size and number during a one-month interval. The papillae were elevated, had flat surfaces, and were pink to gray in color, with fine arborizing surface vessels. A milky thickening of the overlaying conjunctiva was noted in some cases; also, some had mucous strands among the papillae (Figure). No patient had corneoscleral limbal papillae. Most patients had exposed suture knots at some time during their course and mucus had collected on the exposed knots in several. Two patients had epithelial filaments on the upper portion of the corneal graft.

We did not alter the corticosteroid therapy when we diagnosed giant papillary conjunctivitis. When appropriate, loose sutures were removed. One patient had a single giant papilla three months and six giant papillae four months after keratoplasty; one month after removal of

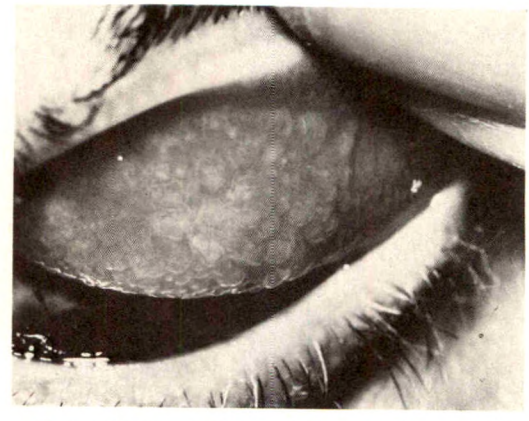


Figure (Sugar and Meyer). Case 6. Giant papillae of upper tarsal conjunctiva two months after keratoplasty.

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two loose sutures all papillae had resolved.

We examined the scrapings from the upper tarsal conjunctivas of five patients. All showed a moderate number of polymorphonuclear leukocytes. One showed moderate eosinophils, two rare eosinophils, and two no eosinophils. We found no plasma cells or basophils.

Discussion

The findings of giant papillae in the upper tarsal conjunctiva, mild mucous discharge, and minimal itching in our patients with interrupted nylon corneal graft sutures are similar to those described in individuals with giant papillary conjunctivitis who wear contact lenses.[1,2] None of our patients wore contact lenses in the involved eye and none had a history of vernal catarrh. These findings may be related to the interrupted nylon sutures with exposed ends. We did not see giant papillary conjunctivitis in any patients who had running nylon sutures. These findings are probably not specific for keratoplasty patients but rather reflect the lack of a conjunctival covering for the sutures used in most cataract patients. That immunologic processes related to the homograft are not involved is suggested by the infrequency of graft rejections in these patients and the occurrence of giant papillary conjunctivitis in one patient with a rotating autograft. The involved patients may have been susceptible to giant papillary conjunctivitis, as has been suggested for some wearers of soft contact lenses.[2,6] There were many more patients with postoperative grafts and interrupted sutures who had normal upper tarsal conjunctivas than there were with giant papillary conjunctivitis. We could not, however, detect any feature that distinguished between the groups. Atopy did not appear to be a factor, just as it is not in wearers of contact lenses.[2]

The finding of eosinophils in conjunctival scrapings from three of five patients tested suggested an allergic origin for the giant papillary conjunctivitis. Deposits of material have been detected on soft contact lenses worn by patients with giant papillary conjunctivitis. These deposits may be a source of antigen. Fowler, Greiner, and Allansmith,[6] however, found similar deposits on the contact lenses of patients without this condition. We have not looked for surface deposits on nylon sutures, although mucus-like deposits were grossly apparent on the knots of many sutures in patients with and without giant papillary conjunctivitis. Aronson and associates[7] demonstrated nonspecific γ-globulin binding to silk corneal sutures, resulting in stimulation of immune inflammatory processes. They stated, however, that nylon sutures are nontoxic and have a limited potential for binding albumin and globulins.[7]

Sutures may play a purely mechanical role in stimulating giant papillary conjunctivitis. Biopsy specimens from the conjunctival surfaces of patients with contact lenses and giant papillary conjunctivitis, unlike specimens from normal subjects, were morphologically consistent with a mechanical process.[8,9] Different abnormalities, also consistent with mechanical trauma, have been found in the conjunctivas of wearers of contact lenses who did not have giant papillary conjunctivitis.[10] The resolution of the condition after removal of loose sutures, even though other nylon sutures remained, is also more easily explained by mechanical irritation than by a true immunologic process. The occurrence of giant papillae in the upper but not in the lower tarsal conjunctiva may be caused by the constant motion of the upper tarsus over the suture ends and the relatively stationary position of the lower eyelid.

Our finding of giant papillary conjunctivitis in patients with interrupted su-

tures but not in those with running sutures suggests the use of running sutures. The ease with which offending sutures can be removed, however, makes this a minor consideration. Possibly, burying the knots of 10-0 nylon sutures would eliminate giant papillary conjunctivitis, but we have found it difficult to remove sutures with completely buried knots. Because the symptoms are mild and respond rapidly to suture removal, we have not altered our surgical technique.

Summary

Giant papillae of the upper tarsal conjunctiva have been found in vernal conjunctivitis and, more recently, in giant papillary conjunctivitis in patients with contact lenses. A similar syndrome occurred in 11 patients who had undergone penetrating keratoplasty with interrupted nylon sutures. Minimal itching, mild mucous discharge, mild blepharoptosis, and giant papillae of the upper tarsal conjunctiva were associated with exposed suture ends. Removal of exposed sutures led to resolution of the condition. Conjunctival scrapings from three of five patients demonstrated eosinophils. Although the syndrome may have an immunologic basis, the findings are also consistent with a mechanical origin related to exposed nylon sutures.

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SAFETY AND EFFECTIVENESS OF CONCOMITANT ADMINISTRATION OF DIPIVEFRIN AND TIMOLOL MALEATE

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Dipivefrin, or dipivalyl epinephrine, is an epinephrine prodromal drug obtained by adding pivalic acid to both ring hydroxyls of the parent epinephrine compound. The pivalyl side chains form a compound which is more lipophilic than epinephrine and which has improved intraocular penetration.[1] The esters are hydrolized, presumably within the cornea, to produce the pharmacologically active epinephrine.[2] Clinical studies have demonstrated that dipivefrin is effective and safe in lowering intraocular pressure,[3-5] and it is relatively free from many of the ocular and systemic side effects associated with topically applied epinephrine therapy.[3,6,7]

Timolol maleate is a topical β-adrenergic blocking agent that effectively lowers intraocular pressure.[8,9] The effects of timolol maleate on intraocular pressure appear to be additive to the effects of miotics, carbonic anhydrase inhibitors, and epinephrine.[10]

This study was designed to explore the safety and effectiveness of combined dipivefrin and timolol therapy.

SUBJECTS AND METHODS

We studied two groups of patient volunteers. All patients in both groups had open-angle glaucoma with increased intraocular pressures when not receiving therapy, gonioscopically open angles, and characteristic visual field and optic nerve changes. None of these patients had a history of ocular intolerance to topically applied epinephrine. The data on pupillary size and intraocular pressure measurements from the two eyes of each patient were averaged, and then analyzed with each patient as a single statistical unit.

The first group consisted of 17 patients (34 eyes) who had been using only 0.25% timolol maleate twice a day in both eyes. This group consisted of eight men and nine women, ranging in age from 54 to 87 years, with a mean age of 70 years. Timolol effectively controlled the intraocular pressure in all patients in this group, as baseline intraocular pressures at the start of this study all measured ≤21 mm Hg.

The second group consisted of seven patients (14 eyes) who had been using only 0.1% dipivefrin in both eyes twice a day. This group consisted of six men and one woman, ranging in age from 34 to 69 years, with a mean age of 60 years. In this group, baseline intraocular pressures at the start of the study were ≤24 mm Hg.

Only patients who were receiving 0.25% timolol in both eyes twice a day or who were receiving 0.1% dipivefrin in both eyes twice a day without other concomitant ocular hypotensive agents were considered for this study. All patients had been using timolol or dipivefrin for at least six months before the study began.

Those patients who had used 0.25% timolol continued to use that medication, but added 0.1% dipivefrin (Group 1). Similarly, those patients who had used 0.1% dipivefrin continued to use that medication, but added 0.25% timolol

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(Group 2). All patients were instructed to instill one drop of each medication in each eye twice a day (8 AM and 8 PM). In all instances, they were instructed to instill the timolol 15 minutes before instilling the dipivefrin. All medications were labeled and known to both patient and physician. Each patient underwent an ocular examination on days 0 (the day before the study began), 14, 28, and 60 (the final day of the study). The examinations included tests for visual acuity and pupil size, ophthalmoscopic and slit-lamp observations, applanation tonometry, and heart rate and blood pressure measurements. We also obtained visual fields with a Goldmann projection perimeter at the start and at the completion of the study. Finally, we asked the patients for their subjective reactions.

RESULTS

Twenty of the 24 patients completed the study. The four patients who left the study were all in Group 1.

Ophthalmoscopic and slit-lamp findings—We found no atypical ophthalmoscopic or slit-lamp results or changes attributable to the study medications in any of the 20 patients. One patient had angular blepharitis at the left outer canthus on the final day of the study, but this appeared to be unrelated to the medication. Corrected visual acuity measurements failed to document any changes directly attributable to the combined use of dipivefrin and timolol. During the course of the study, one patient in Group 1 showed a decrease in visual acuity from the baseline measure of 6/9 (20/30) to 6/22 (20/70). The decrease was caused by progressive nuclear sclerosis. The fellow eye, however, which also had a nuclear cataract, showed an improvement in visual acuity from 6/30 (20/100) to 6/24 (20/80). No other patients experienced a significant decrease in visual acuity. Two additional patients in Group 1 and one patient in Group 2 showed an improvement of two to four lines in visual acuity in at least one eye.

Pupillary size—Of the 17 patients in Group 1, 15 completed the study or participated long enough for adequate pupillary measurements to be obtained. In this group, we noted a significant mean pupillary dilatation of 0.73 mm with the combination therapy (Table). Five of the 30 eyes (16.6%) experienced dilatations between 1 and 2.5 mm. The remaining eyes showed a change in pupillary size of 1 mm or less.

Group 2 showed a mean pupillary constriction of 0.3 mm with the combination therapy (Table). This constriction was not statistically significant. None of the eyes showed pupillary changes of more than 1.5 mm.

Heart rate—The patients in Group 1 showed a mean increase in pulse rate of 2 beats per minute, which was not statistically significant. One patient had a marked increase in pulse rate from 58 to 72 beats per minute.

The patients in Group 2 showed a mean decrease of 13 beats per minute. Two patients in this group showed marked decreases in pulse rate, one from 96 to 68 beats per minute and the second from 94 to 64 beats per minute.

Blood pressure—Group 1 showed a mean decrease in systolic blood pressure of 13 mm Hg and a mean decrease in diastolic blood pressure of 6 mm Hg between baseline and final measurements. Two patients had markedly decreased blood pressures, one from 138/90 to 110/70 mm Hg, and the second from 148/70 to 110/60 mm Hg. Neither had had any blood pressure disorder and neither was receiving any concomitant systemic medication.

Group 2 showed a mean decrease in systolic blood pressure of 6 mm Hg compared with the baseline value. This was not statistically significant. These

TABLE

SIDE EFFECTS OF COMBINED TIMOLOL-DIPIVEFRIN THERAPY

Side Effect	Baseline	Day 60	P*
	Group 1 (Timolol Plus Dipivefrin)		
Pupil size (mm)	2.7 ± 0.6	3.3 ± 0.8	0.001
Heart rate (beats/min)	68 ± 9	71 ± 5	NS
Systolic blood pressure (mm Hg)	142 ± 27	129 ± 25	0.001
Diastolic blood pressure (mm Hg)	83 ± 12	77 ± 12	0.01
	Group 2 (Dipivefrin Plus Timolol)		
Pupil size (mm)	3.5 ± 0.7	3.3 ± 0.7	NS
Heart rate (beats/min)	83 ± 12	70 ± 6	0.02
Systolic blood pressure (mm Hg)	142 ± 17	136 ± 16	NS
Diastolic blood pressure (mm Hg)	91 ± 7	83 ± 7	0.001

*By paired *t*-test; NS indicates not significant.

patients also showed a statistically significant decrease in diastolic pressure of 9 mm Hg. Five of these seven patients showed a decrease in diastolic pressure of 10 mm Hg; only one had a history of systemic hypertension for which he was receiving reserpine systemically.

There was no significant difference in the decreased systolic or diastolic blood pressures between the two groups.

Intraocular pressure—Two patients in Group 1 left the study before we could obtain meaningful data on their intraocular pressures. Data for the other two patients who left the study are included up to the time they left.

The patients in Group 1 showed a change in their mean intraocular pressures from 17.1 mm Hg at baseline to 16.1 mm Hg on day 14 ($P \leq .02$, by paired *t*-test). Intraocular pressure measurements at days 28 and 60 showed no further change (Figure).

The patients in Group 2 showed a decrease in intraocular pressure from 19.0 mm Hg at baseline to 15.4 mm Hg on day 14 ($P \leq .01$ by paired *t*-test). There were no other significant changes on day 28 or day 60 (Figure). The largest decrease in intraocular pressure found in each group was 6 mm Hg.

Subjective side effects—Most of the side effects reported in this study were sporadic, mild, and transient.

Seven patients in Group 1 reported burning sensations, three reported eyelid redness or conjunctival injection, one reported itching, one reported a filmy sensation, one reported eyelid twitching, and one reported tired eyes. Neither the patients nor the physician considered these symptoms significant enough to terminate the study.

Seven patients in Group 1 complained of visual blurring. None of them, however, experienced an objective worsening

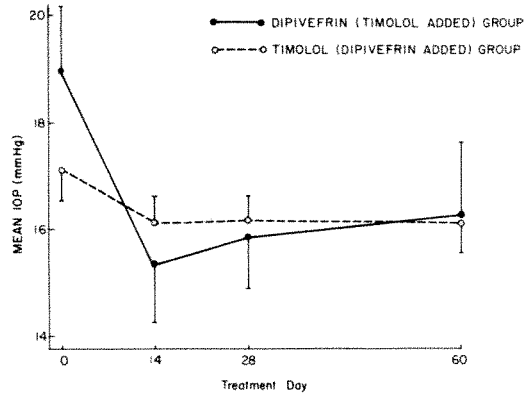


Figure (Keates and Stone). Intraocular pressure response in both study groups.

of visual acuity, and there were no objective findings on slit-lamp and ophthalmoscopic examinations to account for their symptoms. Nevertheless, three patients stopped taking dipivefrin and dropped out of the study because of these symptoms. The one patient who did show an objective worsening of visual acuity related to progressive lens changes experienced no subjective change during the course of this study.

Patients in Group 1 also reported a number of systemic side effects. One patient stopped taking dipivefrin because of headache on the second day of combined therapy. Two other patients described headaches occurring on three occasions but continued the medication. One patient reported a single 20-minute episode of dizziness. One patient gave a vague history of possible palpitations and reported a single episode of a bloody nose. This patient had a history of hypertension, was being treated systemically with propranolol and aldactazide, experienced an increase in pulse rate of 6 beats per minute, and, unlike most patients, an increase of 10 mm Hg in both systolic and diastolic blood pressure during the course of the study.

The patients in Group 2 reported significantly fewer side effects. One patient reported minor irritation when first using timolol, but this subsequently cleared up. One patient reported occasional twitching when timolol was initially added, but this also stopped soon thereafter. In neither instance were objective findings present. One patient in Group 2 also reported visual blurring when first using timolol, but there was no objective change in visual acuity. Slit-lamp and ophthalmoscopic examination disclosed no abnormalities. This patient's symptoms also subsequently cleared. No patients in this group discontinued the study.

Discussion

Dipivefrin, 0.1%,[3-5] and timolol, 0.25%,[8,9] both effectively decrease intraocular pressure. This study demonstrated that the combined use of these topical medications is safe and that they are more effective in combination.

Dipivefrin's high ocular penetration allows administration in lower concentrations than conventional epinephrine drops require. The well-known side effects of topical epinephrine include local side effects such as mydriasis, burning, hyperemia, follicular conjunctivitis, and brow ache, and also systemic side effects, such as cardiac arrhythmias and increases in blood pressure. Previous studies found that, although adverse reactions can occur,[11] dipivefrin is less likely than epinephrine to result in these epinephrine-like side effects[7] and that dipivefrin may be both effective and well tolerated in patients sensitive to topical epinephrine.[6]

In Group 1, the local subjective side effects were epinephrine-like in character and tended to be mild. Four of the 17 patients in this group, however, left the study because of local side effects, three because of visual blurring. Although visual acuity was unchanged and slit-lamp and ophthalmoscopic examinations showed no objective abnormalities, this subjective symptom may be related to the dipivefrin-induced mydriasis noted here and in previous studies.[3,7] The fourth patient stopped taking dipivefrin because of headaches.

There were also systemic side effects in Group 1. Although the group as a whole did not show a significant increase in pulse rate, one patient did show an increase in pulse rate of 14 beats per minute. This group showed significant decreases in both systolic and diastolic pressure. Minute systemic levels of epinephrine can cause depression of

blood pressure because the vasodilator β_2-receptors have a greater affinity for epinephrine than the vasoconstrictor α-receptors.[12] Presumably, such a mechanism accounts for the blood pressure decrease found here. Topical epinephrine also significantly lowers systolic and diastolic blood pressures.[13] These findings conflict with a previous report by Kaback and associates,[3] who found no change in pulse or blood pressure with topical dipivefrin in patients with ocular hypertension.

Group 1 showed a significant mean pupillary dilatation of 0.73 mm with combination therapy. Dipivefrin also causes modest but significant mydriasis.[3,7]

In Group 2, the subjective side effects reported by the patients were fewer and transient. No patient in this treatment group left the study. Timolol alone has no effect on pupillary diameter,[8] but instilling timolol in an eye receiving topical epinephrine can result in further significant mydriasis.[14,15] Although dipivefrin is administered in lower concentrations than epinephrine, the high ocular penetration of dipivefrin results in more comparable intraocular drug levels. Nevertheless, in Group 2, there were no significant changes in pupillary size.

Systemic side effects were also apparent in Group 2. This group showed a decrease in heart rate of 13 beats per minute compared with baseline. Pulse rates markedly decreased (28 to 30 beats per minute) in two patients. Mean systolic and diastolic blood pressures also decreased in this group, although only the diastolic decrease was statistically significant. Both of these findings are well-known systemic effects of β-adrenergic blocking agents and have been reported for topical timolol.[16]

Adding dipivefrin to timolol and adding timolol to dipivefrin caused a further significant decrease in intraocular pressure. In both groups, however, the effects on intraocular pressure were modest because the intraocular pressures of these patients were reasonably well controlled before the study began. In general, adding timolol to dipivefrin had a greater effect on intraocular pressure than adding dipivefrin to timolol. Similarly, adding timolol to epinephrine has a greater effect on intraocular pressure than adding epinephrine to timolol.[16] Although the mean decrease in intraocular pressure was smaller for patients in Group 1, some patients had clinically significant decreases of up to 6 mm Hg, suggesting that adding dipivefrin to timolol may be helpful in some patients. In Group 2, intraocular pressure appeared to increase on days 28 and 60. Although these changes were not statistically significant, they parallel the increase in intraocular pressure often seen after administration of timolol alone.[17] A crossover study assessing the short-term effects of the additional medication is needed to evaluate the mechanism of the interaction of dipivefrin and timolol.

These findings indicated that combined therapy with dipivefrin and timolol should be useful in treating patients with glaucoma; a further decrease in intraocular pressure by combination therapy, even in patients with reasonably well controlled intraocular pressures, is possible. Additionally, patients must be monitored for side effects, particularly for cardiovascular changes. Even in this small series, a substantial number of patients in both treatment groups developed clinically significant changes in pulse rate and blood pressures.

SUMMARY

Dipivefrin, an epinephrine prodromal drug, was used in conjunction with timolol maleate to evaluate the safety and effectiveness of combined therapy. Sev-

enteen patients who had used timolol maleate for at least six months added dipivefrin (Group 1) and seven patients who had used dipivefrin for at least six months added timolol maleate (Group 2). Intraocular pressure decreased further in both groups. Side effects were most prominent in Group 1.

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ENDOSCOPIC ORBITAL SURGERY

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Endoscopic orbital surgery is useful in evaluating trauma to the orbit, removing orbital foreign bodies, exploring fistulas, and removing tumor tissue. A new generation of small endoscopes, made possible by recent advances in wave-guide technology,[1,2] is essential to this surgery. We have used orbital endoscopy in 15 patients.

MATERIAL AND METHODS

There are two endoscopic techniques. In the first, the endoscope is held by hand, permitting quick scanning for simple procedures. In the second, the endoscope is mounted on a positioning device, allowing fine control of movement for bimanual surgical manipulation and prolonged observation.

Because even a small amount of bleeding makes observation difficult, a cannula placed over the endoscope delivers irrigating fluid to the optical tip. The cannula also contains a channel through which microsurgical instruments can be passed. These small instruments are handmade and have long shafts fitting the cannula. They include foreign-body and biopsy forceps and scissors.

Nine of the 15 procedures (Table) were observational, defining the characteristics of an orbital mass. In five cases we looked for orbital foreign bodies and in one case we performed a biopsy of tumor tissue.

We used the freehand technique in all procedures except the tumor biopsy. Examination of suspected tumors required a small incision deepened by blunt and sharp dissection. We approached intraconal lesions through the conjunctiva and extraconal lesions through the skin. We evaluated the tumors in terms of vascularity, color, contour, consistency, transillumination, invasiveness, and location. For the tumor biopsy, we used the bimanual technique, mounting the endoscope and cannula and freeing the hands for manipulation of instruments (Fig. 1).

Removal of retained orbital foreign bodies required a foreign-body microforceps and a cannula permitting their passage (Fig. 2). We introduced the endoscope into the entry wound and passed it through the tract until the retained material was located and extracted. A wound more than 72 hours old will not allow free passage. In a granulomatous orbital lesion with a fistula, dilation may be needed to introduce the endoscope into the fistula. Foreign-body material at the internal end of the fistula is located and extracted with the forceps or held in place while another route of extraction is prepared.

Irrigation in all cases was with isotonic saline controlled by a valve allowing just enough fluid to clear the view to enter the orbit. Illumination was provided by a fiberoptic bundle from a standard light box adapted to the endoscope's self-contained terminal-illuminating fiberoptic bundle.

We used local and retrobulbar anesthesia for short procedures. For the tumor biopsy requiring rigid instrument fixation, we used general anesthesia with curare.

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TABLE

RESULTS OF ENDOSCOPY IN 15 PATIENTS

Case No., Sex, Age (yrs)	Date of Surgery	Preoperative Diagnosis	Endoscopic Impression	Result
1, F, 56	June 22, 1977	Orbital mass	Well-outlined mass; low vascularity	Pseudotumor
2, M, 32	June 28, 1977	Metallic foreign body	Foreign body located	Foreign body removed
3, F, 82	Oct. 18, 1977	Fistula after scleral buckling	Episcleral sponge located	Sponge extracted
4, M, 51	Oct. 25, 1977	Fistula 11 yrs after retinal reattachment	Sponge located	Sponge removed
5, M, 16	Dec. 27, 1977	Vitreous hemorrhage, possible intraocular BB	BB found in orbit outside of eye	BB extracted
6, M, 2	Jan. 10, 1978	Retinoblastoma	No extrascleral extension	Confined to eye
7, M, 30	Jan. 23, 1978	Lacerated canaliculus requiring silicone tubing for repair	Guided silicone tube passage into nose.	Nasolacrimal drainage
8, F, 46	Feb. 2, 1978	Choroidal malignant melanoma	No transscleral extension	No transscleral tension
9, F, 21	June 9, 1978	Orbital floor fracture	Confirmed clinical impression	Orbital floor repair
10, M, 6 mos	July 14, 1978	Optic nerve glioma; blind spot	Solid pink intraconal mass	Optic nerve glioma
11, M, 72	Nov. 7, 1978	Orbital mass	Encapsulated mass	Pseudotumor
12, M, 1½	Nov. 25, 1978	Coats' disease or retinoblastoma	Abnormal vessels around optic nerve; nerve normal	Coats' disease and orbital vascular anomaly
13, M, 41	Jan. 2, 1979	Mucocele originating in frontal or ethmoid sinus	Well-defined yellow mass arising from ethmoid sinus	Mucocele removed by ethmoid sinus approach
14, M, 44	Jan. 5, 1979	Foreign body; double perforation of eye	Unable to locate foreign body in orbit	Retained orbital foreign body
15, F, 64	Feb. 16, 1979	Progressive proptosis; cavernous hemangioma	Vascular tumor; no biopsy	Cavernous hemangioma removed in toto

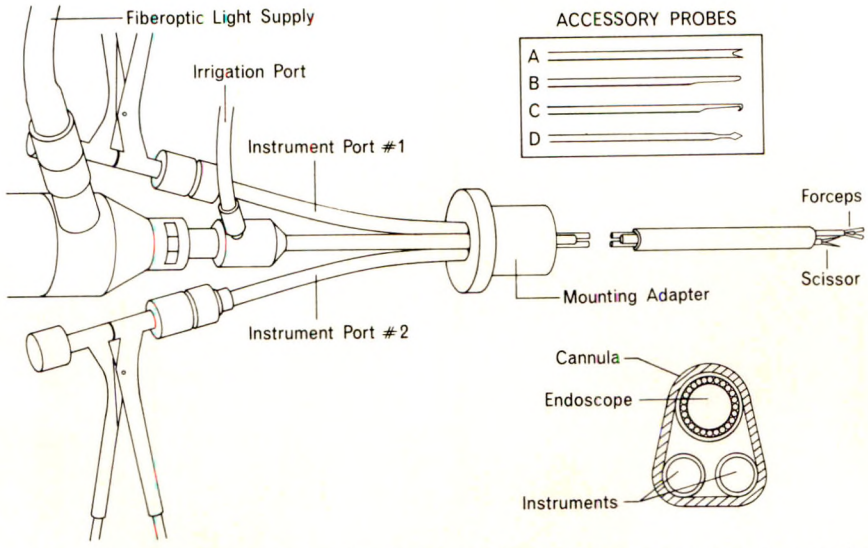


Fig. 1 (Norris and Cleasby). The bimanual biopsy technique requires a mounting adapter for fixed positioner and cannula for two instruments plus the endoscope.

Case report

Case 3—A 51-year-old man had undergone successful retinal reattachment surgery several years previously. The surgeon had used silicone sponge implants. A granulomatous reaction later necessitated removal of the sponge material. The patient was asymptomatic for more than one year. Then, a fistula formed and it was believed that residual sponge material might be present. The surgeon introduced the endoscope, with cannula and microforceps, into the fistula. At the internal end of the tract, surrounded by yellow exudate, there was a piece of sponge. The surgeon grasped it with the forceps but it was too friable to withdraw through the tract. He used the forceps to hold it in place while making a separate entrance for extraction.

Case 11—A 72-year-old man had had a swelling in

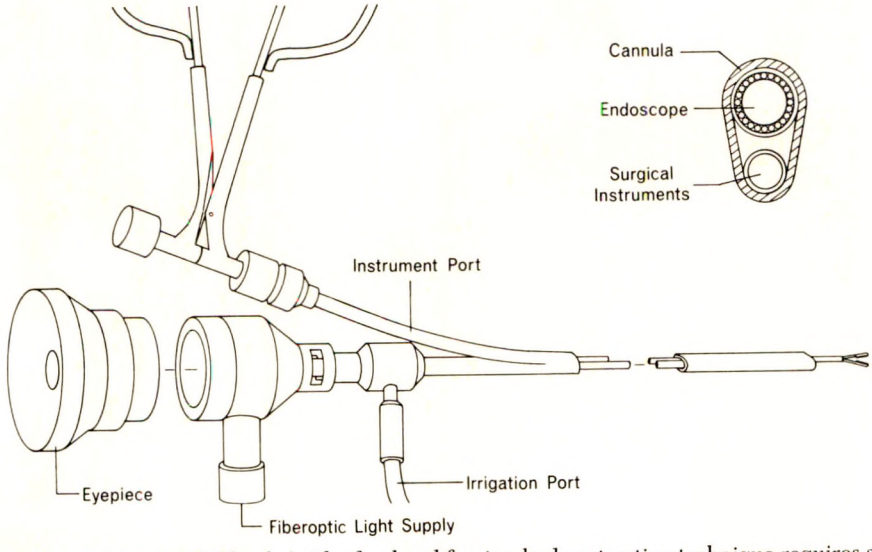


Fig. 2 (Norris and Cleasby). The freehand foreign-body extraction technique requires a cannula for the endoscope and foreign-body forceps.

the right upper eyelid for 18 months. He had a palpable mass beneath the superior orbital rim. The mass was firm, nontender, and slightly movable. X-rays of the orbit showed no bone destruction; the sinuses were normal. A- and B-scan ultrasonograms demonstrated a mass of low reflectivity with occasional high spikes. A computed axial tomographic scan demonstrated a soft tissue abnormality which did not enhance with injection. The endoscope clearly showed the tumor which was visible through a small incision beneath the nasal brow. The tumor appeared to be encapsulated and was not highly vascular. The extent of the lesion and its vascular supply was easily defined. The surgeon removed the tumor tissue with the biopsy forceps and scissors through a mounted operating cannula fitted over the endoscope (Fig. 1). He then decided to enlarge the wound and remove the remainder of the tissue. He verified the completeness of removal by endoscopy. The pathologic diagnosis was lymphoid pseudotumor. In retrospect, we had obtained a satisfactory biopsy specimen for histologic diagnosis with the endoscope and further surgery was not necessary.

RESULTS

We located and extracted four of five orbital foreign bodies (Cases 2 to 5) with the endoscope. One foreign body (Case 14) could not be located. We observed and classified four orbital masses (Cases 1, 10, 13, and 15). Two patients with large intraocular malignancies (Cases 6 and 8) underwent orbital endoscopy immediately before enucleation to establish extrascleral extension. We found no evidence of extension, a finding consistent with later histologic studies. Endoscopy was useful in confirming orbital floor fracture (Case 9) and in nasolacrimal duct reconstruction (Case 7) when the usual technique of intranasal probe location had failed. Endoscopic biopsy with bimanual technique yielded a specimen adequate for pathologic diagnosis (Case 11). There were no complications with the endoscope.

DISCUSSION

Our experience with the endoscope demonstrated that it is a useful way of removing retained orbital foreign bodies. It is easier to locate the foreign material and there is less surgical trauma during the extraction, particularly with posterior lesions. Operating time is also reduced. The value of endoscopy with other situations is not as clearly defined. Too few tumors have been examined endoscopically to establish reliable diagnostic criteria. In one of our cases, endoscopic biopsy provided a correct diagnosis.

Endoscopic procedures in the posterior orbit have several potential problems: injury to adjacent structures, bleeding, and overfilling from irrigation. There is no satisfactory method at present of controlling deep orbital bleeding, should it occur, although we did not encounter this problem because we avoided vascular lesions. The infusion of irrigating fluid also creates pressure within the orbit. The fluid is absorbed directly into the tissues and they rapidly become edematous. Using air in place of saline was not successful.

SUMMARY

Orbital endoscopy is useful in managing various types of orbital problems. A hand-held endoscope can be used for simple procedures. Surgery and prolonged observation require a mounted endoscope. These two techniques provided good results in 15 patients. Endoscopy proved useful in finding and removing orbital foreign bodies. It is potentially useful in the management of orbital tumors, but experience in this area is still limited.

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PROLAPSE OF THE GLOBE INTO THE MAXILLARY SINUS AFTER ORBITAL FLOOR FRACTURE

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A blow-out fracture is a fracture of the orbital wall without involvement of the orbital rim.[1] Subsequently, dislocation of orbital contents into the adjacent sinuses with permanent entrapment of orbital tissue can occur in various degrees.

We studied an unusual case in which the entire globe prolapsed into the superior portion of the maxillary sinus after a blow-out fracture. We documented this radiographically by computed tomography and by orbital tomograms. To our knowledge, there has been only one previously documented case with similar findings.[1]

CASE REPORT

The left eye of a 24-year-old woman sustained a direct blow from a fist. That day the patient was referred from another hospital with the diagnosis of orbital floor fracture of the left eye. The primary physician was unable to find the globe within the orbit after double-everting the patient's eyelids with Desmarres retractors (Fig. 1). The patient had no light perception in the ocular cul-de-sac. Light perception was noted only when the transilluminator was placed into the orbit and directed into the maxillary sinus.

Ultrasonographic studies of the orbit and sinus confirmed the presence of a normal-appearing globe, optically clear vitreous, and an attached retina. Orbital tomograms of the left orbit (Fig. 2) and computed tomograms (Fig. 3) disclosed a blow-out fracture and an air-fluid level in the left maxillary sinus. The globe was displaced inferiorly and posteriorly into the superior portion of the maxillary sinus. Instead of the increased radio-opacity of the left orbit expected from swelling, radiolucent studies indicated loss or displacement of the orbital contents into the upper maxillary sinus (Fig. 2).

The following day, the orbital floor fracture was repaired via a Caldwell-Luc and infraorbital approach. Only the nasal and lateral one eighth of the floor was intact. The globe was found within the maxillary sinus. We elevated the globe back into the orbit with a finger, and secured it in the orbit by packing the maxillary sinus with iodoform gauze through a Caldwell-Luc approach. We inserted a square silicone implant, carved with a No. 15 Bard-Parker blade (from a silicone block) and measuring 23 × 23 mm. The thickness was adjusted to 2.5 mm anteriorly and 4 mm posteriorly. We used a dental drill to bore two holes through the orbital rim, and secured the implant with 2-0 synthetic polyfilament sutures.

Ten days postoperatively, the patient developed a maxillary sinus infection, which was successfully treated with antibiotics. Her visual acuity returned to 6/6 (20/20) despite a 2 + afferent Marcus Gunn pupil. The motility was limited to less than 5 degrees in abduction or adduction and less than 2 degrees in supraduction or infraduction. Fluorescein angiography, performed 15 days postoperatively, demonstrated hyperfluorescence and leakage of dye from the disk of the left eye, probably secondary to optic nerve inflammation. Computed tomography performed postoperatively showed that the globe was normally located within the orbit (Fig. 4).

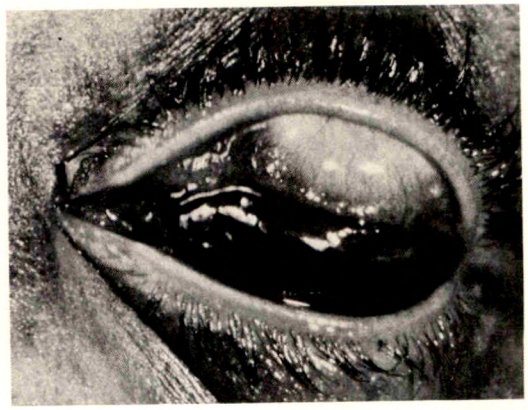


Fig. 1 (Berkowitz, Putterman, and Patel). Preoperatively, the globe was absent from the left ocular cul-de-sac.

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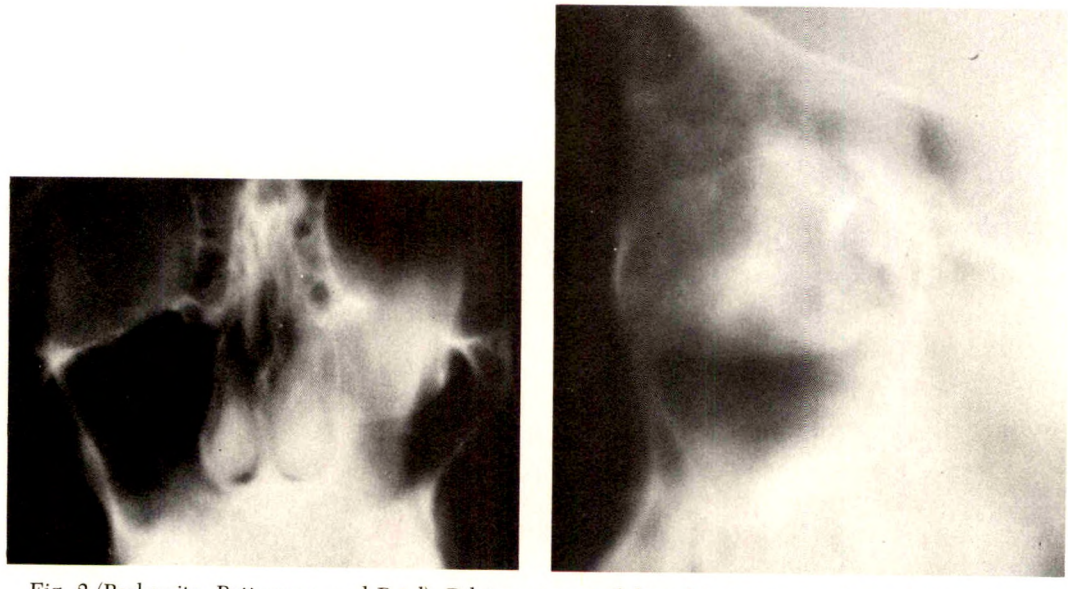


Fig. 2 (Berkowitz, Putterman, and Patel). Polytomograms of the orbit in anteroposterior (left) and lateral (right) projections. Fracture of the floor shows a soft tissue mass occupying the upper half of the maxillary sinus. Note radiolucency of the left orbit instead of the increased opacity expected in swollen eyes. This finding suggests that most of the orbital contents are displaced into the maxillary sinus.

Ten months postoperatively, the patient's visual acuity was 6/6 (20/20), and her visual field was normal. There was no enophthalmos or blepharoptosis and the eyes were horizontally level (Fig. 5). There was no limitation of horizontal ductions, and single binocular vision was present on primary and horizontal gazes. She was able to elevate her eye 20 degrees and depress it 5 degrees. The generated muscle force test showed that only minimal force was available to move the eye downward, suggesting paresis of the inferior rectus muscle.[2] The forced duction test was slightly positive on upgaze. There was an increase in intraocular pressure on upgaze (28 mm Hg) compared with primary gaze and downgaze (8 to 10 mm Hg). (This indicated that the inferior rectus muscle was probably paralyzed and that there was some restriction in the inferior rectus muscle from secondary contracture or entrapment of tissue. As the patient attempted to gaze upward, the inferior rectus muscle pressed on the globe and increased the intraocular tension.[2] We believed that the nerve to the inferior rectus muscle was severed against the floor of the orbit or severely traumatized as the globe entered the maxillary sinus.)

Discussion

When an eye is struck forcibly by a large blunt object, such as a fist, rock, or ball, the globe, as a rule, does not rupture; instead, a blow-out fracture develops.[3] A blow-out fracture acts like a pressure valve in that the force of the blow to the eye, besides being absorbed by the elastic ligaments of the eye and orbital fat, is also absorbed by the bony rupture and by compression of air in the paranasal sinuses.[4]

The globe is suspended and supported within the orbit by a combination of the bulbar fascia (Tenon's capsule), muscles, ligaments, and orbital fat. The lower part of the bulbar fascia is thickened to form a sling or hammock, also known as the suspensory ligament of Lockwood, on which the globe rests. The bulbar fascia is a thin fibrous membrane that envelops the globe from the corneoscleral limbus to the optic nerve. The bulbar fascia is pierced by the tendons of the extrinsic muscles. These muscles in turn send out fibrous expansions, called check ligaments, to the orbital bones. The fibrous expansion of the lateral rectus muscle attaches to the orbital tubercle on the

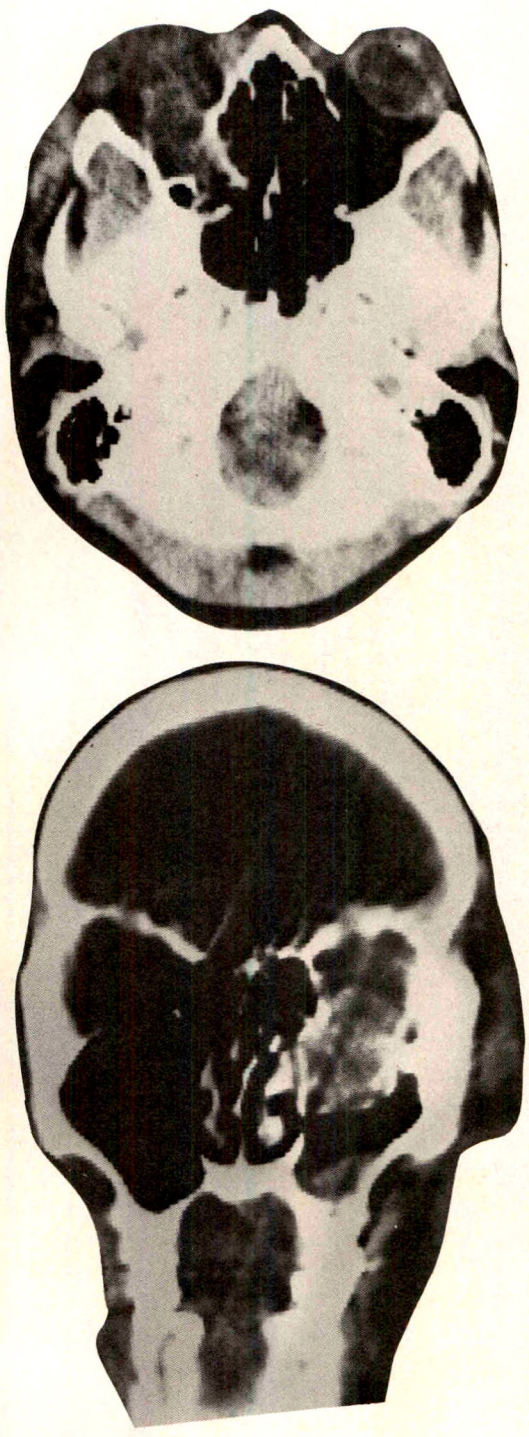


Fig. 3 (Berkowitz, Putterman, and Patel). Computed tomographic sections obtained in an axial plane at the level of the orbits (top left) and at the level of the maxillary sinus (top right) and in a coronal plane (bottom left). A soft tissue mass (the globe) occupies the upper half of the maxillary sinus. There is also distortion of soft tissues, but no proptosis.

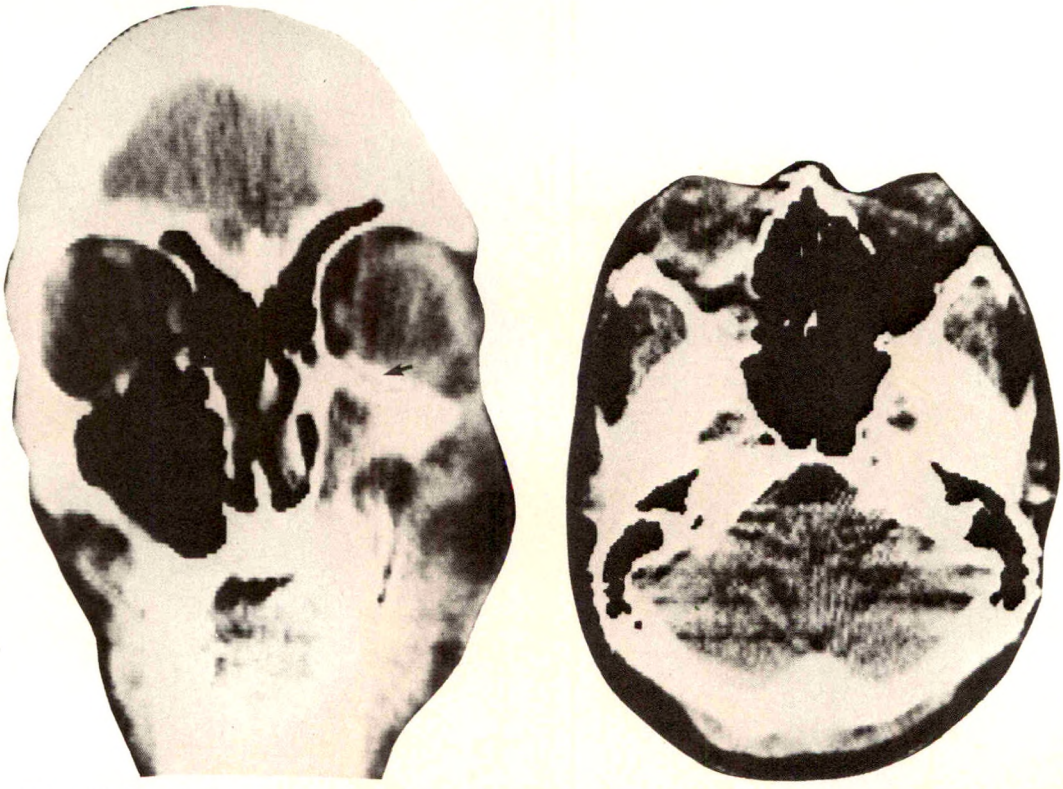


Fig. 4 (Berkowitz, Putterman and Patel). Left, Postoperatively, coronal section of the orbits shows normal location of the left globe with resolving changes in the left maxillary sinus and deformity of the fracture. (Arrow denotes silicone implant). Right, Axial plane shows the left globe within the orbit and part of the silicone implant beneath the globe.

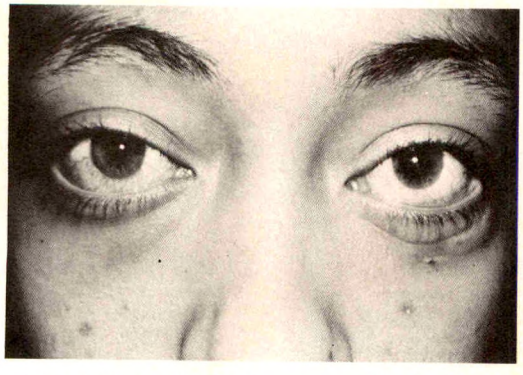


Fig. 5 (Berkowitz, Putterman, and Patel). Postoperatively, eyes were horizontally level without enophthalmos.

zygomatic bone, and the medial rectus muscle attaches to the lacrimal bone.[5] The globe is also suspended from the trochlea of the frontal bone by way of the superior oblique tendon.

The globe does not sink significantly after removal of the maxilla and orbital floor,[5,6] demonstrating that these structures are able to support the globe. Many of these attachments must have been ruptured or severely damaged before the globe could be displaced so far inferiorly in our case.

Summary

A 24-year-old woman sustained a blow-out fracture of the orbital floor with

prolapse of the globe into the maxillary sinus. This was treated by replacing the globe in the orbit surgically and reconstructing the orbital floor. Postoperatively, the eye retained normal visual acuity.

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TESTICULAR SEMINOMA METASTATIC TO THE ORBIT

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Bilateral exophthalmos, possibly caused by hormonal influence, has been documented in three patients with localized seminoma[1-3] but, to our knowledge, no case of orbital seminoma metastatic from the testis has been previously documented. Three previously described cases of orbital tumor metastatic from the testes[4,5] were choriocarcinomas rather than the less aggressive testicular neoplasm, the seminoma. We studied a case of testicular seminoma metastatic to the orbit in a patient whose ocular symptoms went into remission after orbital irradiation.

CASE REPORT

A 28-year-old man was seen on March 12, 1980, for evaluation of painful exophthalmos of the right eye of four months' duration. He had been well until June 1979, when a seminoma of the right testis, which had been present for 18 months, was removed by radical scrotal orchiectomy.

Preoperative and postoperative serum values for human chorionic gonadotropin, beta subunit, were 49 and 21 mU/ml, respectively (normal for males, 0 mU/ml). Radiation therapy was completed and he was well until November 1979, when transient right periorbital pain and eyelid swelling suddenly developed. Two weeks later diplopia and proptosis occurred; in February 1980 he noted blurred vision in his right eye.

On March 12, 1980, his visual acuity was R.E.: 6/24 (20/80) and L.E.: 6/6 (20/20). The right eye was proptotic by 5 mm (Hertel exophthalmometer at base 106, R.E.: 21 mm and L.E.: 16 mm) and was displaced 10 mm inferiorly. No orbital mass was palpable superiorly. There was partial ophthalmoplegia of the right eye but no blepharoptosis or chemosis. The pupils were normal. The right optic disk was elevated, the posterior pole was indented, and choroidal striae were present. The left eye was normal. Serum T_3 and T_4 values and chest roentgenograms were normal. A computed axial tomographic scan of the right orbit showed a diffused density. He began a three-week course of prednisone, with the dosage gradually decreased, on March 15, 1980.

On April 14, 1980, visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/6 (20/20). An afferent pupillary defect was present in the right eye and the optic disk was edematous. A mass was palpable through the upper eyelid. A computed axial tomographic scan showed a discrete mass in the right superior orbit. On April 15, 1980, a metastatic seminoma was found during an anterior orbitotomy and biopsy (Figure). Postoperatively, 3,000 rads of radioactive cobalt (^{60}Co) was delivered to the right orbit. On May 14, 1980, visual acuity was R.E.: 6/12 (20/40). The optic neuropathy was improved but the choroidal striae persisted.

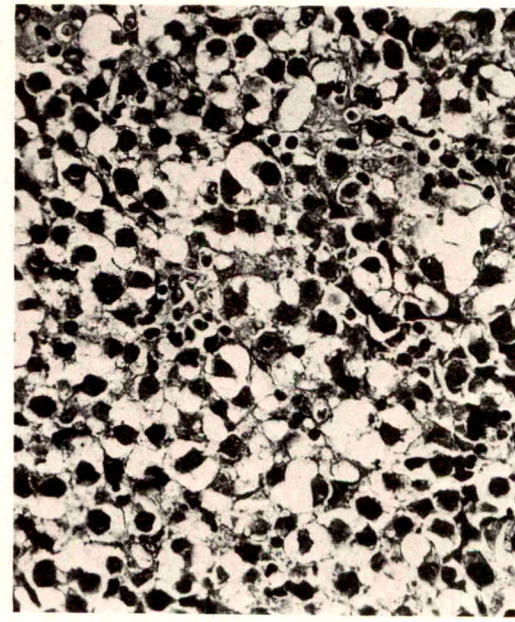


Figure (Rush, Older, and Richman). Photomicrograph of the metastasis of the right orbit, showing sheets of malignant cells with large nuclei, prominent nucleoli, and a mild degree of nuclear anaplasia. There are a few mitoses. Most tumor cells have clear cytoplasm with well-demarcated cytoplasmic borders. Small lymphocytes are interspersed among the tumor cells. This pattern was typical for pure seminoma, and no histologic features of any other germ cell neoplasm were seen (hematoxylin and eosin, × 340).

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Discussion

Neoplasms of the testes constitute only 1% of all malignant tumors in men but are the most common malignancies affecting men 29 to 35 years of age.[6] Malignant testicular tumors include seminoma, embryonal carcinoma, teratocarcinoma, and choriocarcinoma. Each usually occurs as a single cell type.[7] Almost one third of all cases are pure seminomas.[8] The nonseminomatous neoplasms of the testes are invasive tumors. Orbital metastases from testicular choriocarcinomas have been reported.[4,5] Testicular seminomas, however, are sensitive to radiation therapy, and patients treated with orchiectomy and lymph node irradiation have a survival rate of more than 95%. They are unlikely to have orbital metastases.

Bilateral exophthalmos without orbital metastases has been reported in three middle-aged patients with localized seminoma.[1-3] In two of these patients, symmetrical, painless exophthalmos and restricted ocular motility were the initial manifestations of a testicular seminoma. Conjunctival chemosis was striking, and thyroid exophthalmos was suspected, but serum T_3 and T_4 values were normal. The ocular symptoms went into remission in one patient who was treated with prednisone,[2] and permanent resolution of the exophthalmos and chemosis occurred in both patients after orchiectomy. The authors thought a remote hormonal effect of the testicular neoplasm caused the proptosis in these patients.[1,2] A third patient with seminoma who had bilateral exophthalmos, presumably of hormonal origin, had a mediastinal seminoma at autopsy examination, but no orbital tumor.[3]

The bilateral orbital involvement in these cases differed from the unilateral orbital symptoms in our patient, in whom pain, diplopia, and proptosis without chemosis occurred. More than 90% of cases of metastatic orbital carcinoma are unilateral; the primary symptoms are pain and diplopia.[9] Our patient's symptoms resembled those caused by metastatic orbital carcinoma and were different from those caused by bilateral, congestive proptosis of presumed endocrine influence reported in other patients who had localized seminomas.[1-3]

Less than 15% of all seminomas produce distant metastases[10]; these usually metastasize to lymph nodes, bones, and lung.[11] Because our patient had not had the mass in his right testicle treated immediately, the diagnosis of the primary tumor was delayed, allowing tumor cells to seed his orbit before the delivery of abdominal irradiation. Henderson[9] noted the significance of orbital symptoms developing in patients who have carcinoma; we emphasize that the orbit can also be the first site of disseminated seminoma.

The prognosis of seminomas is ultimately related to the cellular constituents of the tumor. One subgroup, the trophoblastic seminoma, which contains syncytiotrophoblastic cells that secrete human chorionic gonadotropin, is less sensitive to radiation therapy than the typical seminoma and may develop into the more aggressive choriocarcinoma.[12] Although the histologic findings of the seminoma in our patient were those of a typical seminoma, we found increased serum levels of human chorionic gonadotropin, beta subunit. This may indicate a poor prognosis.

Thirty percent of all seminomas metastasize as a different histologic type, presumably because of unknown factors at metastatic sites that act upon persistent stem cells and influence cellular differentiation.[12] The orbital biopsy in our patient did not show this evolution, however, and reports of good results with radiation therapy[11] and chemotherapy[13] in cases of pure metastatic seminoma

suggest a favorable outcome for our patient.

Summary

Painful exophthalmos, ophthalmoplegia, and visual loss in the right eye developed in a 28-year-old man four months after he underwent a radical orchiectomy and irradiation for a seminoma of the right testicle. Prednisone therapy failed to relieve the symptoms, and an orbital biopsy found a metastatic testicular seminoma. Orbital irradiation with radioactive cobalt reduced the exophthalmos, reversed the ophthalmoplegia, and improved his visual acuity. Previous cases of nonmetastatic exophthalmos in patients who had testicular seminoma have been reported, but, to the best of our knowledge, this is the first report of a patient who had orbital seminoma metastatic from the testis. Successful irradiation of the orbital focus can minimize ocular morbidity and may improve the prognosis.

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NOTES, CASES AND INSTRUMENTS

SUBSTITUTING THE ALCOHOL SWAB FOR THE ALCOHOL LAMP

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AND

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Corneal scrapings for microbiologic investigation of presumed bacterial or fungal keratitis are commonly performed with a Kimura platinum spatula. Appropriate technique requires flame sterilization of the spatula's tip between innoculations of different media. An alcohol lamp is generally recommended for this purpose.

On occasion an alcohol lamp may be either inaccessible or nonfunctional. There is another method for thermal disinfection that requires only a packaged alcohol swab and a blade.

Slits are made in the middle of the package through which the swab is tented to form a wick. Lighting the wick produces a flame that lasts approximately 2½ to three minutes and sterilizes the spatula (Figure).

This alternative makes unnecessary systems, such as the battery-powered cautery,[1] which do not have the more appropriate spatula proposed by Kimura.

SUMMARY

The alcohol swab is a very reliable and easily obtained substitute for the alcohol lamp in performing a routine culture.

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Figure (Salisbury, Cobo, and Brown). Alcohol swab sterilizer.

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A CASE OF SYPHILITIC UVEITIS

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At one time syphilis was second only to tuberculosis as a cause of uveitis,[1] but it

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became relatively rare with the advent of penicillin.[2,3] Between the years 1955 and 1979 there was a fourfold increase in the incidence of syphilis in the general population[4] and the opthalmologist must again be mindful of this disease in any case of progressive or severe uveitis. In 1977 alone more than 20,000 cases of primary and secondary syphilis were reported in the United States.[4] We studied a case of syphilis that first appeared as anterior uveitis and then progressed to panuveitis, neuroretinitis, and optic atrophy.

Case report

A 38-year-old man consulted an ophthalmologist because he had had an acutely red right eye for 24 hours. The eye had ached for four days. Uncorrected visual acuity was R.E.: 6/6-3 (20/20-3) and L.E.: 6/6 (20/20). The eyes appeared normal except for episcleral injection inferonasally in the right eye. He was treated with a topical vasoconstrictor and told to return in two weeks if his symptoms had not improved. Two weeks later, the patient complained of increased pain in and around the eye, and visual acuity was 6/22 (20/70) in the right eye. The episclera and conjunctiva were 3+ injected, and a slit-lamp examination disclosed 2+ cells and flare in the anterior chamber. No keratic precipitates were present. The patient was treated for iritis with topical corticosteroids and mydriatics. During the following weeks the right eye continued to deteriorate despite increasingly aggressive therapy with topical and systemic corticosteroids and cycloplegic agents. Visual acuity decreased to 6/60 (20/200), and the uveitis progressed to involve the vitreous. Ophthalmoscopy then disclosed a possible exudative retinal detachment in the posterior pole. There was an afferent pupillary defect and posterior synechiae were present. Because of these findings, the patient was referred here for consultation.

Visual acuity in the right eye was limited to counting fingers at 4 feet; it was 6/6 (20/20) in the left eye. Slit-lamp examination of the right eye showed a hyperemic conjunctiva, small keratitic precipitates, 4+ flare and cells, and posterior synechiae from the 3 to 7 o'clock positions. The vitreous humor was diffusely hazy with inflammatory cells, making examination of the fundus difficult. The optic disk was pale and edematous with blurred margins. The retina, especially in the posterior pole, was markedly edematous and showed scattered yellow-white exudates, many of which were along the vessels. On fluorescein angioscopy, we saw staining of the involved subretinal space and hyperfluorescence of the retinal lesions. The left eye was normal. The possibility of a parasitic infection was discussed with the patient and a toxoplasmosis fluorescent antibody test, a VDRL test, and a fluorescent treponemal antibody-absorption (FTA-ABS) test were done.

One week later the patient returned and admitted that because of the possibility of a parasitic infection, he sought and received treatment for syphilis at a local venereal disease clinic. The VDRL and FTA-ABS laboratory tests were positive. In response to questioning, he said that he had a heterosexual contact six to eight weeks previously in which the disease may have been contracted, but he was not certain. He refused a lumbar puncture; there were no signs of secondary syphilis. The patient was advised to continue his penicillin therapy.

Two weeks later his vision had improved to 6/9 (20/30) in the right eye, but he complained of "tunnel vision." Goldmann visual fields were contricted to 15 degrees in the right eye. The optic disk was pale and fluorescein angiography confirmed the presence of optic nerve atrophy.

Discussion

Although syphilis is notorious for its ability to mimic any inflammatory disease of the eye, the most common clinical signs are uveitis, diffuse chorioretinitis, and vasculitis.[2]

Our patient had iridocyclitis but his ocular disease progressed in the absence of treatment to periphlebitis, disseminated chorioretinitis, and optic neuritis. This characteristic triad of findings has been described as neuroretinitis papulosa.[5] The patient was finally treated with penicillin, but he suffered secondary optic nerve atrophy.

Although the standard ophthalmologic textbooks list syphilis in the differential diagnosis of uveitis, our experience in an active referral practice shows that many physicians do not order laboratory studies to find the cause of uveitis but instead prefer to use an empirical approach, probably because the cause of most cases of uveitis remains unknown. However, as our case and others similar to it have demonstrated, iridocyclitis and a red eye may signal the presence of a severe systemic disease process and may have dire consequences if not recognized and treated in time.

Although the search for an undetected systemic disease associated with the uveitis is frequently unsuccessful, a selected uveitis survey should be obtained. A detailed history is the most important

element in making the diagnosis. Some of the tests that we have found useful in evaluating selected patients with uveitis include the following: (1) complete blood count and urinalysis; (2) chest X-ray and angiotensin-coverting enzyme and serum lysozyme tests to detect sarcoidosis; (3) Sabin-Feldman dye test to rule out toxoplasmosis; (4) VDRL and FTA-ABS tests to detect syphilis; (5) purified protein derivative skin test to detect possible tuberculosis; (6) rheumatoid factor, antinuclear antibody, and histocompatibility antigen (HLA-B27) as screening tests for possible rheumatoid arthritis; (7) enzyme-linked immunosorbent assay to detect ocular *Toxocara canis*.

The current treatment of acquired syphilis of less than one year's duration consists of benzathine penicillin G, 2.4 million units given as an initial intramuscular dose, followed by either 1.2 million units of benzathine penicillin G or 600,000 units of procaine penicillin G intramuscularly daily for eight days.[6]

Summary

A 38-year-old man had anterior uveitis that failed to respond to increasingly aggressive therapy with topical and systemic corticosteroids and mydriatrics. His disease progressed to panuveitis and neuroretinitis and was finally cured with penicillin injections for acquired syphilis. He suffered secondary optic nerve atrophy.

Because of the resurgence of the incidence of syphilis in the general population and the dire consequences for the patient in the absence of appropriate therapy, the ophthalmologist needs to consider the possibility of syphilis in his patients with uveitis. He should obtain serologic studies for syphilis.

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A NEW TECHNIQUE FOR PORTABLE GONIOSCOPY

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We have devised a new technique for portable, low-power gonioscopic examination of the anterior chamber angle structures. This technique has proven useful when a slit lamp has not been available or usable, as in bedside examinations, nursing home consultations, and in examinations of infants, restless patients, and patients with severe cervical arthritis. We used this technique when examining some bedridden patients with markedly increased intraocular pressure and were able to distinguish between closed- and open-angle glaucoma, and thus able to initiate appropriate therapy.

We use a pair of magnifying loupes and a halogen-streak retinoscope focused on the anterior chamber angle which is seen through a gonioscopic lens. This gives a clear view of the angle (Figs. 1 and 2).

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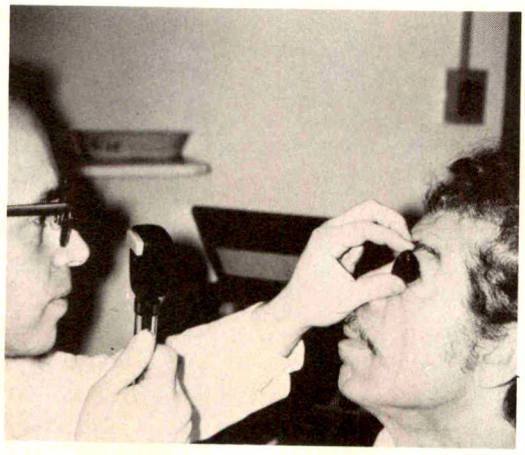


Fig. 1 (Perlstein and Galst). Examiner wears ×4 loupes and focuses a narrow streak retinoscopic beam on the anterior chamber angle structures through the Goldmann gonioscopic lens.

The halogen-streak retinoscope is an excellent source of illumination that can be focused to provide a narrow, bright beam of light. For magnification, we have found a set of ×4 binocular loupes convenient, because they provide a steady stereoscopic view while freeing the examiner's hands for holding the retinoscope and stablizing the gonioscopic lens. A direct ophthalmoscope set at +15 or +20 diopters can also be used for both illumination and magnification, but it provides neither a stereoscopic view nor a focusable slit beam for enhanced observation. When patients are seated, we use the Goldmann or Zeiss gonioscopic lens; when patients are lying down, we use the Koeppe lens.



Fig. 2 (Perlstein and Galst). Unmagnified view of anterior chamber angle obtained with a focused streak retinoscopic beam for illumination.

Summary

We have developed a new technique for low-power gonioscopic examination of the anterior chamber angle. We use ×4 binocular loupes for magnification and a portable halogen-streak retinoscope as the light source focused on a gonioscopic lens.

SLIT-LAMP EXAMINATION OF THE BEDRIDDEN PATIENT

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Slit-lamp examination generally requires the patient to sit with his or her chin on the headrest. However, it is hard to perform biomicroscopy on patients who are unable to sit or move to the slit lamp, such as bedridden patients with severe arthritis, paralysis, or stroke.

For examining patients whose weakness or paralysis is confined to the lower half of the body, we recommend the use of a stretcher. The patient lies prone, elevates the upper body on the arms or on pillows, and holds the head erect. The ophthalmologist then maneuvers the stretcher and the height of the slit lamp

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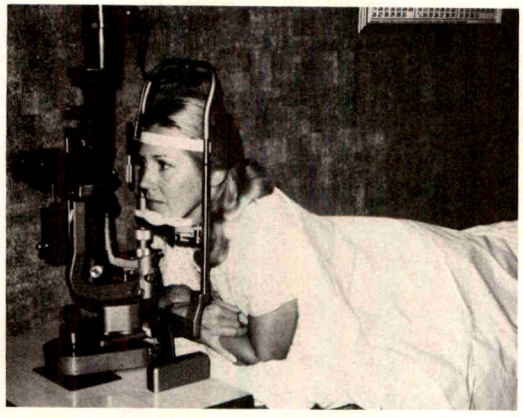


Figure (Partamian and associates). The patient lies prone, elevates the upper body on the arms, and holds the head erect. The ophthalmologist then maneuvers the stretcher and the height of the slit lamp so that the patient's head is in the headrest.

so that the patient's head is in the headrest (Figure).

This method is simple and useful for examining bedridden hospital patients sent for ophthalmologic consultation.

Summary

Slit-lamp examination of bedridden patients is facilitated by using a stretcher with the patient lying prone and holding the head erect.

CONTRAINDICATIONS FOR MANNITOL IN APHAKIC GLAUCOMA

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Ophthalmologists use mannitol to decrease intraocular pressure in angle-closure glaucoma. Mannitol can produce potentially dangerous complications, however. In rare instances, these may be fatal.

Case report

A 72-year-old woman with diabetes mellitus, vascular hypertension, and mild renal insufficiency was hospitalized after the sudden onset of pain in her right eye. She had undergone cataract surgery on the same eye 13 months previously. Because of diabetic nephropathy and progressive azotemia, an arterial venous fistula had been created in her right forearm to prepare for eventual hemodialysis. Despite her nephropathy, urine output exceeded 1,000 ml/day. Laboratory tests done at the time of admission gave the following values: serum sodium, 128 mEq/liter; serum potassium, 4.7 mEq/liter; serum chloride, 102 mEq/liter; serum carbon dioxide content, 15 mmoles/liter; blood urea nitrogen, 36 mg/100 ml; and serum creatinine, 2.6 mg/100 ml.

Visual acuity was no light perception in the right eye and 6/30 (20/100) in the left eye. Intraocular pressure by Goldmann applanation tonometry was R.E. 80 mm Hg and L.E.: 14 mm Hg. The right eyelids were swollen. The conjunctiva was swollen and injected, and there was microcystic edema of the corneal epithelium. The right eye was aphakic and showed rubeosis iridis, an occluded pupil, a small peripheral iridectomy (possibly patent), a shallow anterior chamber caused by secondary pupillary block, and a partial hyphema. The fundus could not be seen by ophthalmoscopy. The left eye was normal, except for a cataract consistent with a visual acuity of 6/30 (20/100).

During the ensuing 60 hours, the patient received 1,300 ml of 20% mannitol but there was only a modest decrease in intraocular pressure. Anuria developed and led to severe pulmonary edema with accompanying confusion. Serum sodium concentration fell to 99 mEq/liter and serum creatinine rose to 5.2 mg/100 ml (Table). Despite intensive hemodialysis, the pulmonary edema, acidemia, and anuria persisted, and the patient died. Serum sodium concentration at the time of death was 112 mEq/liter.

Discussion

Mannitol is a six-carbon alcohol derivative of mannose and has a molecular weight similar to that of glucose. It disperses uniformly in the extracellular space, is inert, and undergoes little metabolic change.[1] When renal function is normal, the half-life of mannitol in the circulation is 15 minutes, and 90% of an injected dose is excreted in 24 hours. If renal insufficiency develops, the half-life

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TABLE

CLINICAL FINDINGS AFTER MANNITOL INFUSION

Finding	Day 1	2	3	4
Amount of 20% mannitol administered (ml)	0	300	1,000	0
Serum sodium (mEq/liter)	128	114	102	99
Serum creatinine (mEq/liter)	2.6	2.6	3.7	5.1
Urine output (ml)	1,100	700	0	0

of mannitol increases, and the drug is retained in the extracellular space because of diminished excretion. Infusion of mannitol creates an increased extracellular tonicity and results in transport of water out of cells and into the extracellular space. Although mannitol may act as a water diuretic when renal function is normal, and thus induce hypernatremia,[2] its initial osmotic expansion of extracellular volume lowers the sodium concentration in that compartment. When decreased renal function precludes the removal of free water, the hyponatremia[3] may be profound and lead to lethargy, obtundation, seizures, and coma, as happened in this patient.

Unlike most other instances of hyponatremia when tonicity also falls, this situation may be accompanied by paradoxical hypertonicity. Ordinarily plasma osmolality is maintained within a rigidly defined range and perturbations of tonicity in either direction are associated with changes in mentation. In general, the greater the deviation from a normal osmolality, the more pronounced the mental changes become. Plasma osmolality is increased when mannitol is infused and continues when the substance is retained in the extracellular space.[4] It is this property that leads to the beneficial effect of mannitol in decreasing intracranial pressure and brain edema.[5] Nevertheless, continued infusion in the presence of anuria leads to a rising concentration of mannitol, and plasma osmolality remains increased despite the decrease in intracranial pressure and resulting hyponatremia. Thus, a sustained hyperosmolar state can supervene, as occurred in this case, when the serum osmolality was calculated at 340 mmoles/kg of water. Therefore, both hyponatremia and hyperosmolality may have contributed to the progressive neurologic deterioration.

Furthermore, acidosis affecting cerebrospinal fluid pH has been associated with delirium, obtundation, and coma.[6] Silber and Thompson[7] suggested that there is a connection between retained extracellular mannitol and cerebrospinal fluid pH. When dogs with renal insufficiency were made acidotic but not given mannitol, cerebrospinal fluid pH remained normal. When mannitol was administered intravenously to these same animals, cerebrospinal fluid acidosis developed and paralleled the level of acidemia. It is possible, therefore, that the persistence of mannitol in this patient may have precipitated cerebrospinal fluid acidosis and contributed in that way to neurologic deterioration.

Additionally, mannitol may have contributed to the progressive renal failure in this patient. Although hypertonic mannitol administered in low concentration generally produces no change in glomerular filtration rate, higher concentrations can induce a decline in both glomerular filtration rate and renal blood flow.[8] The

rapid development of anuria may have been partially related to an adverse effect of high concentrations of mannitol in the blood.

Once toxicity is established in the presence of renal insufficiency, extracorporeal hemodialysis is the best therapy.[4] This treatment promotes the rapid removal of mannitol from the extracellular space, returns plasma osmolality to normal, and corrects hyponatremia. Peritoneal dialysis has also been used, but in some cases[1] this has been associated with a paradoxical increase in plasma osmolality and further derangement of neurologic status.

SUMMARY

When prescribing mannitol to decrease intraocular pressure, the physician must be alert to potential complications. A 72-year-old woman suffered obtundation, intractable pulmonary edema, acidemia, and irreversible renal insufficiency despite vigorous hemodialysis.

When renal function is compromised, careful monitoring of electrolyte levels, daily urine output, and renal function is necessary with mannitol therapy.

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DIAMOND-COATED ALL-PURPOSE FOREIGN-BODY FORCEPS

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The removal of intraocular foreign bodies has always been difficult and many instruments have been designed for this purpose. With the advent of intraocular microsurgery, even more sophisticated foreign-body retrievers are needed. Among the desired qualities are precise guidance, minimal distraction during grasping procedures, self-closing mechanisms to avoid tiring the hand, and better designs for holding the foreign bodies. We have found that existing foreign-body forceps are difficult to manipulate because of coarse or counterdirectional movements during grasping maneuvers. A squeeze-handle, for example, necessitates activation of the instrument, resulting in irregular movements of the tip.

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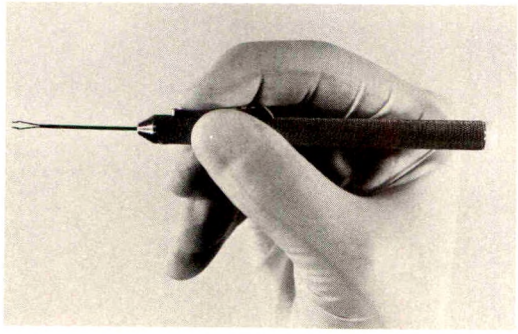


Fig. 1 (Hickingbotham, Parel, and Machemer). Two-jaw foreign-body forceps. The jaws are opened by sliding the control backwards. They close automatically by spring action.

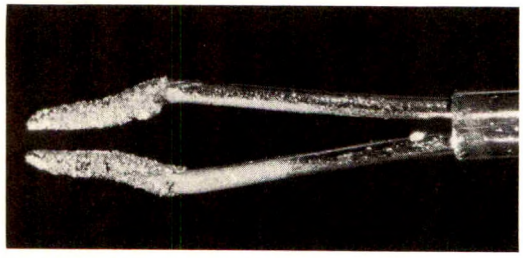


Fig. 2 (Hickingbotham, Parel, and Machemer). Magnified view of the diamond-coated jaws (× 7).

Rotation of the instrument is difficult because activation of the grasping jaws can be done only in a certain position. During retraction maneuvers, the surgeon's hands get tired when holding the foreign body for an extended period of time. The great variety of foreign bodies demands a great variety of jaw designs adapted to each specific case.

We have developed an instrument capable of removing any intraocular foreign body regardless of its size, shape, or composition.* This instrument has two parallel diamond-coated jaws (Figs. 1 and 2) protruding from a stainless steel tube 1.2 mm wide. The hand-piece is a knurled stainless steel tube (9.5 mm in diameter and 115 mm in length). A wedge-shaped control is comfortably situated on the hand piece. Sliding the forefinger back on this control pulls the stainless steel tube back and opens the jaws while the position of the jaws does not change. They are closed by automatic spring action which can be further reinforced by additional finger pressure.

Rotation of the handle during grasping maneuvers is easy. Tiring of the fingers after the foreign body has been grasped does not occur because of the self-closing action.

The key to this instrument is the diamond-coating of the jaws. Because diamonds are one of the hardest materials in existence, diamond splinters dig into the surface of any material. This, together with the automatic closing action, results in a firm grip on the foreign body. The simple two-jaw design permits the forceps to be used for foreign bodies of any shape.

*This instrument will be produced by Grieshaber and Co., AG, CH-8203 Schaffhausen, Switzerland.

Summary

A new foreign-body forceps with diamond-coated jaws can grasp and hold intraocular foreign bodies of any shape and composition. A self-closing mechanism holds the material once grasped.

MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

AMERICAN ACADEMY OF OPHTHALMOLOGY—1980 MEETING

Under the aegis of President Edward W. D. Norton the Academy returned to Chicago for the first time since 1969, but with a difference. Rather than in the friendly confines of the Palmer House, the meeting was held at the giant convention center, McCormick Place and McCormick Place West.

The meeting was originally scheduled for New Orleans. Last January it became evident that New Orleans did not have the hotel space or the meeting rooms to manage the convention. Fortunately, Chicago was available because the week of the presidential election (November 2 to 7, 1980) is generally not a popular one for meetings. The late booking of the Convention made it impossible to ready the entire McCormick Place in time, and thus the scientific and technical exhibits were held in McCormick Place West. Considering that major conventions are often scheduled ten years in advance and that this year's Academy meeting was booked less than a year in advance, the meeting went off unusually well.

Nonetheless, the technical and scientific exhibits were separated from the meeting rooms by a quarter-mile walk. The exhibits were situated in a two-floor display space. In an effort to woo strollers past technical exhibits, escalators were programmed so that the escalators going up were situated an eighth of a mile from those going down. Despite this, the meeting was the usual extraordinary Academy success, with 11,999 registered. This number included spouses, technical exhibitors, ophthalmic nurses, orthoptists, and ophthalmic assistants and technicians, as well as ophthalmologists. The weather cooperated by providing a spectacular Indian summer so that old-timers were happy to return to Chicago.

At the opening session President Edward Norton presented the guests of honor: Pierre Amalric of Albi, France, a member of the Academy since 1968 and a recipient of Academy prizes for scientific exhibits on two occasions; Ernst Custodis, Emeritus Professor of Ophthalmology in Dusseldorf; Dohrmann K. Pischel, president of the American Academy of Ophthalmology and Otolaryngology in 1960; and Paul G. Wetzig, a practicing ophthalmologist of Colorado Springs, who pioneered in photocoagulation treatment of diabetic retinopathy. The Distinguished Public Service Award was presented to Mary Lasker, President of the Albert and Mary Lasker Foundation. Mrs. Lasker has led the drive for research in cancer, cerebral palsy, high blood pressure, heart disease, stroke, and ocular diseases.

These awards were followed by the 37th Edward Jackson Memorial Lecture, "Clinical and Research Aspects of Subtotal Open-Sky Vitrectomy," given by Charles L. Schepens. This lecture is published elsewhere in this issue of THE JOURNAL. The remainder of the morning was devoted to a symposium on diabetic retinopathy co-chaired by President Norton and George W. Blankenship. These sessions were held in the cavernous Arie Crown Theatre, which seats 4,300. Nonetheless, there was standing room only.

Because Nov. 4 was election day, there were only two symposia scheduled along

with instruction courses. The evening was reserved for the presidential reception. There were numerous television sets in the grand ballroom to permit the fellows and their guests to participate in the expected thrills of election night, but by the time the reception started the results were known and the excitement never materialized.

More than 260 papers were given between Sunday night and the conclusion of the meeting on Friday morning. Over 100 additional papers were presented by related groups at associated meetings. There were 361 instruction courses, which lasted from one to nine hours. All in all, it was a busy time.

There were 45 exhibits and 74 poster sessions; six films were shown. The prize winners of the scientific exhibits were as follows: First Place—"Spontaneous Vitreous Hemorrhage. Etiology and Management" (principal presenter, Richard L. Winslow; co-presenters, Bruce C. Taylor and Anna Wiley); Second Place—"Alternatives in the Management of Intraocular Tumors" (principal presenter, Jerry A. Shields; co-presenters, James J. Augsburger, Jay Federman, Jeffrey L. Shakin, Gary C. Brown, Norman T. Felberg, and Terrance L. Tomer); Third Place—"Evaluation and Control of Endothelial Damage in Intraocular Lens Insertion" (principal presenter, David B. Soll; co-presenters, Sol E. Harrison, Mustapha Shayegan, and Frank Arturi).

The meeting on Monday afternoon to discuss the merger with the American Association of Ophthalmology was dominated by proponents of the measure but opposition was more vocal at the business meeting. The proponents emphasized the value of a single group speaking for ophthalmology, whereas those opposed questioned the value of a merger with an organization that had failed to enlist the membership of a majority of ophthalmologists at the expense of increased dues and admission to the Academy of noncertified specialists. The issue will be decided by a mail vote of the members of the Academy. An estimated 3,000 members of the American Association of Ophthalmology are also members of the Academy.

Officers for 1981 include the following: David Shoch, president; Marshall M. Parks, president-elect; Stanley N. Truhlsen, first vice-president; Du Pont Guerry III, second vice-president; and Homer E. Smith, third vice-president. Councillors are Byron H. Demorest, Thomas D. Duane, Theodore Steinberg, and Thomas P. Kearns. Bruce E. Spivey continues as executive vice-president and Paul Henkind as editor of "Ophthalmology." Paul R. Lichter is Secretary for Program; Melvin L. Rubin, Secretary for Instruction; David Paton, Secretary for Continuing Education; and Robert D. Reinecke, Secretary for Government Relations.

Future meetings of the American Academy of Ophthalmology are as follows: Atlanta, Nov. 1 to 5, 1981; San Francisco, Oct. 31 to Nov. 6, 1982, in conjunction with the XXIV International Congress of Ophthalmology; Chicago, Oct. 30 to Nov. 3, 1983; Atlanta, Nov. 11 to 15, 1984; San Francisco, Sept. 30 to Oct. 4, 1985. In 1986 the meeting is tentatively scheduled for Chicago, Las Vegas, or Washington, D.C.; in 1987 tentatively for Dallas, Las Vegas, or Washington, D.C.; 1988, Las Vegas, San Francisco, or Washington, D.C.; 1989, Chicago, Dallas, Las Vegas, or Washington, D.C.; 1990, Dallas, Las Vegas, or Washington, D.C.; and 1991, Dallas or San Francisco.

FRANK W. NEWELL

EUROPEAN OPHTHALMIC PATHOLOGY SOCIETY

The European Ophthalmic Pathology Society held its Annual Meeting in Stockholm, June 11 to 14, 1980. Professor Raymon Font (Houston, Texas) was the

guest of honor. The scientific program included 38 presentations by members and guests and 16 countries were represented. For each presentation a protocol, histopathologic sections, and appropriate clinical, macroscopic transparencies and electron microscopic prints were provided.

The majority of the case presentations dealt with the diagnosis, classification, and management of neoplasia in the globe and orbit. A large group of cases was concerned with metabolic disorders and with retinal dystrophies. The remainder dealt with inflammatory disease.

The 33 members who attended were: S. Ry Andersen (Denmark); K. Arnesen (Norway); N. Ashton (United Kingdom); J. Babel (Switzerland); E. Balestrazzi (Italy); D. R. Barry (United Kingdom); M. Brihaye-van Geertruyden (Belgium); A. Brini (France); B. Daicker (Switzerland); P. Dhermy (France); P. C. Donders (Holland); H. Fanta (Austria); A. Garner (United Kingdom); A. Hamburg (Holland); A. Henriquez (Spain); O. A. Jensen (Denmark); E. Kock (Sweden); E. Landolt (Switzerland); W. R. Lee (United Kingdom); O. Litricin (Yugoslavia); O.-E. Lund (West Germany); W. A. Manschot (Holland); G. Morgan (United Kingdom); J. Mullaney (Ireland); G. O. H. Naumann (West Germany); A. H. Nover (West Germany); M. Quintana (Spain); A. H. S. Rahi (United Kingdom); R. Seitz (West Germany); F. H. Stefani (West Germany); A. Tarkkanen (Finland); D. Toussaint (Belgium); and M. Vogel (West Germany).

Professor P. Bec (France), Professor J. H. Cunha-Vaz (Portugal), and D. M. Hanssens (Belgium) were unable to attend.

The five guests who attended were D. De Wouff-Rouondaal (Holland); G. Goder (East Germany); M. Lahav (Israel); D. Lucas (United Kingdom) and P. Naeser (Sweden).

Dr. Erik Kock (Sweden) acted as host to the meeting and was assisted by Dr. Dan Ebstein. The meeting was very well organized and the highlight of the social program was a visit to the City Hall of Stockholm, which is the location for many ceremonies associated with the Nobel Prize Awards.

The next meeting will be held in Geneva, Switzerland (May 24 to 28, 1981) and will be a combined meeting with the Verhoeff Society. Professor J. Babel will act as organizing secretary.

NORMAN ASHTON

AMERICAN JOURNAL OF OPHTHALMOLOGY™

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AMERICAN OPHTHALMOLOGY AND INTERNATIONAL STANDARDS

A small number of ophthalmologists within the United States have maintained leadership and balance in the development of ophthalmic standards since the early 1950s. Originally, such standards concerned only ophthalmic lenses and were largely nonfederal. Broadening of the United States Food and Drug Administration (FDA) programs, however, created a new federal involvement, particularly after the 1976 Medical Device Amendments. Producers of ophthalmic and optical products in this nation were unconcerned about such federal activities until the FDA Bureau of Medical Devices entered the field of lens standards in 1971. Lessons in procedure, previously learned by the American pharmaceutical industry, were gradually introduced into the planning of our optical industries during the 1970s.

Because ophthalmologists traditionally have been concerned with medicine rather than profits, they have provided impartial leadership for those interested in developing and merchandising ophthalmic and optical products. The recent rise of consumerism and the ever-expanding activities of the United States Federal Trade Commission (FTC), through its Bureau of Competition, have generated many publications critical of nearly everyone in the ophthalmic community. These publications have evaluated ophthalmic care and devices with varying degrees of accuracy. FTC employees obtaining repeated ocular examinations in the offices of ophthalmologists and optometrists have reported a superficial comparison of skills. Other FTC agents have had spectacles duplicated by opticians, optometrists, and dispensing ophthalmologists in an attempt to assess accuracy.

Fortunately, the American National Standards Institute, with the support of the American Academy of Ophthalmology and with representatives for all the appropriate national organizations, has published a series of voluntary reference standards for ophthalmic lenses, industrial ocular safety devices, contact lenses, contact lens materials, accessory solu-

tions, sunglasses, and ophthalmic frames. We hope that intraocular lens implants will be included by early 1981. Additional standards are being discussed for electrodiagnostic instruments, visual aids, soft contact lenses, and over-the-counter spectacles for near reading.

Without question, these documents, each developed during a span of several years and revised at least every five years, have a stabilizing and often beneficial effect on the quality of ophthalmic products. The standards organizations of many other technically advanced nations have issued documents concerning spectacles, trial cases, microscopes, geodetic devices, and a large range of optically implemented scientific equipment. West Germany, with its strong optical industry, has played a primary role since its reconstruction after World War II. The importance of international standards for nomenclature and scientific communication has long been recognized informally and by professional organizations such as the International Council of Ophthalmology, which has defined visual standards for driving periodically since 1927. More recently, the International Perimetric Society established in 1974 with E. Aulhorn as president, with the National Academy of Sciences/National Research Council Committee on Vision, has published a large glossary and set of standards for perimetric devices. German attention to detail has led the world in construction of classical indentation tonometers, and the Goldmann standard of excellence by applanation devices.

In 1946, the International Organization for Standardization, Geneva, Switzerland, was created to facilitate international terminology, measures, and marketing. Its secretary-general is Olle Sturen. Technical committees have been established on anesthetic equipment, dentistry (Technical Committee 106), contraceptives, surgical implants (Technical Committee 150), and many nonmedical fields. Technical Committee 170 on Surgical Instruments has been working under its chairman H. F. Kittel of Germany since its inaugural meeting in June 1978. In 1960, the International Organization for Standardization had approximately 100 international agreements; there are now almost 4,000 standards covering more than 50,000 pages. The intergovernmental General Agreement on Tariffs and Trade, dating from 1947–1949, is the most commanding of all contracts bearing on the international use of products. This Agreement provides that all existing relevant international standards shall be used by the signatory governments. Before the widespread acceptance of this Agreement, publications of the International Organization of Standardization were issued as Recommendations; subsequent publications were indexed as International Standards. The United States Congress, by its 1979 enactment of Title IV of The Trade Agreement Act, further committed all federal agencies to observing appropriate international standards.

The International Organization for Standardization particularly affects ophthalmologists and the ophthalmic industry through Technical Committee 172 on Optics and Optical Instruments. The initial meeting was organized by German leaders of the Deutsches Institut fur Normung, in May 1979 at Pforzheim, Germany, the heart of the German optical industry and only a few kilometers from Oberkochen. Earlier committees included Technical Committee 42 on Photography, created in 1972 and by default the only group that encompassed optics and ophthalmic instruments, and Technical Committee 94 on Personnel Protection, including Subcommittee 6 on Industrial Eye Wear. Technical Committee 173 deals with visual aids.

Germany, with a 25-person delegation

(by far the largest of all delegations), thoroughly prepared for the inaugural meetings. The Germans were helped significantly by Italian and Swiss colleagues. The participating nations subdivided Technical Committee 172 into nine subcommittees. The nation chairing a subcommittee then used its own national standards as the base for further development. The initial subcommittees were Subcommittee 1 on Fundamental Standards and Measures (Chairman, Dr. Walter of Rodenstock), Subcommittee 2 on Materials for Optical Processing, Subcommittee 6 on Geodetic Instruments, Subcommittee 7 on Other Optical Instruments and Endoscopes, Subcommittee 8 on Ophthalmic Optics (chaired by M. Galceran of France), and Subcommittee 9 on Other Optical Systems. Germany chaired three subcommittees, France two, and the United Kingdom two. The participation of the United States was limited with only three members to cover all fields. Japan elected to delay because of distance and language difficulties.

The Optical Manufacturers Association (Arlington, Virginia) recently recognized the reality of international standards and became the Administrator for the U.S. Technical Advisory Group to Technical Committee 172. In addition to taking on the complex clerical needs of this group, the Optical Manufacturers Association contributes $2,500 annually to support the American National Standards Institute and, thus, the International Organization for Standardization.

The meetings of the International Organization for Standardization have been carried on in an elegant and formal atmosphere with English and French as the only official languages. The leader of each national committee, or Technical Advisory Group as the United States group is called, is the only one authorized to speak for his country's participants at the conference. An enormous amount of preliminary work is performed and reference documents become the mainstays of most discussions and maneuvers. Meetings usually require three to four days with the first half-day devoted to protocol, organization, and identification of tasks by subcommittees or by working groups. There are generally twice as many observer nations present as participating nations. Canada is currently an observer.

German standards are so high that products entering that nation are not marketed unless they conform to adopted standards. Thus, standards may serve as barriers, as well as improve quality. Complying with international standards will open many markets to the United States.

The U.S. Technical Advisory Group to Technical Committee 172 consisted of 43 members in September 1980. The chairman is Donald B. Whitney, Director of Vision Correction Technology Research and Development for American Optical Corporation. The ophthalmologists are H. K. King, R. W. Ryan, M. A. Mainster, O. H. Dabezies, G. Waters, L. Goldman, and A. H. Keeney. David A. LaMarre, replacing James O'Neil as chairman of the Z87 Committee for Occupational and Educational Eye and Face Protection, of the American National Standards Institute, represents both the National Society to Prevent Blindness and the American Optical Corporation. About 16 other members have been longtime participants in the Z80 Committee on Ophthalmic Standards and in the Z87 Committee of the American National Standards Institute.

Though some subcommittees have not yet convened, Subcommittee 8 on Ophthalmic Optics held its inaugural meeting in Paris, May 19 to 21, 1980; M. Galceran of France was unanimously approved as chairman. This subcommittee deals with spectacle lenses, contact lenses (exclud-

ing bandage lenses), and prescription frames but not with plano industrial protective eyewear and plano sunglasses. Three working groups were established. Of the six nations represented, France had the largest delegation, 15 individuals; the United States had five delegates. However, the American National Standard Institute's "Recommendations for Prescription Ophthalmic Lenses" (Standard Z80.1) was adopted in several parts (Progressive Power Lenses, and Major Reference Point vs Optical Center). The meeting was held at the headquarters of the Association Française de Normalisation, the French standards organization comparable to the American National Standards Institute. The second meeting will be held in June 1981.

The initial meeting of Subcommittee 7 on Optical Instruments (including keratometers, focimeters, slit lamps, ophthalmoscopes, and endoscopes) chaired by H. Ganse was held September 24 to 26 in Pforzheim, Germany; that of Subcommittee 5 on Microscopes, October 7 to 9 in Pforzheim; and that of Subcommittee 6 on Geodetic Devices, October 23 and 24. A lead document on all tonometers was introduced by the United Kingdom in the summer of 1980.

The small American delegations were greatly helped by the previously published standards of the American National Standards Institute. It was an advantage that large numbers of scientific definitions have been hammered out in the United States. Fundamental questions, however, plague the deliberations. One major controversy is the wavelength of light to be used in determining refractive powers. Although the sodium-D line (589.3 μ), actually a double line, is well established in our country, Germany, France, and Italy have moved to the mercury-e line (546.07 μ), and the United Kingdom and Japan use the helium-d line (587.6 μ). As recently as 1960 almost every nation used the sodium-D, but Germany, Switzerland, and Italy shifted to the significantly shorter (and elegantly narrower band) wavelength in 1967. They were followed by France in 1975. Although the diopter is used in most countries, some European nations have selected the reciprocal meter (m^{-1}) to express this unit.

If the United States is to maintain its ophthalmic leadership, there must be scientific support for the tools of our trade. This nation produces millions of lenses, thousands of microscopes, countless lasers, and ophthalmic protective devices every year. All physicians concerned with products, appliances, devices, implants, or even noninvasive diagnostic instruments should support United States participation in the voluntary standard-setting process. Logical scientific standards and an improved import-export balance can be secured if our ophthalmologists and ophthalmic organizations join fully in these efforts.

ARTHUR H. KEENEY

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Mitral Valve Prolapse Questioned

Editor:

In their article, "Retinal emboli in patients with mitral valve prolapse" (Am. J. Ophthalmol. 90:534, 1980), N. D. Caltrider, A. R. Irvine, H. J. Kline, and A. Rosenblatt suggested that many patients with unexplained retinal arterial occlusive disease may have mitral valve prolapse with emboli to the eye. As a possible example they cite an article[1]

published in THE JOURNAL in 1976 that described a 34-year-old woman who had retinal arteriolitis with retinal vaso-occlusion and neovascularization. Stimulated by this article, we recalled this patient. She is now 40 years old and is still in excellent health. She no longer uses birth control pills. She recently described occasional episodes of blurring of vision in her right eye that last several hours. An ophthalmologic examination disclosed a visual acuity of 6/9 (20/30) in both eyes. Ophthalmoscopy showed that both optic disks were pale. The arterioles were attenuated and there was pigmentary mottling in the right macula. The periphery of both eyes, but particularly the right fundus, showed evidence of old retinal vaso-occlusion with large areas of capillary nonperfusion. There were no new areas of vascular closure. The neovascularization previously treated was not perfused. There was no evidence of active retinal arteriolitis.

The patient was referred to cardiology for evaluation of possible mitral valve prolapse. Results of this examination, which included echocardiography, were normal.

Although an occasional case of mitral valve prolapse may be missed by cardiologic examination and echocardiography, it seems likely that our patient does not have mitral valve prolapse. The origin of her vascular disease, which seems inactive at present, remains uncertain.

LEE M. JAMPOL
Chicago, Illinois

REFERENCES

1. Jampol, L. M., Isenberg, S. J., and Goldberg, M. D.: Occlusive retinal arteriolitis with neovascularization. Am. J. Ophthalmol. 81:583, 1976.

Flying and Intraocular Gas Bubbles

Editor:

In his letter, "Flying after vitreous injection" (Am. J. Ophthalmol. 90:580, 1980), J. R. Brinkley, Jr., reported the occurrence of severe pain in a patient who traveled by commercial airplane after retinal surgery with placement of intraocular gas. Dr. Brinkley suggested that his case might be the first actual report of difficulty with intraocular gas bubble expansion during air travel.

In 1976 Dr. E. W. D. Norton and I[1] reported several cases of post-vitrectomy patients with intraocular gas who experienced severe ocular pain while flying home after vitreous surgery. One patient gave a vivid account of experiencing severe pain in his operated eye as the plane reached 36,000 feet at which point he lost all vision in the eye. The pilot was notified and brought the plane rapidly down to 12,000 feet where vision returned and the pain disappeared.

Commercial planes typically fly with a cabin pressure equivalent to 7,000 feet. Simple mathematics shows that 1 cc of gas at sea level occupies 1.25 cc at 7,000 feet. Commercial jets normally take 25 to 30 minutes to reach cruising altitude. During this period of gradual decrease in cabin pressure, the tendency of the gas bubble to expand forces fluid from the eye through normal outflow channels. If the ascent is gradual, the bubble is not too large, and ocular outflow channels are competent, only a moderate increase in intraocular pressure should occur.

For several years I have routinely permitted postoperative patients to fly when the remaining air or sulfahexafluoride bubble is one third of the volume of the vitreous cavity or less. I usually give them acetazolamide approximately two hours before flying. Additionally, I suggest that they assume a head-down position if pain occurs during the flight. No problems have occurred to date.

Whether or not a given patient with gas in his eye will have difficulty during air travel depends not only on the size of the bubble but also on the facility of outflow.

Patients who have demonstrated postoperative glaucoma during their hospital stay should be particularly cautious about air travel while large intraocular gas bubbles remain.

Dr. Brinkley's letter is valuable in re-emphasizing the importance of caution. I cannot agree, however, with his statement that patients should not fly until all gas has disappeared from the eye.

DWAIN FULLER, M.D.
Dallas, Texas

REFERENCES

1. Norton, E. W. D., and Fuller, D. G.: The use of intraocular sulfahexafluoride in vitrectomy. In Irvine, A. R., and O'Malley, C. (eds.): Advances in Vitreous Surgery. Springfield, Charles C Thomas, 1976, pp. 535–542.

Hereditary Fuchs' Dystrophy

Editor:

I read with interest the article, "Hereditary Fuchs' dystrophy" (Am. J. Ophthalmol. 90:455, 1980) by P. Rosenblum, W. J. Stark, I. H. Maumenee, L. W. Hirst, and A. E. Maumenee.

The authors claim that Fuchs' dystrophy is a true corneal dystrophy with autosomal dominant inheritance, yet are unable to explain why there is a female-to-male ratio of approximately 7:1 among probands. Furthermore, a 3:1 predominance of females was discovered in 79 cases of penetrating keratoplasty performed for phakic Fuchs' dystrophy between 1940 and 1978. The average age of the patients was 65.3 years. The authors conclude that Fuchs' dystrophy, although inherited in an autosomal dominant fashion, is X-chromosome-dependent in its expressivity and more severe in females.

The preponderance of females in this late-onset corneal dystrophy is probably the result of the longer life expectancy of women. Average life expectancy for men in 1940 was only 60.8 years, whereas that for women was 65.2 years. By 1977 the discrepancy had increased still further: life expectancy for men was 69.3 years, whereas that for women was 77.1 years. It is unnecessary to postulate other X-chromosome-dependent differences in the incidence and course of Fuchs' dystrophy.

LINAS A. SIDRYS, M.D.
Gainesville, Florida

Reply

Editor:

We set out to demonstrate that Fuchs' endothelial dystrophy is an autosomal dominant disorder, with an inheritance pattern there would ordinarily produce an equal number of affected men and women without any difference in severity. We obtained an indication that this mode of inheritance was applicable through pedigree analysis which showed male-to-male transmission, the hallmark of autosomal dominant inheritance.

We then postulated that the excess number of affected females, described by others and also seen in our sample, and the apparent increased severity of this dystrophy in women were caused by a bias of ascertainment. One evident bias is introduced by the longer average survival of women in the United States' population, as suggested by Dr. Sidrys in his letter. Another source of bias may be an increased tendency for women to seek medical attention.

We hoped to eliminate such biases from our study sample by examining first-degree relatives and by excluding the probands from the analysis. Within the sample thus defined there was no difference between the sexes in the number of affected individuals, but we had too few cases classified by severity to confirm the presence of an X-chromosome-dependent difference in severity. In our sample, an age-adjusted comparison of severity between males and females did

demonstrate an observable but not statistically significant trend toward increased severity in females (P = .15, by the Mantel-Haenszel test). Our sample, however, contains only eight males and 14 females.

Lastly, the female-to-male ratio of 3:1 among patients in our study who required surgery also suggests the possibility of an X-chromosome-dependent difference in severity, although data for that group of patients were not age-adjusted, and the differences may have been the result of longer female survival only.

IRENE H. MAUMENEE, M.D.
WALTER J. STARK, M.D.
Baltimore, Maryland

Dangers of Intravenously Administered Diazepam

Editor:

The recent article, "Cardiopulmonary arrest after retrobulbar block" (Am. J. Ophthalmol. 90:425, 1980), by R. M. Rosenblatt, D. R. May, and K. Barsoumian, stimulated my interest as I hope it did that of other ophthalmologists. I disagree, however, with the authors' conclusions and would like to offer my own explanation of this "cardiopulmonary arrest."

When I was a resident, I saw many similar cardiac arrests produced by the intravenous injection of diazepam. Diazepam is a respiratory depressant and this patient responded exactly as predicted for an intravenous injection of diazepam. The only variation is that she responded to a low dose of 5 mg. This, however, is not unusual, especially for someone with her medical problems. Additionally, pressure on the globe exerted by a retrobulbar injection can cause a vagal response, creating the indicated cardiac changes. According to this case report, this patient did not have a cardiac arrest at all because 15 seconds of asystole cannot be considered a cardiac arrest. Her cardiac response was as predicted for a respiratory arrest in that she had tachycardia and increased blood pressure. Her heart apparently was strong and she sustained this cardiac effort without any permanent loss. Her respiratory arrest lasted as predicted for the metabolizing of diazepam and she responded with resumed respiratory effort in approximately 30 minutes.

I believe that many anesthesiologists have forgotten how dangerous diazepam can be when given intravenously. Respiratory arrests are most likely to occur when the anesthesiologist is working with the ophthalmologists on a standby basis because these patients are often old and have other medical problems. The authors were fortunate that this injection did not result in the patient's death. When the patient's face is covered and diazepam is injected intravenously, the patient's quietness may be caused by a respiratory arrest rather than by the sedative effect of diazepam. This respiratory arrest may be undetected until hypoxia causes cardiac irregularities; by then, there may be brain damage. Because the anesthesiologist normally controls the patient's airway, this problem does not occur except in local cases when the patient's face is covered.

In addition to these problems, I dislike using intravenously administered diazepam in elderly patients who are excited or nervous about undergoing surgery. I have found that diazepam sometimes has an unpredictable or adverse effect on elderly patients when given intravenously and sometimes makes the patients confused and more agitated. I have had so many problems with intravenously administered diazepam that I no longer request that an anesthesiologist stand by. I find the drug is given sometimes before I can say something to the anesthesiologists. If I do have an anesthesiologist

stand by, I am very careful to request that diazepam not be used.

W. B. OWEN EDELEN, M.D.
Louisville, Kentucky

Reply

Editor:

Dr. Edelen is quite correct in his observation that diazepam can induce respiratory depression and, possibly, a cardiopulmonary arrest. Interestingly, the scientific data to support this observation have been lacking until recently. The elegant study by Jordan, Lehane, and Jones[1] has now confirmed the respiratory effects of diazepam.

Notwithstanding this, Dr. Edelen's explanation is most probably incorrect based upon the sequence of events we encountered. Although the patient was elderly, her response to intravenously administered diazepam was not accentuated. The diazepam was administered in several incremental injections during a five-minute period. At the time of the arrest the patient was awake and conversing with the surgeon. Thus, clinically significant diazepam-induced respiratory depression almost certainly was not a predisposing factor in the subsequent cardiopulmonary arrest. Several other factors also argue against Dr. Edelen's explanation. Induction of general anesthesia can be accomplished with diazepam but this requires a much larger dosage and the maintenance of anesthesia with diazepam alone is exceedingly difficult. Prolonged apnea and lack of response to painful stimuli would not be anticipated without the concomitant use of narcotics and muscle relaxants. Neither the cardiac instability nor the two hours of post-arrest hyerreflexia with diminished sensation can be accounted for by the administration of diazepam. Although an ocular-cardiac reflex would have occurred, it would not by itself explain the noncardiac abnormalities. Lastly, the metabolizing of diazepam, despite Dr. Edelman's statement, is exceedingly slow, particularly in the elderly.

Whether diazepam should be used in ophthalmic surgery depends on its advantages and disadvantages in each specific case. All-encompassing statements forbidding its use display a nonscientific attitude on the part of the author. Because one of us is an anesthesiologist (R.M.R.), we would like to know what alternative "superior" sedatives Dr. Edelen uses in his practice rather than diazepam and how his patient's safety is enhanced when he does not request anesthesia coverage in his local cases.

RICHARD M. ROSENBLATT, M.D.
DONALD R. MAY, M.D.
Davis, California

REFERENCE

1. Jordan, C., Lehane, J. R., and Jones, C.: Respiratory depression following diazepam. Anesthesiology 53:293, 1980.

BOOK REVIEWS

Ocular Pathology Update. Edited by Don H. Nicholson. New York, Masson Publishing USA, Inc., 1980. Hardcover, 291 pages, index, 420 black and white figures. $55

Every subspeciality in ophthalmology likes to appear at its best, and Dr. Nicholson is to be congratulated for having accomplished this task for ophthalmic pathology. In a mere 283 pages of text, the editor has managed to confect a discriminating sampler of clinical and pathologic research on practically every ocular and adnexal tissue and on a wide spectrum of diseases. His achievement is the result of having engaged an all-star cast of contributors, not the least successful of whom is the editor himself, who

supplied three outstanding chapters. The following topics are covered in a concise fashion with illustrations that have reproduced well: Metabolic diseases, corneal dystrophies, the pathology and pathogenetic mechanisms of the glaucomas, retinal vascular diseases, proliferative retinal membranes, senile macular degeneration, immunologic aspects of ocular and adnexal tumors, the intraocular melanoma controversy (including diametrically opposed points of view), pathologic and metabolic advances in retinoblastoma, epibulbar tumors, and lacrimal gland lesions. What is most exciting about this book is that many of the chapters are original contributions; others are summaries of fields that have undergone rapid development, where the approach is fresh and one never fails to learn something new. As significant advances are increasingly dispersed within a plethora of subspecialty journals, books of this type and quality that gather together in one place the important breakthroughs provide a welcome convenience and intellectual pleasure.

FREDERICK A. JAKOBIEC

On Writing, Reading and Dyslexia. By Arthur Linksz. New York, Grune and Stratton, Inc., 1973. Hardcover, 256 pages, index, glossary, 24 black and white figures. $27.25.

This book has come to my attention some seven years after it was published. A survey of THE JOURNAL for the appropriate years did not locate a previous review. Because I found it a most clear and dispassionate account of the management of dyslexia and with the approval of the Editor, I am pleased to call the attention of the readers of THE JOURNAL to this monograph, even belatedly.

I should point out that the book is addressed to the intelligent layman but there is an enormous amount of material that every ophthalmologist who sees children with reading problems can learn. First of all, Dr. Linksz defines dyslexia in the broadest terms, that is, as all types of reading difficulties whether they are caused by specific hereditary dyslexia, associated with mental retardation, or simply the result of poor reading because of poor teaching. The latter group is the largest and the one with which Dr. Linksz is most concerned. The devil here is the "see-and-say" school of teaching of reading. Dr. Linksz points out that with this method a frequent response that one hears is "that's a word I haven't had." This obviously limits the child to reading fewer words than he has heard and in effect has a negative influence on his reading ability. The answer to the problem is phonics. If the child is taught to sound out syllables and to learn a few very simple rules such as "a silent E or an R changes the sound of the preceding vowel," he can quickly learn to read very long words because he need only apply the rules to each syllable. The answer to most reading problems, then, is to teach reading by the old phonics method.

The second point that Dr. Linksz makes is that writing should precede or at least accompany reading. Learning to write easily makes reading literally child's play. Further, we live in a right-handed world and all writing, at least in the Indo-European languages, goes from left to right, and writing from left to right is much more easily done with the right hand. Therefore, although children need not be dissuaded from using their left hands for playing baseball or other games, gentle coercion may be required to make them write with their right hands (Dr. Linksz's instructive pun). A certain number of psychologists are going to scream at this advice, but the author convinced me of the correctness of this position.

Finally, although we all know that vision is the core of the sensory world, one of the volume's appendices will also

convince you that it is the core of the motor world as well. Read the appendix to find out why the pyramidal tracts are crossed and read the entire book for a delightful experience.

DAVID SHOCH

Lecture Notes on Ophthalmology. By Patrick D. Trevor-Roper. Oxford, Blackwell Scientific Publications, 1980. Paperbound, 128 pages, preface, appendix, index, 14 color figures, 76 black and white figures. $12.25

This introduction to ophthalmology for the medical student is skillfully presented with the engaging style of Trevor-Roper. The introduction, which briefly describes the anatomy and examination of the eye, is followed by two chapters on the eyelids, lacrimal apparatus, and orbit and then by a section on the painful red eye. This is followed by a section of gradual and sudden loss of sight in quiet eyes. Also included are chapters on injuries, refractive errors, squint and nystagmus, the central mechanism of vision, and an appendix of questions and multiple-choice questions.

Trevor-Roper's comments on multiple-choice questions are germane:

> First, words such as "always" and "never" rarely apply to the discipline of medicine, and such questions should arouse strong suspicion as to their validity. In contrast, almost anything "can" or "may" occur. Such key words may give the answer away, even when the student does not know the content of the question.
>
> Secondly, although blind guessing is to be discouraged, "hunches" and "first impressions" are more often right than wrong. Re-appraisals commonly lead to errors. It may be valuable, therefore, to assess approximately how many marks you need to ensure a pass. If you are home and dry, then taking calculated risks is unnecessary. However, if you are struggling for marks, act on "hunches."
>
> Such strategy forms the basis of sound examination technique.

This is a most attractive series and provides a fine book to use in a sequence of six or eight lectures to medical students. There are a variety of colored figures. The writing is exemplary and the topics well chosen. It may be highly recommended.

FRANK W. NEWELL

Adler's Physiology of the Eye. Clinical Application, 7th ed. Edited by Robert A. Moses. St. Louis, C. V. Mosby Co., 1981. Hardcover, 747 pages, index, biological index, 475 black and white figures. $41

This is a worthy successor to previous editions. Dr. Moses has expanded this edition by asking 20 specialists in various fields to contribute. This has permitted the in-depth analysis of subjects not previously covered. Dr. Moses states in the preface that a multi-author work makes for redundancy, but what he does not say is that it also makes for unevenness in literary style and exposition. For the most part, the level is high.

The chapters have been rearranged. They start with the physiology of the eyelids and then move to the lacrimal system and the cornea. The subject matter includes the extraocular muscles, ocular circulation, aqueous, intraocular pressure, vitreous, lens, and accommodation. The last 11 chapters are devoted to the physiology of the retina, the optic nerve, and the visual pathways.

Mosby has done its usual splendid job, although the technical changes which have occurred in the printing industry are evident.

The book is no longer a textbook, but has become a resource book. The bibliography of each chapter is extensive. One wonders whether the subtitle "Clinical Application" should not be dropped.

The book is highly recommended.

DANIEL SNYDACKER

Perimetry. Principles, Technique, and Interpretation. By Carl Ellenberger, Jr. New York, Raven Press, 1980. Hard-

cover, 124 pages, references, index, 43 black and white figures. $12

This is a brief text on visual fields intended for those beginning the study of perimetry. Its objective is to provide residents in ophthalmology, neurology, and neurosurgery with sufficient information to diagnose clinical problems as well as to help them pass their board examinations.

The first chapter deals with the principles of perimetry. It begins with a review of the anatomy of the visual system. There is then a discussion of modern perimeters with emphasis placed on the Goldmann and Tübinger instruments. The chapter ends with a review of stimulus variables which influence the accuracy of visual field examinations.

The second chapter covers perimetric technique. It contains a discussion of confrontation testing followed by an extensive review of projection perimetric methods. Both kinetic and static testing are discussed in detail.

The third and final chapter covers the interpretation of visual fields. It starts with a review of visual field abnormalities caused by retinal lesions. Next, the author discusses visual field defects caused by conduction disturbances along the visual pathway. This chapter is well organized and contains much valuable information.

Perhaps a weakness of this text is its cursory treatment of tangent screen visual fields. In the offices of many ophthalmologists, neurologists, and neurosurgeons, the only method available for testing visual fields is a tangent screen. Since the orientation of this book is toward nonophthalmologists as well as ophthalmologists, a more detailed discussion of this technique seems warranted.

Many illustrations of various field defects are interspersed throughout the book. For those interested in further reading, there are 121 references. This is a moderately priced, concise, and easy-to-read text. It is well organized, informative, and can be recommended.

WALTER M. JAY

Corneal Surgery. Advanced Techniques in Ophthalmic Microsurgery. Volume 2. By Louis J. Girard. St. Louis, C. V. Mosby Co., 1980. Hardcover, 289 pages, index, 164 color figures, 196 black and white figures. $82.50

About one year ago the indefatigable Louis Girard published Volume 1 in this series. That book was devoted primarily to cataract surgery via a pars plana incision and fragmentation of the lens. In Volume 2 he has moved anteriorly to the cornea and devotes his not inconsiderable talents to the surgery of this organ. Although there is a small introductory chapter on the anatomy and physiology of the cornea and a fairly large section on the indications for corneal transplantation, the major portion of this book is a "how-I-do-it" presentation and Dr. Girard does it very well indeed. As before, the line drawings are excellent and there are a fair number of very good black and white photographs which was not true in the previous volume. There still are some that are out of focus and blurred but there is great improvement generally. Of interest are the stereoscopic photographs for which one uses a pair of +7 spheres that come with the book. In the previous volume the stereoscopic photographs were mounted in Viewmaster reels. The new method is obviously less expensive and, surprisingly, works just as well if one has good stereoscopic vision. An improvement would be the use of a device that allows for a change in the interpupillary distance.

Dr. Girard has introduced an interesting variation by a concluding chapter devoted entirely to the results of the

various procedures he describes. Most of the results are his, collected over a lifetime of surgery on the cornea, but he also quotes other authors as well. If there is any criticism to be made, it is the fact that in most cases Dr. Girard reports the procedures and data of other people uncritically. This is probably reasonable in a compendium, but one would like to hear Dr. Girard's views on the usefulness of such procedures as thermal keratoplasty, epikeratoprothesis, and radial keratotomy. In fairness it must be said that he does comment when the data are his own.

Corneal surgery, like most other surgery, must be learned by doing, but it is not possible for any student to visit all the surgical centers of the United States and a book such as this is valuable because it presents the techniques and opinions of an excellent, experienced surgeon.

DAVID SHOCH

Current Ocular Therapy. By Frederick F. Fraunfelder and F. Hampton Roy. Philadelphia, W. B. Saunders Co., 1980. Hardcover, 647 pages, preface, index. $42.50

This is an interesting volume that will be widely used. The editors have brought together some 380 experts who discuss some 400 different topics. The volume is divided into three parts: Part 1 deals with generalized disorders, Part 2 with the eyes and adnexa, and Part 3 is devoted to a roster of drugs. Most topics follow the same outline: an introductory statement defining the disease; therapy, systemic, supportive, surgical, and medical; ocular and periocular manifestations; precautions; comments; and references.

The list of authors reads like a Who's Who of international ophthalmology. Their individual contributions are competently prepared. The editors have been remarkably successful in avoiding duplications. The sole criticism relates to the many references of secondary sources, such as Duke-Elder, Duane, and similar books, while many of the references to journals are not current. This is a minor objection. The book is first-class and I feel certain that those who use it will be looking for the latest in surgical and medical treatment and not for a review of the current medical literature.

FRANK W. NEWELL

SYMPOSIA

Colour Vision Deficiencies. Volume V. Edited by G. Verriest. Bristol, England, Adam Hilger, Ltd., 1980. Hardcover, 450 pages, author index, subject index, 195 black and white figures. $95

STELL, W. K.: **Photoreceptor-specific synaptic pathways in goldfish retina. A world of colour, a wealth of connections**

MARC, R. E.: **Retinal colour channels and their neurotransmitters**

SPERLING, H. G: **Blue receptor distribution in primates from intense light and histochemical studies**

DJAMGOZ, M. B. A., AND RUDDOCK, K. H.: **Evidence for a GABA-ergic feedback loop in the outer plexiform layer of the vertebrate retina**

CREUTZFELDT, O.: **Neuronal responses to long wavelengths. Investigation on the primate lateral geniculate body**

ZWICK, H., BEATRICE, E. S., AND GARCIA, T. A.: **Long-term and progressive changes in rhesus spectral sensitivity after low-level coherent light (514 nm) exposure**

BOWMAKER, J. K., AND MOLLON, J. D.: **Primate microspectrophotometry and its implications for colour deficiencies**

MACLEOD, D. I. A., AND BOYNTON, R. M.: **Rectangular chromaticity diagram showing cone excitations at constant luminance**

SMITH, V. C., AND POKORNY, J.: **Cone dysfunction syndromes defined by colour vision**

BIRCH, J., HAMILTON, A. M., AND GOULD, E. S.: **Colour vision in relation to the clinical features and extent of field loss in diabetic retinopathy**

LAKOWSKI, R., DRANCE, S. M., AND CARSH, A.:

Colour vision in relation to perimetric and dark adaptation functions in glaucomatous eyes

KING-SMITH, P. E., ROSTEN, J. G., ALVAREZ, S., AND BHARGAVA, S. K.: Human vision without tonic ganglion cells?

ROBBINS, D. O., AND ZWICK, H.: Changes in spectral acuity following brief laser (647 nm) exposure

STILES, W. S.: The two-colour threshold method and π mechanisms. Historical note

RUDDOCK, K. H.: Psychophysical evidence concerning independent processing of visual stimulus parameters

CARTA, F., MAIONE, M., LETTIERI, S., AND BONDI, L.: Stiles, mechanisms in clinical ophthalmology

VOLA, J. L., CARRUEL, C., AND CORNU, L.: Further studies on Stiles' technique in acquired dyschromatopsias

MOLLON, J. D., NEWCOMBE, F., POLDEN, P. G., AND RATCLIFF, G.: On the presence of three cone mechanisms in a case of total achromatopsia

PALMER, D. A.: Colour matching at high illuminances

PEREIRA, S. M.: Luminance dependence of anomaloscope matches

DE MATTIELLO, M. L. F., AND GUIRAO, M.: Influence of illumination on the discrimination of colorimetric purity

LEPRINCE, G., VOLA, J. L., AND LANGLE, D.: Influence of the spectral distribution of the illuminant on the results of clinical colour vision tests at high-level luminance

ZWICK, H., AND JENKINS, D. L.: Coherency effects on retinal neural processes (ERG) of Pseudemys

ZWICK, H., ROBBINS, D. O., AND KNEPP, A.: Changes in tectal spectral sensitivity and receptive field organisation following coherent light exposure

HANSEN, E.: The disturbance of colour vision after sunbathing

DAIN, S. J., PEREIRA, S. M., PALMER, B., LEWIS, P., AND HAMMOND, T.: Illuminance and the FM 100 hue test

HILL, A. R., AND ASPINALL, P. A.: An application of decision theory to colour vision testing

ROTH, A., REPIQUET, C., RENAUD, J. C., AND VIENOT, J. C.: The Besançon anomalometer

DAIN, S. J., STRANGE, G., AND BOYD, R.: A solid state anomaloscope

OHTA, Y., IZUTSU, Y., MIYAMOTO, T., AND SHIMIZU, K.: An experimental anomaloscope based on the interference filter system, and test results

MORELAND, J. D.: A modified anomaloscope using optokinetic nystagmus to define colour matches objectively

RICHTER, M.: New equipment for the rapid taking of spectral sensitivity curves for normal and defective colour vision

FLETCHER, R.: Second edition of the City University test

DAIN, S. J.: Assessment of the FM 100 hue derivative tests

HUKAMI, K., ICHIKAWA, H., AND TANABE, S.: Evaluation of the standard pseudoisochromatic plates (first edition)

FLETCHER, R.: Testing the Daltonic child

ARIZAGA, R. A., AND DE MATTIELLO, M. L. F.: Influence of retinal locus on the discrimination of colorimetric purity

OBSTFELD, H.: Human colour vision mechanisms along four cardinal meridians

DAIN, S. J., BARD, D., AND SUE, G.: New targets for clinical colour perimetry

FRIEDMANN, A. I.: A preliminary report on the use of colour filters in the Mark II visual field analyser

PAULUS, W., AND KRÖGER-PAULUS, A.: A model combining trichromatic and dichromatic vision

VOGT, G.: Special mixture curves with interference filters for monochromatic, dichromatic and trichromatic subjects

KLINGAMAN, R. L., ZRENNER, E., AND BAIER, M.: Increment flicker and hue spectral sensitivity functions in normals and dichromats. The effect of flicker rate

VOKE, J., AND VOKE, P.: Colour vision deficiencies in Saudi Arabia

COBB, S. R.: Red-green colour defect among the Roman Catholic and nondenominational population of Glasgow

PICKFORD, R. W., PICKFORD, R., BOSE, J., JOARDAR, B. S., NAG, P. K., RAY, G. G., AND SEN, R. N.: Incomplete achromatopsia in Bishnupur. Genetics and heterozygote problems

VAN HEEL, L., WENT, L. N., AND VAN NORREN, D.: Frequency of tritan disturbances in a population study

HILL, A. R.: Decision uncertainty for a homozygous or heterozygous female

VAN DE MERENDONK, S., AND WENT, L. N.: Two cases of inherited deutan and tritan disturbances in the same person, and a study of their families

YASUMA, T., ICHIKAWA, H., AND TANABE, S.:

Characteristics of genetic carriers of congenital colour vision defects. Deuterocarriers

LANTHONY, P.: **Boynton's colour-naming method in acquired dyschromatopsias**

WOLF, E., SCHEIBNER, H., AND PASCHKE, G.: **Colour vision in a case of retinopathia pigmentosa**

ROBERTSON, K. M., AND MORELAND, J. D.: **Study of familial retinitis pigmentosa sine pigmento combined with cystoid maculopathy**

ANTALOVÁ, K.: **Colour vision in central and peripheral tapetoretinal dystrophy**

BEGG, I. S., AND LAKOWSKI, R.: **A comparison of colour vision with other methods of clinical assessment in diabetics with macular oedema**

MORELAND, J. D.: **Tritanomaloscopy of diabetics undergoing photocoagulation therapy**

WILLIAMS, C. M., AND LEAVER, P. K.: **Visual acuity and colour vision in central serous retinopathy**

SERRA, A., MASCIA, C., DESSY, C., AND CASTI, R.: **Chromatic discrimination and haematological data in beta-thalassaemia and diabetes juvenilis**

KALMUS, H.: **Differences in adaptation and colour sensation between the eyes of a patient with latent ocular herpes**

LAGERLÖF, O.: **Drug-induced colour vision deficiencies**

ALKEN, R. G., MILLER, A., SEMJAN, R., AND RIETBROCK, N.: **Colour vision deficiencies detected by Farnsworth's 100 hue test in patients under long-term treatment with digitalis**

ASPINALL, P. A., HILL, A. R., AND CAMERON, D.: **An evaluation of the Ames Clinitest**

VERRIEST, G., NEUBAUER, O., MARRÉ, M., AND UVIJLS, A.: **New investigations into the relationships between congenital colour vision defects and road traffic safety**

COBB, S. R., AND SHAW, F. G.: **The effect of industrial exposure to lead**

VERRIEST, G., AND UVIJLS, A.: **Colour vision defects and electronics**

VERRIEST, G., AND UVIJLS, A.: **Does a tri-band illuminant improve performance in colour vision defective people?**

WERNER, J. S., AND WOOTEN, B. R.: **Age changes in ocular media density and consequences for colour vision**

RONCHI, L. R., BARCA, L., PERINI, R. P., AND VACCARI, G.: **Some remarks on visual acuity, colour discrimination and entoptical scatter, in subjects of different ages**

DRUM, B.: **Forced-choice colour discrimination in the dark-adapted parafovea**

RUDDOCK, K. H., AND WATERFIELD, V. A.: **Determination of post-receptoral spectral functions in humans**

SCHMIDT, I.: **Testing dark adaptation with two-colour stimuli**

HILL, A. R., RODGER, G., AND SMALRIDGE, L.: **Some further observations on the colour flicker phenomenon**

ARECCHI, F. T., AND RONCHI, L. R.: **Transient responses to Rayleigh matching**

VALETON, J. M., AND VAN NORREN, D.: **The retinal origin of transient tritanopia determined with the local electroretinogram**

RUDDOCK, K. H.: **Review. Colour vision at low illumination levels**

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Ophthalmologica

THE ALTERATION AND ASYMMETRY OF CUP AND DISC DIAMETERS. Bengtsson, B. (Dept. Experimental Ophthalmol., Dalby Community Care Res. Ctr., Lund, Sweden). Acta Ophthalmol. 58:726, 1980.

Increases in cupping with time and differences in cup size between the two eyes of one person were studied in a material derived from a general ophthalmic population survey. Cup and disc diameters were measured on fundus photographs. The effect of refraction on the magnification of the eye-camera system was compensated for by the use of a simple correcting factor. The disc diameter increased slowly but steadily with advancing years but the rim area remained unaffected and as a consequence the rim breadth diminished. The increase in disc diameter and the decrease in rim breadth combined to cause a marked increase in cup diameter with aging. Side differences in cup diameter were strongly dependent on side differences in disc diameter. It appears that neither an enlarging cup nor a diminishing rim breadth should be considered pathognomonic of glaucomatous disease. (1 figure, 2 tables, 11 references)—David Shoch

CORNEAL THICKNESS AND ENDOTHELIAL DAMAGE AFTER INTRAOCULAR LENS IMPLANTATION. Olsen, T., and Eriksen, J. S. (Dept. Ophthalmol., Århus Kommunehospital, DK-8000 Århus C, Denmark). Acta Ophthalmol. 58:773, 1980.

The corneal thickness and the specular appearance of the corneal endothelium are reported in 100 patients with unilateral intraocular lens implantation. Post-operative time ranged from one to 42 months. An average central endothelial cell loss of uncomplicated cases of 46%, range 1 to 83%, with no correlation with time after the operation was found. A significantly higher cell loss was found in cases with technical complications, shallow anterior chamber or increased intraocular pressure post-operatively. No correlation was found between the corneal thickness and the endothelial cell loss. Irrespective of the occurrence of surgical complications the presence of a guttate endothelium was found to be a major determinant of the corneal thickness increase and could be ascribed as a cause of persistent corneal swelling in six of twelve patients with elevated corneal thickness. The progression of guttate changes occurred independently of the cell loss. (5 figures, 3 tables, 30 references)—Authors' abstract

American Journal of Roentgenology

IMAGES OF THE OPTIC NERVE: ANATOMIC-CT CORRELATION. Unsöld, R., DeGroot, J., and Newton, T. H. (Dept. Anat., Univ. California, San Francisco, Calif. 94143). Am. J. Roentgenol. 135:767, 1980.

The course of the intraorbital portion of the optic nerve, as demonstrated on anatomic sections in various planes, is correlated with the appearance of the nerve in computed tomographic images. The discrepancies do occur and these are due to the fact that the optic nerve has a sinuous course in both the horizontal and vertical planes. Therefore no thin axial or sagittal section can be entirely parallel to the nerve. Any section will transsect the optic nerve obliquely, leading to an apparent hypodensity or "thinning" of certain segments shown on computed tomography. The appearance of the course of the nerve on reformatted computed tomographic images depends on the plane of the original computed tomographic section. Computed tomograms that are obtained with the eye in primary position do not allow reliable judgments regarding the course and size of the optic nerve. (11 figures, 5 references)—David Shoch

RADIATION DOSE ESTIMATE TO OCULAR LENS IN IRRADIATION FOR GLIOBLASTOMA MULTIFORME. Shin, K. H., O'Brien, P., Geggie, P. H. S., Muller, P. J., and Arthur, K. (Dept. Rad. Oncol., Southern Alberta Cancer Ctr., 2104 2nd St., S.W., Calgary, Alberta, Can. T2S 1S5). Am. J. Roentgenol. 135:860, 1980.

With the use of cobalt irradiation patients with glioblastoma multiforme have a better prognosis therefore they may live long enough to develop cataracts as a result of radiation. By using a model the authors were able to demonstrate that the lens received about 13% of the tumor dose when there was no special penumbra block of the eye and that this reduced to about 6% if such a block was used. Since somewhere between 5,500–6,000 rad are usually given over a five to six week period this means that without protection the lens receives around 750 rad and about half of this if a block is used. Of a group of 158 patients who had blocks there no cataracts were observed in the survivors at the end of three years. (1 figure, 1 table, 5 references)—David Shoch

Annals of Internal Medicine

CYTOMEGALOVIRUS RETINITIS IN IMMUNOSUPPRESSED HOSTS I. NATURAL HISTORY AND EFFECTS OF TREATMENT WITH ADENINE ARABINOSIDE. Pollard, R. B., Egbert, P. R., Gallagher, J. G., and Merigan, T. C. (Div. Infectious Diseases, Dept. Med., Stanford Univ. Med. Ctr., Stanford, Calif. 94305). Ann. Intern. Med. 93:655, 1980.

Fourteen patients with cytomegalovirus retinitis were diagnosed by culture of cytomegalovirus from urine or throat specimens, elevated complement fixation titers to cytomegalovirus, and characteristic ophthalmoscopic appearance. Ten of 11 had decreased cytomegalovirus-specific cell-mediated immune responses. Three of seven who received no specific therapy improved after decreasing dosages of immunosuppressive drugs. Seven patients with progressive disease despite minimal immunosuppressive therapy were treated with adenine arabinoside at doses from 1 to 20 mg/kg of body weight per day. Daily dosages of 20 mg/kg in five patients were associated with decreased inflammatory activity and improvement of retinal lesions and quantitative decreases in urinary virus excretion. Adenine arabinoside may have some beneficial effects on selective patients with progressive cytomegalovirus retinitis, however, it should be pointed out that adenine arabinoside has significant gastrointestinal, hematologic, and neurologic side effects. (5 figures, 3 tables, 34 references)—David Shoch

CYTOMEGALOVIRUS RETINITIS IN IMMUNOSUPPRESSED HOSTS II. OCULAR MANIFESTATIONS. Egbert, P. R., Pollard, R. B., Gallagher, J. G., and Merigan, T. C. (Stanford Univ. Med. Ctr., Boswell Bldg., Room A-227, Stanford, Calif. 94305). Ann. Intern. Med. 93:664, 1980.

The authors examined 21 eyes of 14 immunosuppressed patients in whom a diagnosis of cytomegalic virus retinitis was made. The post-mortem examination was then done on ten eyes from seven patients. At initial presentation, the retinitis was often asymptomatic and diagnosed during routine examination. The ophthalmoscopic picture was characteristic of cytomegalovirus; the early lesion was a small opaque, white granular area of retinal necrosis that spread in a centrifugal, brush-fire-like manner over one to eight months. Vessel sheathing and hemorrhages appeared as the disease progressed. In two patients new foci of retinitis developed remote from the original lesion. Four weeks to four months (average, ten weeks) elapsed from the most extensive disease to total resolution. Resolution of active disease left a subtle retinal scar, and final visual acuity was reduced in one half the eyes. (10 figures, 3 tables, 12 references)—David Shoch

Annals of Neurology

LONG-TERM CEREBRAL EFFECTS OF SMALL DOSES OF X-IRRADIATION IN CHILDHOOD AS MANIFESTED IN ADULT VISUAL EVOKED RESPONSES. Yaar, I.,

Ron, E., Modan, M., Peretz, H., and Modan, B. (Med. Neurol. Branch, Natl. Inst. Health, Bethesda, Md. 20205). Ann. Neurol. 8:261, 1980.

In Israel between the years 1949 and 1960, approximately 20,000 children ranging from one to 15 years of age were irradiated for eradication of tinea capitis. Radiation to the cerebral hemispheres ranged up to 140 rads. Of those treated, now adults, 44 patients were chosen for analysis of their visual evoked responses. A control group of 57 subjects similar in age and ethnic origin was chosen. The analyses detected significant differences between the visual evoked response averages in study subjects compared with controls. These differences may reflect delayed, probably permanent functional damage to the central nervous system caused by small doses of x-radiation to immature normal human brain. The left hemispheres appeared to be more involved, consistent with the fact that these children received, on the average, more x-radiation to the left side of the brain, as was shown in simulated phantom models. This disparity is reflected in a greater degree of difference between the left visual evoked responses of the irradiated versus control group. The major differences between the visual evoked responses occurred in the first part of the secondary response and suggest possible subcortical involvement. The results strengthen previous evidence of potential hazards of x-radiation in children. (3 figures, 1 table, 46 references)—David Shoch

Archives of Ophthalmology

CONJUNCTIVAL HYPEREMIA AND CORNEAL INFILTRATES WITH CHEMICALLY DISINFECTED SOFT CONTACT LENSES. Mondino, B. J., and Groden, L. J. (Dept. Ophthalmol., Eye and Ear Hosp., 230 Lothrop St., Pittsburgh, PA 15213). Arch. Ophthalmol. 98:1767, 1980.

Three patients wearing soft contact lenses for cosmetic purposes developed conjunctival hyperemia and anterior stromal infiltrates of the cornea. All microbiologic tests were negative and occlusive patch tests showed positive delayed hypersensitivity reactions and occlusive patch disinfectants in all three patients. Further patch tests with thimerosal suggested that this preservative was responsible for the reactions. (2 figures, 12 references)—David Shoch

ACQUIRED ORBITAL RETRACTION SYNDROME. Osher, R. H., Schatz, N. J., Duane, T. D. (Bascom Palmer Eye Inst., 900 NW 17th St., Miami, Fl. 33152). Arch. Ophthalmol. 98:1798, 1980.

Four patients with infiltrative orbital disease involving an extraocular muscle demonstrated a characteristic retraction-motility pattern. The cardinal feature was retraction of the globe on attempted gaze opposite the field of action of the involved muscle. Variable eye movement limitation was present in the field of action and opposite to the field of action of the involved muscle. When motility was restricted, the forced-duction test was positive. Modest proptosis and episcleral vascular congestion over the extraocular muscle insertion were associated physical findings. Dysthyroid eye disease, inflammatory myositis, and neoplasms are among the infiltrative myopathies that may produce an acquired orbital retraction syndrome. (10 figures, 14 references)—Authors' abstract

POLYVINYL ALCOHOL AS A PROTECTIVE COATING ON INTRAOCULAR LENSES. Olson, R. J., Kolodner, H., Morgan, K. S., Escapini, H., Sevel, D., and Kaufman, H. E. (Div. Ophthalmol., Univ. Utah School of Med., 50 N. Medical Dr., Salt Lake City, Ut 84132). Arch. Ophthalmol. 98:1840, 1980.

Sixty-eight intraocular lenses were implanted in cats to compare the efficacy of a new polyvinyl alcohol coating in protecting the corneal endothelium from lens-cell contact damage. The mean endothelial density after implantation of the coated lens was significantly higher than the cell density in those eyes receiving the uncoated lens. Whole-eye histopathologic study disclosed no signs of inflammation or toxicity related to the coating. Ten coated lenses were placed in balanced salt solution or perfused with balanced salt solution at 0.5 mL/min. The coating remained intact for 60 minutes, affording corneal endothelial protection for this period. In a rabbit anterior chamber toxicity study, using balanced salt solution as a control, no

differences in reaction in the anterior chamber or intraocular pressure were noted. (1 figure, 4 tables, 11 references)—Authors' abstract

British Journal of Ophthalmology

MANAGEMENT OF OPTIC NERVE GLIOMAS. Wright, J. E., McDonald, W. I., and Call, N. B. (Orbital Clin., Moorfields Eye Hosp., City Road, London EC1V 2PD). Br. J. Ophthalmol. 64:545, 1980.

Seventeen patients thought to have orbital optic nerve gliomas when first seen have been reviewed after up to 12 years. Enlargement of the optic canal was present in 15 of the 16 patients examined, but this finding was unreliable as an indicator of the posterior extent of the tumor. Nine patients had a stable course with little change over a period of up to eight years; there was optic atrophy in all and neurofibromatosis was relatively common (7/9). Eight patients showed progressive enlargement of the tumor; six had swollen discs, and the incidence of neurofibromatosis was relatively low (3/8). The optic nerve was excised in seven of the latter group. Biopsies of the optic nerve taken from the region of maximal enlargement were difficult to interpret and unhelpful in planning management. Radical surgery should be reserved for the minority of patients in whom there is progressively enlarging tumor without evidence of chiasmal involvement. (2 figures, 4 tables, 19 references)—Authors' abstract

PRIMARY OPTIC NERVE MENINGIOMA. Wright, J. E., Call, N. B., and Liaricos, S. (Orbital Clin., Moorfields Eye Hosp., City Road, London EC1V 2PD). Br. J. Ophthalmol. 64:553, 1980.

Twenty-seven patients with a meningioma arising from within the optic nerve sheath have been seen during the past 12 years of whom 21 were women, the majority aged 39–64 years. The men were younger, all except one being between 20 and 41 years when first seen. There were no patients younger than 20 years. Twenty-two patients had noticed a deterioration in vision as their initial symptom. The optic nerve head was abnormal in all patients; 14 were swollen and 13 atrophic and flat. Neurofibromatosis was not seen in any patients in this group. Treatment was essentially surgical. (3 figures, 1 table, 11 references)—E. Lee Stock

Canadian Journal of Neurological Sciences

GIANT AXONAL NEUROPATHY: VISUAL AND OCULOMOTOR DEFICITS. Kirkham, T. H., Guitton, D., and Coupland, S. G. (Montreal Neurol. Hosp., Neuro-Ophthalmol. Dept., Room 201, 3801 University St., Montreal, Quebec H3A 2B4). Can. J. Neurol. Sci. 7:177, 1980.

Giant axonal neuropathy, a generalized disorder of neurofilaments, presents as a chronic, progressive peripheral neuropathy in childhood. Evidence for central nervous system involvement is demonstrated in this study of four male patients with giant axonal neuropathy who had defective visual function and abnormal ocular motility. The visual system was studied by electroretinography, which showed normal retinal function, and by visual evoked potentials, which showed disease of both optic nerves and retrochiasmal visual pathways. The ocular motility disorder, studied by electrooculography, comprised defective pursuit, inability to maintain eccentric gaze with gaze paretic and rebound nystagmus, abnormal optokinetic responses and failure of suppression of the vestibulo-ocular reflex by fixation. These findings suggested involvement by giant axonal neuropathy of the cerebellar and brain stem pathways important in the control of ocular motility. (8 figures, 3 tables, 37 references)—Authors' abstract

Journal of Clinical Investigation

GYRATE ATROPHY OF THE CHOROID AND RETINA WITH HYPERORNITHINEMIA: DEFICIENT FORMATION OF GUANIDINOACETIC ACID FROM ARGININE. Sipilä, I., Simell, O., Arjomaa, P. (Children's

Hosp., Univ. Helsinki, Helsinki, Finland). J. Clin. Invest. 66:684, 1980.

Patients with gyrate atrophy of the choroid and retina have 10- to 20-fold increased ornithine concentrations in body fluids and significantly reduced activity of ornithine aminotransferase in lymphocytes and cultured fibroblasts. The authors administered intravenous arginine to six patients and six controls to study in vivo inhibition by high ornithine concentrations of arginine-glycine transamidinase, the rate-limiting enzyme in creatine synthesis. Serum arginine concentration curves after administration were similar for the two groups. The increment in serum ornithine was more than three times as great in patients as in controls. There was also diminished excretion of guanidinoacetate and creatine excretion in the effected group. The authors conclude that in gyrate atrophy formation of guanidinoacetate and creatine is inhibited at the transamidination step by high concentrations of ornithine. Deficiency of the high-energy phosphates may underlie the pathogenesis of the eye and muscle atrophies. (3 figures, 1 table, 13 references)—David Shoch

Journal Francais d'Ophtalmologie

RETROBULBAR INJECTION OF CHLORPROMAZINE IN THE ABSOLUTE GLAUCOMA. (French) Fiore, C., Lupidi, G., and Santoni, G. (Clinique Univ. d'Ophthalmology, Policlinico Monteluce, 06100 Perugia, Italy). J. Fr. Ophthalmol. 3:397, 1980.

The authors used retrobulbar chlorpromazine as an alternative to alcohol in the treatment of absolute glaucoma. One milliliter of procaine was given retrobulbarly followed by 1 ml (25 mg) of chlorpromazine using the same needle. They treated 63 eyes and had complete relief of pain in 71%. There were 18% failures which required enucleation. The only complication was one case of ptosis which resolved subsequently. There were no retrobulbar hemorrhages, keratitis, entropion or permanent ptosis. The authors conclude that chlorpromazine gives a better effect with fewer complications than retrobulbar alcohol. (1 table, 6 references)—E. Lee Stock

Lancet

EFFECTS OF PRESCHOOL SCREENING FOR VISION AND HEARING ON PREVALENCE OF VISION AND HEARING PROBLEMS 6–12 MONTHS LATER. Feldman, W., Sackett, B., Milner, R. A., Gilbert, S. (Dept. Pediatr., McMaster Univ. Med. Ctr., Hamilton, Ont. L8N 3Z5). Lancet 2:1014, 1980.

In order to determine whether children screened 6–12 months previously for vision and hearing had fewer vision and hearing problems than a non-screened cohort, a trained nurse-tester examined 763 screened and 743 non-screened kindergarten children matched for age, sex, and socio-economic status. The screened cohort had significantly fewer vision problems (10%) than the unscreened (15%). The difference for moderately severe problems (visual activity 20/50 (6/15) or worse) was also significant. Fifty-eight percent more screened than unscreened children were wearing glasses. The screened cohort had more hearing problems (16.8%) than the non-screened group (14%), but the difference was not statistically significant. Preschool hearing screening, unlike vision screening, is not associated with improved end results. (1 figure, 2 tables, 11 references)—Authors' abstract

Neurology

DIABETIC OCULOMOTOR PARESIS WITH PUPIL FIXED TO LIGHT. Gilmore, P. C., and Carlow, T. J. (Veterans Admin. Med. Ctr., Neuro-Ophthalmol. Sec. 2100 Ridgecrest S.E., Albuquerque, N.M. 87108). Neurology 30:1229, 1980.

A 51-year-old man with a ten-year history of diabetes complained of double vision. He showed a rather typical right third nerve lesion except that the right pupil measured 5 mm and did not react to a bright light. A space occupying lesion was under consideration but investigation of this diagnosis was delayed by the fact that the pupils reacted briskly upon convergence. In this case the light-near dissociation and diabetic ocular motor palsy combined to simulate a pupillary fixed third nerve disorder. It is important to

test the near pupil reflex to avoid invasive tests that a fixed pupil might suggest in third nerve palsies. (1 figure, 6 references)—David Shoch

Ophthalmologica

COMPLICATIONS IN 1,000 CONSECUTIVE INTRACAPSULAR CATARACT EXTRACTIONS. François, J. and Verbraeken, H. (Ophthalmol. Clin., Univ. Ghent, Ghent). Ophthalmologica 180:121, 1980.

Four surgeons performed all the cases here described. The most significant operative complications were capsule rupture in 6.5% and vitreous loss in 3%. The most significant postoperative complications were delay in wound closure in 2.7%, iritis in 2.9%, ocular hypertension in 3.4%. Late complications were Irvine-Gass syndrome in 2.8%, epithelial downgrowth in the anterior chamber in 0.6% and retinal detachment in 2%. Alpha-chymotrypsin caused a temporary ocular hypertension in about one-fourth of the cases. If there was no pre-existing ocular pathology 90% of the cases achieved vision of 6/9 (20/30) or better. (11 tables, 3 references)—David Shoch

THE EFFECT OF NATURAL AND ARTIFICIAL LIGHT VIA THE EYE ON THE HORMONAL AND METABOLIC BALANCE OF ANIMAL AND MAN. Hollwich, F., and Diekhues, B. (Antonienstrasse 1, D-8000 Munchen 40, Germany). Ophthalmologica 180:188, 1980.

Color change in the plaice and frog and the development of testes in the duck are induced by light stimuli via the eye. That these effects are due to action on the endocrine system is further suggested by the authors as a result of experiments in which they measured the blood ACTH and cortisol levels in patients both permanently and temporarily blind. In both of these groups the levels were significantly lower than in normals and in those patients temporarily blinded by cataracts the levels rose after cataract extraction. A further study on healthy persons showed that if they worked in artificial light for long periods of time there were stress-like levels of ACTH and cortisol present in the blood. The authors feel that this may account for the agitated, mental and physical behavior of children who spend long days in artificial illumination with marked deviation from the normal spectral distribution of sunlight. They therefore suggest that the spectrum of artificial illumination be similar to that in natural light. (15 figures, 31 references)—David Shoch

Radiology

COMPUTED TOMOGRAPHY OF THE OPTIC CHIASM. Daniels, D. L., Haughton, V. M., Williams, A. L., Gager, W. E., and Berns, T. F. (Dept. Radiol., Milwaukee County Med. Complex, 8700 W. Wisconsin Ave., Milwaukee, Wisc. 53226). Radiology 137:123, 1980.

Using a CT/T 8800 scanner, axial and coronal images of the optic chiasm were obtained in a cadaver, 41 normal individuals, and six patients with symptoms of chiasmal glioma. The size and shape of each chiasm were determined. The normal chiasm appeared to be U- or boomerang-shaped in axial sections, and U- or dumbbell-shaped in coronal sections. The average normal chiasm diameters were 1.8 cm (transverse) by 0.8 cm (anteroposterior) by 0.4 cm (vertical). The chiasm was globular and larger than 0.6 cm (vertical dimension) in all cases of chiasmal glioma. The abnormal chiasm can be distinguished accurately from the normal chiasm by computed tomography. (8 figures, 6 references)—Authors' abstract

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

FRENCH SOCIETY OF OPHTHALMOLOGY CONGRESS

The Congress of the French Society of Ophthalmology will be held in Paris, May 10–14, 1981. For further information, write Secretariat, Société Française d'Ophtalmologie, 9, rue Mathurin-Régnier, 75015 Paris, France.

SIXTH INTERNATIONAL CONGRESS OF NEUROGENETICS AND NEURO-OPHTHALMOLOGY

The Sixth International Congress of Neurogenetics and Neuro-Ophthalmology will be held June 9–12, 1981, in Zurich, Switzerland. For further information, write Prof. Dr. A. Huber, Stadelhoferstrasse 42, CH-8001, Zurich, Switzerland.

AMERICAN ASSOCIATION FOR PEDIATRIC OPHTHALMOLOGY AND STRABISMUS: SEVENTH ANNUAL MEETING

The American Association for Pediatric Ophthalmology and Strabismus will hold its seventh annual meeting, May 7–10, 1981, in Orlando, Florida. For further information, write Ms. Tricia Stevens, Coordinator, The American Association for Pediatric Ophthalmology and Strabismus, 5700 W. Grace St., Suite 109, Richmond, VA 23226.

COLLEGE OF MEDICINE AND DENTISTRY OF NEW JERSEY AND EYE INSTITUTE OF NEW JERSEY: SIXTH INTRAOCULAR LENS COURSE

The College of Medicine and Dentistry of New Jersey and the Eye Institute of New Jersey are sponsoring the Sixth Intraocular Lens Course, April 4, 1981, in Newark, New Jersey. The fee is $75. For further information, write Ms. Sharon Dobrina, Medical Education/Ophthalmology, 15 S. Ninth St., Newark, NJ 07107.

WILMER INSTITUTE: DIAGNOSIS AND MANAGEMENT OF RETINAL VASCULAR AND MACULAR DISORDERS

The Retinal Vascular Center of the Wilmer Institute will celebrate its 11th anniversary with a course entitled "Diagnosis and Management of Retinal Vascular and Macular Disorders" at Big Sky, Montana, July 12–17, 1981. For further information, write Big Sky Course Coordinator, 17 Turner, 720 Rutland Ave., Baltimore, MD 21205.

COLLEGE OF MEDICINE OF THE PENNSYLVANIA STATE UNIVERSITY: PRACTICAL ASPECTS OF OFFICE NEURO-OPHTHALMOLOGY

The College of Medicine of the Pennsylvania State University will sponsor a neuro-ophthalmology seminar on May 15, 1981. For further information, write Continuing Education, Dept. 4004, Milton S. Hershey Medical Center, Hershey, PA 17033.

SEVENTH COLBY POST-GRADUATE OPHTHALMOLOGY SEMINAR: INTERNAL INFLAMMATORY DISORDERS OF THE EYE

Colby College, in conjunction with the Mid-Maine Medical Center, Waterville, Maine, will present its seventh annual post-graduate Ophthalmology Seminar Aug. 9–13, 1981. For further information, write Dr. Kevin Hill, Special Programs, Colby College, Waterville, ME 04901.

UNIVERSITY OF SOUTH CAROLINA: THE EYE IN FAMILY MEDICINE

The University of South Carolina will present a continuing medical education program, The Eye in Family Medicine, April 25, 1981. The fee is $35. For further information, write Jeri McClain, Administrative Assistant, Office for Academic Affairs, USC School of Medicine, Columbia, SC 29208.

The Page and William Black Post-Graduate School of Medicine: Indirect Ophthalmoscopy and Peripheral Retinal Disorders

The Department of Ophthalmology and The Page and William Black Post-Graduate School of Medicine of the Mount Sinai School of Medicine announce a postgraduate course, Indirect Ophthalmoscopy and Peripheral Retinal Disorders, May 1 and 2, 1981, in New York, New York. For further information, write Director, The Page and William Black Post-Graduate School of Medicine, Mount Sinai School of Medicine, One Gustave L. Levy Place, New York, NY 10029.

Yale Alumni in Ophthalmology: Annual Educational Meeting

The Yale Alumni in Ophthalmology will hold their annual educational meeting June 5, 1981, in New Haven, Connecticut. For further information, write Yale Alumni in Ophthalmology, c/o David Silverstone, M.D., Temple Medical Center, 60 Temple St., New Haven, CT 06510.

Fight for Sight, Inc.: 1980–1981 Awards

Fight for Sight, Inc., announced the following 1980–1981 Grants-in-Aid, Emergency Awards, and Children's Eye Clinic Awards:

Fight for Sight, Inc., Awards 1980–1981

Grants-In-Aid

Investigator and Institution	Project Title	Amount Awarded
John H. Chen, Ph.D. New York University Dental Center New York, New York	Molecular studies of opsin mRNA from eye retinas	$8,800
Darlene Dartt, Ph.D. Tufts University Boston, Massachusetts	Role of calcium-dependent regulator protein in lacrimal gland secretion	7,000
Katherine V. Fite, Ph.D. University of Massachusetts Amherst, Massachusetts	The origin of retinal dystrophy. An experimental analysis utilizing embryonic eye transplantation	5,550
Shao-Ling Fong, Ph.D. Baylor College of Medicine Houston, Texas	Vitamin A storage and metabolism in human eye	6,300
Jack V. Greiner, Ph.D. University of Illinois Chicago, Illinois	Mucus secretory vesicles in nongoblet epithelial cells of the conjunctiva	3,600
Juan Grunwald, M.D. Scheie Eye Institute Philadelphia, Pennsylvania	Laser doppler measurement of blood flow in the optic nerve	8,000
Joann M. Hashimoto Massachusetts Eye & Ear Infirmary Boston, Massachusetts	Metabolic aspects of aqueous humor outflow	3,120
Bor-Shyue Hong, Ph.D. Texas Tech University Lubbock, Texas	Structure, function and properties of a distinctive type of collagen in the cornea	5,000

Investigator and Institution	Project Title	Amount Awarded
Ronald Kalil, Ph.D. University of Wisconsin Madison, Wisconsin	Effects of esotropia and exotropia on the response properties of lateral geniculate neurons in the cat	$5,000
Ehud Kaplan, Ph.D. The Rockefeller University New York, New York	Input-output relationships in the monkey LGN	7,000
Edward Kean, Ph.D. Case Western Reserve University Cleveland, Ohio	Adsorptive endocytosis of macromolecules by the pigment epithelium, influence of terminal carbohydrates on the process	5,000
M. Cristina Kenney, Ph.D.* University of California Davis, California	Rabbit corneal endothelial cells in vitro. Possible relationship in pathological states	8,000
Kenneth R. Kenyon, M.D.* Eye Research Institute Boston, Massachusetts	Experimental xerophthalmia and keratomalacia in vitamin A deficiency	8,500
Robert Knighton, Ph.D. Douglas Anderson, M.D. University of Miami Miami, Florida	Visual function in glaucoma	8,500
Michael S. Korey, M.D. Washington University St. Louis, Missouri	Compliance with topical pilocarpine therapy	5,000
David W. Meltzer, M.D. Washington University St. Louis, Missouri	Energy dispersive X-ray analysis of intraocular lenses	6,000
Joseph F. Metcalf, Ph.D. University of California Berkeley, California	Protection from experimental ocular herpetic keratitis by a heat-killed virus vaccine. Mechanism of the protective effect	5,000
Richard Pino, Ph.D.* Louisiana State University New Orleans, Louisiana	Permeability of rat choriocapillaris and Bruch's membrane	6,500
Kathryn S. Pokorny, Ph.D. Mount Sinai School of Medicine New York, New York	Fine structure and cytochemistry of the eye in senile macular degeneration	7,500
James M. Schaeffer, Ph.D. University of California La Jolla, California	Biochemical studies of retinal degeneration diseases	8,000

*Continuation Award

Investigator and Institution	Project Title	Amount Awarded
David J. Schanzlin, M.D. University of Southern California Los Angeles, California	The histopathology of corneal neovascularization	$5,000
Paul A. Sieving, M.D. University of Illinois Chicago, Illinois	Human early receptor potential (ERP) studies of several hereditary retinal and choroidal diseases	7,000
Gareth A. Tabor, Ph.D. Baylor College of Medicine Houston, Texas	Melanin turnover in the mammalian retinal pigment epithelium	7,534
Vernon L. Towle, Ph.D. New England Medical Center Hospital Boston, Massachusetts	Assessment of optic nerve damage from glaucoma and ocular hypertension using visually evoked potentials and contrast sensitivity thresholds	5,500
Robert J. Ulshafer, Ph.D. Baylor College of Medicine Houston, Texas	The role of cyclic nucleotides in retinal metabolism and photoreceptor degeneration	4,545
William Woodward, Ph.D. University of Oregon Portland, Oregon	Morphologic and functional changes in visual pathways induced by the neurotoxin kainic acid	5,000

Emergency Awards
(Following June Review Meeting)

Anthony M. Gawienowski, Ph.D. University of Massachusetts Amherst, Massachusetts	Investigation of monoamine oxidase in eye tissue	$3,000
S. Robert Hilfer, Ph.D. Temple University Philadelphia, Pennsylvania	Early ocular development	5,300

Clinical Research Service Projects

Continuation awards to the Fight For Sight Children's Eye Clinics to help defray the annual maintenance of these facilities.

Institution	Director of Clinic	Amount Awarded
Bascom Palmer Eye Institute University of Miami Miami, Florida	John T. Flynn, M.D.	$11,500
Columbia-Presbyterian Medical Center New York, New York	Howard M. Eggers, M.D.	22,424

INSTITUTION	DIRECTOR OF CLINIC	AMOUNT AWARDED
Eye Institute of New Jersey Newark, New Jersey	Anthony R. Caputo, M.D.	$25,000
Eye and Ear Hospital of Pittsburgh Pittsburgh, Pennsylvania	David A. Hiles, M.D.	13,624
The Wills Eye Hospital Philadelphia, Pennsylvania	Joseph H. Calhoun, M.D. and Donelson R. Manley, M.D.	30,750

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4 COURSES—EACH 3 DAYS LONG
LIMITED TO 2 OPHTHALMOLOGISTS FOR EACH COURSE

April 28th, 29th, 30th, 1981
May 26th, 27th, 28th, 1981
June 23rd, 24th, 25th, 1981
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Introductory Lectures, Live surgery—2 days
Cooper Medical Center, Camden, N.J. Underwood-Memorial Hospital, Woodbury, N.J.

Post-operative examination of 50 cases, 6 years to 1 day post-op
Copeland, 2 & 4 Loop Binkhorst, Medallion, Platina, Choyce, Shearing, Simcoe, Sheets, Sinsky and Kratz
(Experience: 1800 IOL Implantations)

TUITION: NONE

Only a sincere interest in intraocular lenses with a clinical practice of significant magnitude to utilize training
CME Category 1 Credit 27 Hours

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Attention: **Mrs. G. Sheridan**

FIFTY-FOURTH ANNUAL
NEW YORK EYE AND EAR INFIRMARY
CLINICAL CONFERENCE
MAY 7 and 8, 1981

MARRIOTT'S ESSEX HOUSE
NEW YORK CITY

"OPHTHALMOLOGY 1981"

JAMES E. BURRIS, M.D., Program Chairman

EXTENDED WEAR LENSES—*Moderator:* Frank B. Hoefle, M.D.
Speakers: Joseph A. Baldone, M.D., Jorge N. Buxton, M.D., James J. Koverman, Miguel Refojo, D.Sc.

THYROID OPHTHALMOPATHY—*Moderator:* Hampson A. Sisler, M.D.
Speakers: Robert Chase, M.D., William Cooper, M.D., Robert Day, M.D., Joseph Ogura, M.D., Robert Schimek, M.D., John T. Simonton, M.D., Stephen Trokel, M.D.

GLAUCOMA—*Moderator:* James E. Burris, M.D.
Speakers: Alfonse A. Cinotti, M.D., Max Forbes, M.D., G. Peter Halberg, M.D., Bernard Kronenberg, M.D., George Spaeth, M.D.

INTRAOCULAR LENSES—*Moderator:* John R. Finlay, M.D.
Speakers: Leeds E. Katzen, M.D., Larry G. Leiske, M.D., Steven P. Shearing, M.D., John H. Sheets, M.D.

AMA-CME Credits, Category 1: 16

Conference fee: $200.00 (Luncheons included) Residents: $100.00 (Luncheons included)
Dinner Dance, Promenade Cafe, Rockefeller Center: $50.00 per person

For registration and further information, please write:

Jane Stark, Conference Registrar,
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310 East Fourteenth Street,
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CATARACTS: SURGERY, COMPLICATIONS & APHAKIC CORRECTIONS

A Symposium On the Options & Problems of Modern Cataract Surgery

The Washington Hospital Center, Washington, D.C.

April 10 & 11, 1981 • (Cherry Blossom Season)

FACULTY

Joel M. Engelstein, M.D., Program Director

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Jack Hartstein, M.D.	C. William Simcoe, M.D.	Lorenz Zimmerman, M.D.
David Hiles, M.D.	Casimir Swinger, M.D.	

SUBJECTS

Optics of Aphakic Spectacles, Contact Lenses & Intraocular Lenses • Pediatric Cataracts & Aphakic Management • Intracapsular vs. Extracapsular Cataract Extraction • Intraocular Lenses: Iris Fixation, Anterior & Posterior Chamber • Pathology Of Aphakic & Pseudophakic Complications • Contact Lenses: Hard, Soft & Extended Wear • Refractive Keratoplasty • Astigmatism: Avoidance & Correction • Surgical Complications: Glaucoma, Infection, Retinal Detachment, Vitreous Management, Corneal Edema • Cystoid Macular Edema

An AMA-CME 12 Hour Accredited Course
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Please make checks payable to: W.H.C.—Cataract Symposium

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THIRD CANADIAN CONTACT LENS CONFERENCE TORONTO, CANADA

Sponsored by Ophthalmic Educational Seminars in cooperation with the Contact Lens Association of Ophthalmologists, Inc. (USA) and Centennial College

Four Seasons Yorkville Hotel in the fashionable Bloor-Yorkville area, Toronto
Friday and Saturday, May 1st and 2nd 1981

Plus added Cataract and Intraocular Lens meeting Sunday, May 3rd, 1981

OUTSTANDING INTERNATIONAL GUEST FACULTY:

Maxwell K. Bochner Memorial Lecture: HERBERT KAUFMAN, M.D., NEW ORLEANS
Overseas Visiting Lecturer: THOMAS SPRING, M.D., MELBOURNE, AUSTRALIA

James Aquavella, M.D., Rochester, N.Y.
Joseph Baldone, M.D., New Orleans, LA.
Ronald Barnet, M.D., Phoenix, AZ.
Elliott Blaydes, M.D., Bluefield, WV.
Henry Brent, M.D., Toronto, ONT.
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John Morgan, M.D., Kingston, ONT.
Bernard Slatt, M.D., Toronto, ONT.
Joseph Soper, F.C.L.S.A., Houston, TX.
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This conference will include the latest advances in contact lens technology, gas permeable lens, fitting and care systems for extended wear lenses, problem solving for both hard and soft contact lenses, orthokeratology, the management of high astigmatism and keratoconus with contact lenses and surgical alternatives for correcting myopia.

Advanced registration fee for Contact Lens Conference May 1 & 2, 1981 $150.00
(Residents $50)

Added Cataract and Intraoclar Lens Update May 3, 1981 $50.00

This conference is approved for 11 hours AMA-CME credit, category I

For information write: Dr. HAROLD STEIN, CHAIRMAN
170 Bloor Street West • The Park Plaza Hotel • Toronto, Canada M5S 1T9

Saturday June 20, 1981
St. Mary's Hospital in cooperation with the
Canadian Implant Association
The Queen Elizabeth Hotel
Montreal Quebec Canada
Lens Implant Complications
Radial Keratotomy
Surgery for Corneal Astigmatism
Guest speaker:
Professor Svatoslav N. Fyodorov

Speakers:
Robert Azar, M.D.
D. Boyaner, M.D.
R. D. Binkhorst, M.D.
Miles Galin, M.D.
M. L. Kwitko, M.D.
David Hiles, M.D.
Leeds Katzin, M.D.
Steven Ostbaum, M.D.
Casimir A. Swinger, M.D.

Registration Fee: $125.00

Recent innovations in anterior segment surgery has led to intense interest in these modalities. The symposium is intended to bring together noted authorities in these new surgical fields to explore and evaluate their potential. Pediatric lens implantation, errors in lens power calculation, treatment of excess astigmatism, surgical treatment of myopia, treatment and prevention of lens implant complications will all be discussed.

For information write:
Dr. Marvin L. Kwitko, Program Chairman
5591 Cote des Neiges Road, Suite 1
Montreal, Quebec, Canada. H3T 1Y8

The University of Southern California School of Medicine
Presents a
POSTGRADUATE COURSE
DIAGNOSIS AND MANAGEMENT OF MACULAR AND RETINAL VASCULAR DISEASES

GUEST FACULTY
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The course is oriented for the practicing clinician. The common, but leading, causes of visual loss, e.g., senile macular degeneration, diabetic retinopathy, etc., will be considered in detail as well as a wide range of fungus disorders. The clinical presentations and diagnostic evaluations will be well illustrated with color photographs, fluorescein angiography, and other diagnostic tests. The prognosis of the natural course as well as the indications for treatment will be discussed. The results, complications, and contraindications will be emphasized. Informality with free interchange will characterize the meeting.

28 Category I AMA/CMA hours
Tuition: $350.00
For further information please contact:
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School of Medicine, Postgraduate
Division, 2025 Zonal Avenue,
Los Angeles, CA 90033,
Phone (213) 224-7051

JUNE 20-27, 1981
Mauna Kea Beach Hotel • Kamuela, Hawaii

THE RETINA RESEARCH FUND OF ST. MARY'S HOSPITAL AND MEDICAL CENTER, SAN FRANCISCO
announces SPRING 1981

RETINA WORKSHOPS

YOSEMITE NATIONAL PARK
Ahwahnee Lodge, March 26, 27, 28, 1981

&

CARMEL VALLEY
Quail Lodge, May 21, 22, 23, 1981

Course Instructors:
Howard Schatz, M.D.
Lawence J. Singerman, M.D.
Richard E. Goldberg, M.D.

Enrollment: Limited to 40 at each workshop
Course Time: 3 days, 4 hours each day
Credit: 12 hours, AMA and CMA, Category 1

Subject: Practical problems and recent advances in the diagnosis, management, and treatment of retinal, macular, and vitreous diseases. Real case studies and photographs will be utilized in a **problem-solving workshop atmosphere.**

For information and brochure call 415/921-4860 or write: Medical Staff Secretary, St. Mary's Hospital and Medical Center, 450 Stanyan, San Francisco, CA 94117

APRIL 9th & 10th, 1981
LENOX HILL HOSPITAL
New York City

THE RED EYE
Diagnosis and Management

This seminar will be devoted to a comprehensive overview of the diagnosis and management of the Red Eye resulting from external and intraocular infections and inflammations. A distinguished panel of experts will discuss the latest advances in the diagnosis, pathogenesis and treatment, emphasizing the practical aspects of management. Clinical case problems submitted by panelists and audience will be analyzed at each session.

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Stuart I. Brown, M.D.
Robert S. Coles, M.D.
Richard K. Forster
Peter Laibson, M.D.
Bruce Ostler, M.D.
Michael B. Starr, M.D.
Frederick H. Theodore

REGISTRATION FEE: $200.00 Residents: $75.00

FOR INFORMATION WRITE:

Lori Dioszeghy
Department of Ophthalmology
Lenox Hill Hospital
100 East 77th Street
New York, New York 10021

**This course is approved for 16 credit hours in Category I towards the Physicians Recognition Award of the A.M.A.



The University of Texas Medical School at Houston
Department of Ophthalmology
is conducting a two day accrediting seminar and workshop in

OCULAR AND ORBITAL ULTRASONOGRAPHY

Hermann Eye Center, Houston, Texas
Thursday and Friday, March 5 and 6, 1981

FACULTY

Charles Garcia, M.D., Course Director
University of Texas Medical School and M.D. Anderson Hospital and Tumor Institute, Houston, Texas

Richard D. Binkhorst, M.D.
St. Vincent's Medical Center, New York, New York

Richard Dallow, M.D.
Harvard Medical School, Boston, Massachusetts

Yale Fisher, M.D.
Manhattan Eye, Ear and Throat Hospital, New York, New York

Richard Lewis, M.D.
Baylor College of Medicine, Houston, Texas

Stanley Handel, M.D.
U.T. Medical School at Houston, Houston, Texas

Mary Smith
Cornell University Medical School, New York, New York

TOPICS

Physics of Ultrasound • Contact Versus Immersion Scanning • Anterior Segment Pathology
Ultrasound Measurement and IOL Power Calculations: Intraocular Tumors, Evaluation and Management
Orbital Inflammation • Orbital Tumors • Vitreous and Retinal Pathology • Ocular Trauma and Foreign Bodies
Ultrasonography and Computerized Axial Tomography in Orbital Disease
Therapeutic Ultrasound • and Supporting Workshops

FEE: $250.00
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INFORMATION, REGISTRATION, RESERVATIONS
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Hermann Eye Center, 7th floor, University of Texas Medical School, Houston, Texas 77030
Approved for 14 credit hours Category I CME—Enrollment Limited

AJO-210

REGISTRATION FORM

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Fee enclosed_____ Bill me_____

5th ANNUAL OPHTHALMOLOGY CURRENT CONCEPTS SEMINAR '81

March 19-20, 1981

The Sheraton Inn and Conference Center
Madison, Wisconsin

Presented by:
University of Wisconsin School of Medicine

Department of Ophthalmology and University of Wisconsin

Continuing Medical Education

The conference will encompass all aspects of ophthalmologic medicine, featuring extensive workshop sessions, plenary sessions, and discussions.
Presentations will be made by the University of Wisconsin faculty and Joel Sugar, M.D., Abraham Lincoln School of Medicine, University of Illinois.
The program is accredited for 19 hours AMA Category I credit.

For further information contact:
Sarah Aslakson
University of Wisconsin
Department of Continuing Medical Education
610 Walnut Street, Madison, WI 53706
(608) 263-2856

Practical Aspects of Office Neuro-Ophthalmology

sponsored by The Pennsylvania State University College of Medicine • Division of Ophthalmology

MAY 15, 1981

Location:
The Milton S. Hershey Medical Center
Hershey, Pennsylvania

Guest Faculty:
H. Stanley Thompson, MD, University of Iowa
Joel Sacks, MD, University of Cincinnati

The Pennsylvania State University College of Medicine • Division of Ophthalmology Faculty:

Barton Hodes, MD
Joel Weinstein, MD
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Curriculum includes:
Ocular signs and symptoms of cerebrovascular disease—diagnosis and management; Differential diagnosis of the swollen disk; Visual field screening; Neuro-ophthalmologic diagnosis in children.

7 hours Category I Credit • Registration Fee: $95.00
For further information, contact:
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Intended for the vitreo-retinal surgeon with some experience.

Detailed presentations and discussions of surgical techniques, complications and instrumentation used in the management of traction retinal detachments.

Discussion of taped surgery.

Ample time for exchange of experiences.

All this in the beautiful spring environment of North Carolina from April 10 to 11, 1981.

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AMA Category I

Applications To:
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Duke University Eye Center
P.O. Box 3802
Durham, N.C. 27710
Phone: 919-684-3891

Registration Fee: $200.

THE DEPARTMENT OF OPHTHALMOLOGY AT THE UNIVERSITY OF IOWA SPONSORS AN INTERNATIONAL SYMPOSIUM ON STRABISMUS:

PROBLEM SOLVING IN STRABISMUS

Panel will be presented with common problems that are encountered in strabismus and will address themselves in short presentations to these questions. Panel discussions and questions from the audience will be included.

in Iowa City, Iowa, September 17-19, 1981

Course Coordinator: **William E. Scott, M.D.**

Faculty to include: Drs. Apis (Mexico), Ciancia (Argentina), deDecker (Germany), Harcourt (England), Helveston (Indiana), Jampolsky (California), Lang (Switzerland), McNeer (Virginia), Parks (Washington, D.C.), Pittar (Australia), Pratt-Johnson (Canada), Velez (Columbia).

Eye Research Institute of

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Announces its 9th Annual Course in

PRACTICAL ASPECTS OF PHOTOCOAGULATION

April 9, 10, and 11, 1981

The curriculum will encompass both the fundamental and practical aspects of xenon arc and argon laser photocoagulation and will consist of lecturers, clinical demonstrations, small seminars, and personal use of instruments.

LECTURE SUBJECTS INCLUDE:

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Principles/Techniques of Photocoagulation
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Clinical Results of Treatment
Complications of Photocoagulation
Potential New Uses for Lasers
Clinical Demonstrations
Diseases Treated by Photocoagulation

FACULTY:

Charles L. Schepens, M.D.
J. Wallace McMeel, M.D.
H. MacKenzie Freeman, M.D.
Ronald C. Pruett, M.D.
Felipe I. Tolentino, M.D.
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John J. Weiter, M.D.
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Lloyd M. Aiello, M.D.
Francois Delori, Ph.D.
Richard Simmons, M.D.
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Stephen R. Deppermann, M.D.
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REGISTRATION:

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Wills Eye Hospital
Philadelphia, Pennsylvania

David W. Vastine, M.D.
Pacific Medical Center
San Francisco, California

Lorenz E. Zimmerman, M.D.
Armed Forces Institute of Pathology
Washington, D.C.

DATES: April 3-5, 1981
LOCATION: Williamsburg Hospitality House, Williamsburg, Virginia

REGISTRATION FEE: $250.00
CREDIT: 15½ Hours Category 1 CME Credit Approved

For additional information, please call or write:
Miss Davi Lang
Continuing Medical Education
Medical College of Virginia
Box 48, MCV Station
Richmond, Virginia 23298
(804) 786-0494

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APRIL, 24, 25, 1981

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FOR INFORMATION CONTACT COURSE REGISTRAR
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Manor Building Suite 400,
Chicago, Illinois 60657
Course Fee: $450

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Inquiries, along with a current curriculum vitae and bibliography should be sent to:

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Professor and Chief
Division of Ophthalmology
Pennsylvania State University
Medical School
Milton S. Hershey Medical Center
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Department of Ophthalmology
University Hospitals & Clinics
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External Disease Fellowship

Stipend: $15,000.00 per year

Number: 2

Requirements:

1) Curriculum vitae and bibliography
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3) Summary of proposed project
4) Deadline for application is April 15, 1981

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Walter Stark, M.D., Johns Hopkins University
Denis M. O'Day, M.D., Vanderbilt University
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Contact:

Walter Stark, M.D.
Johns Hopkins University
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OPHTHALMOLOGIST: 30-years-old, married, completing vitreo-retinal fellowship, excellent university residency. Seeks general practice opportunity involving some retinal-vitreous work. All areas considered. Box 407 AJO

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OPHTHALMOLOGIST: Completing prestigious Boston residency July 1981. Mature, hard-working, compassionate with extensive clinical, microsurgical and research experience. All practice arrangements considered. Part time academic affiliation preferred. Box 410 AJO

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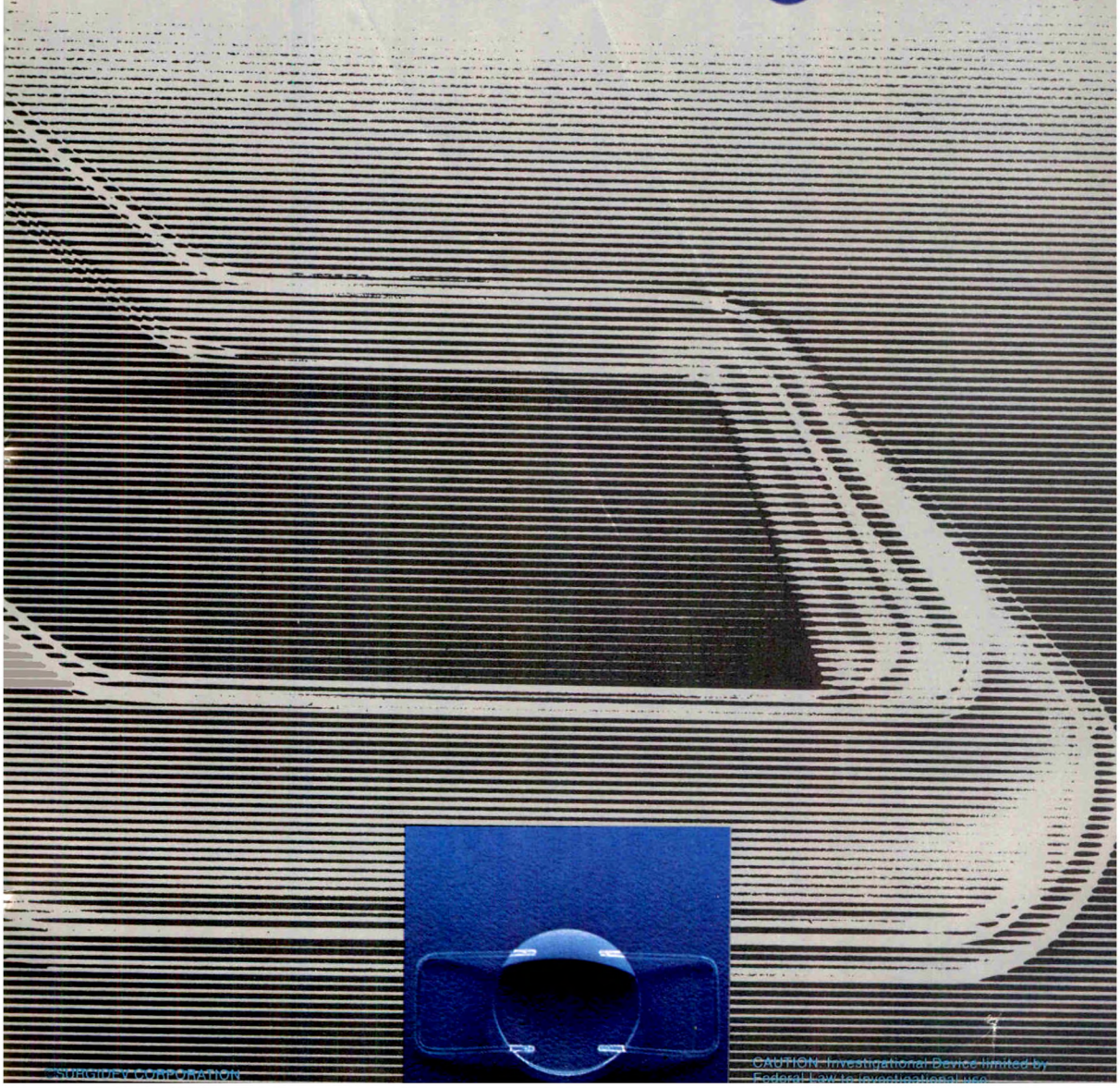
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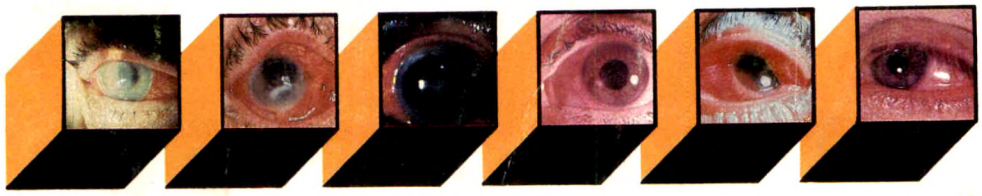
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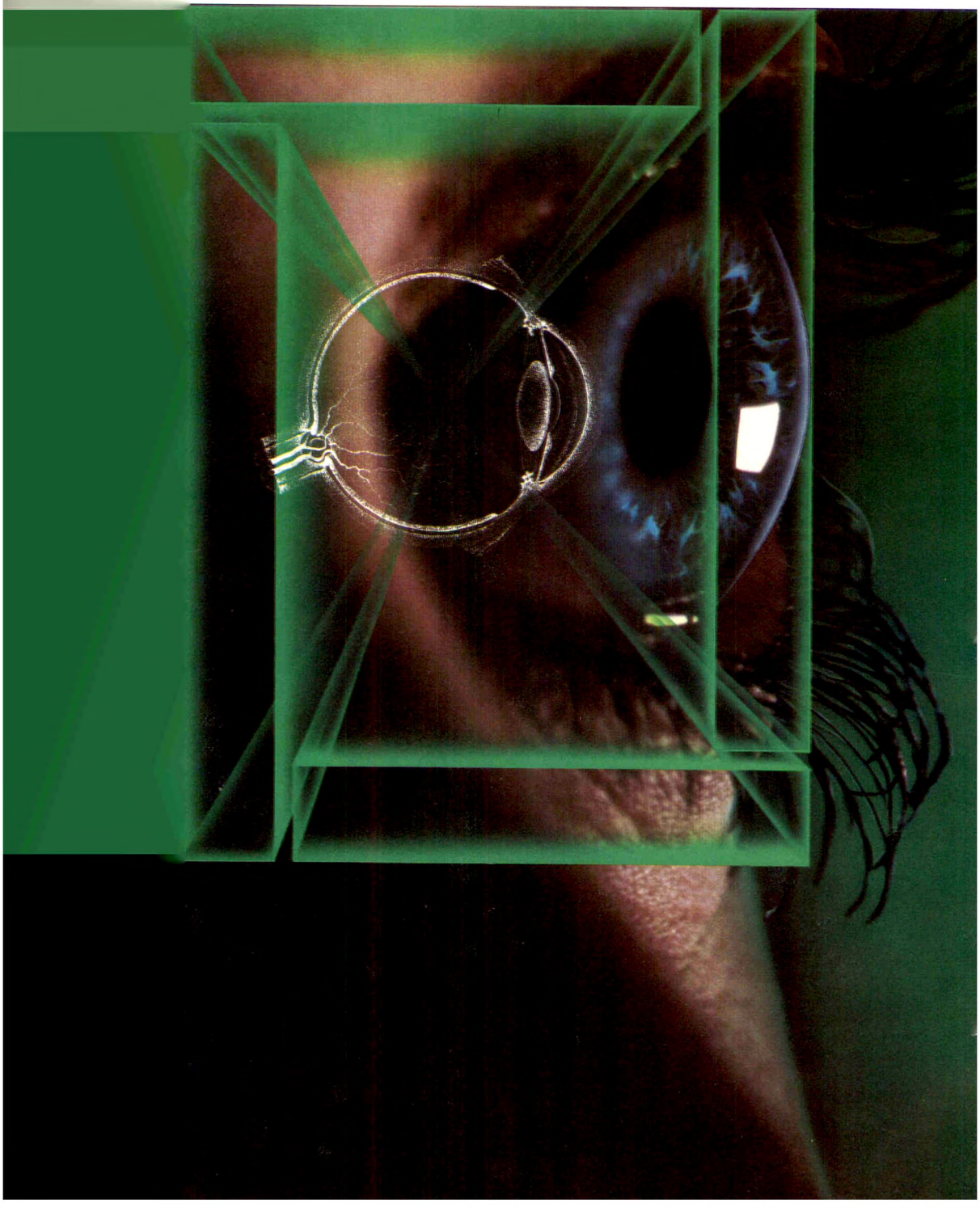
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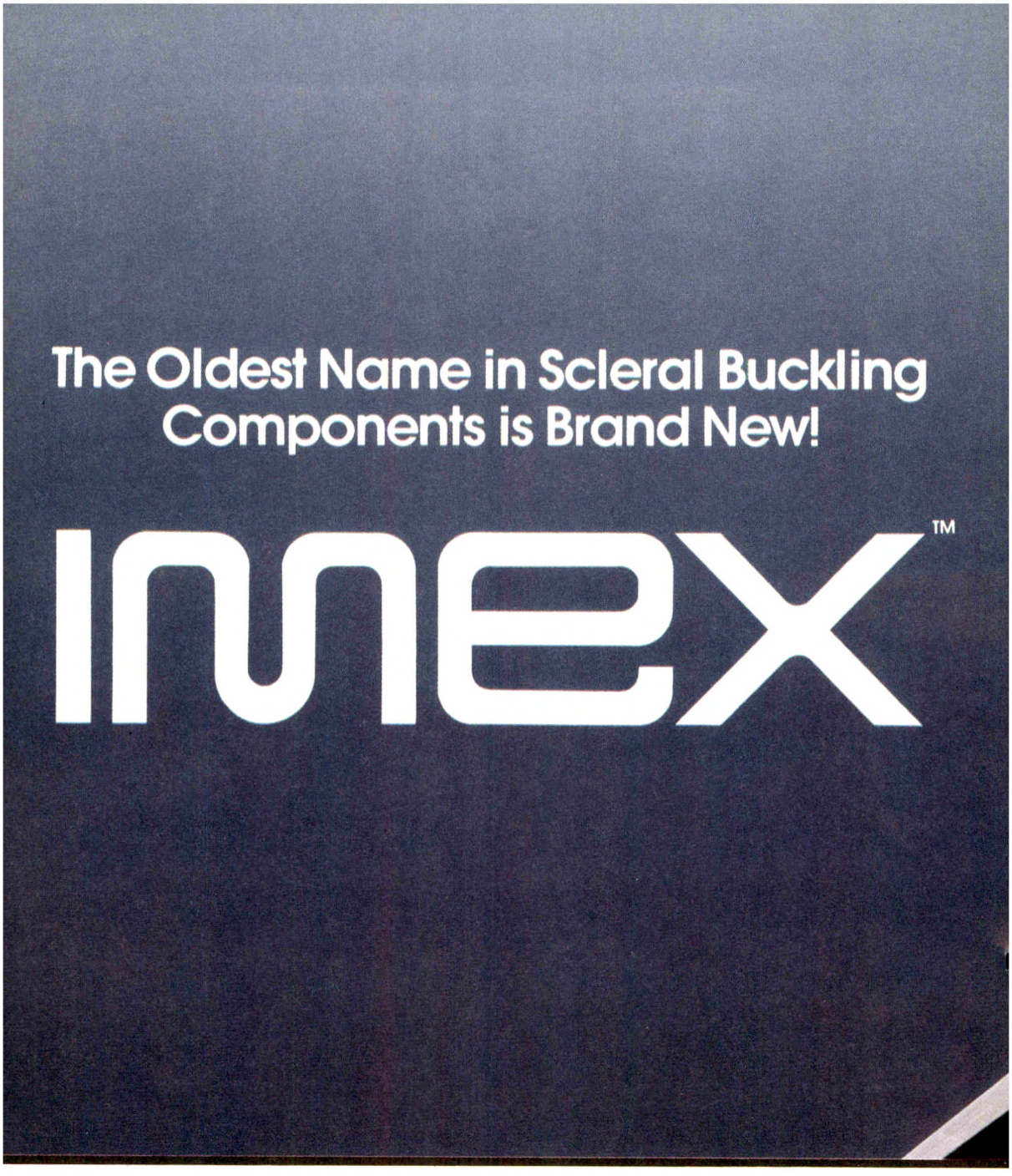
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1. Data on file at Alcon Laboratories.
2. Zimmerman, T. J., Ophthalmology Audio Digest, Vol. 17, No. 1

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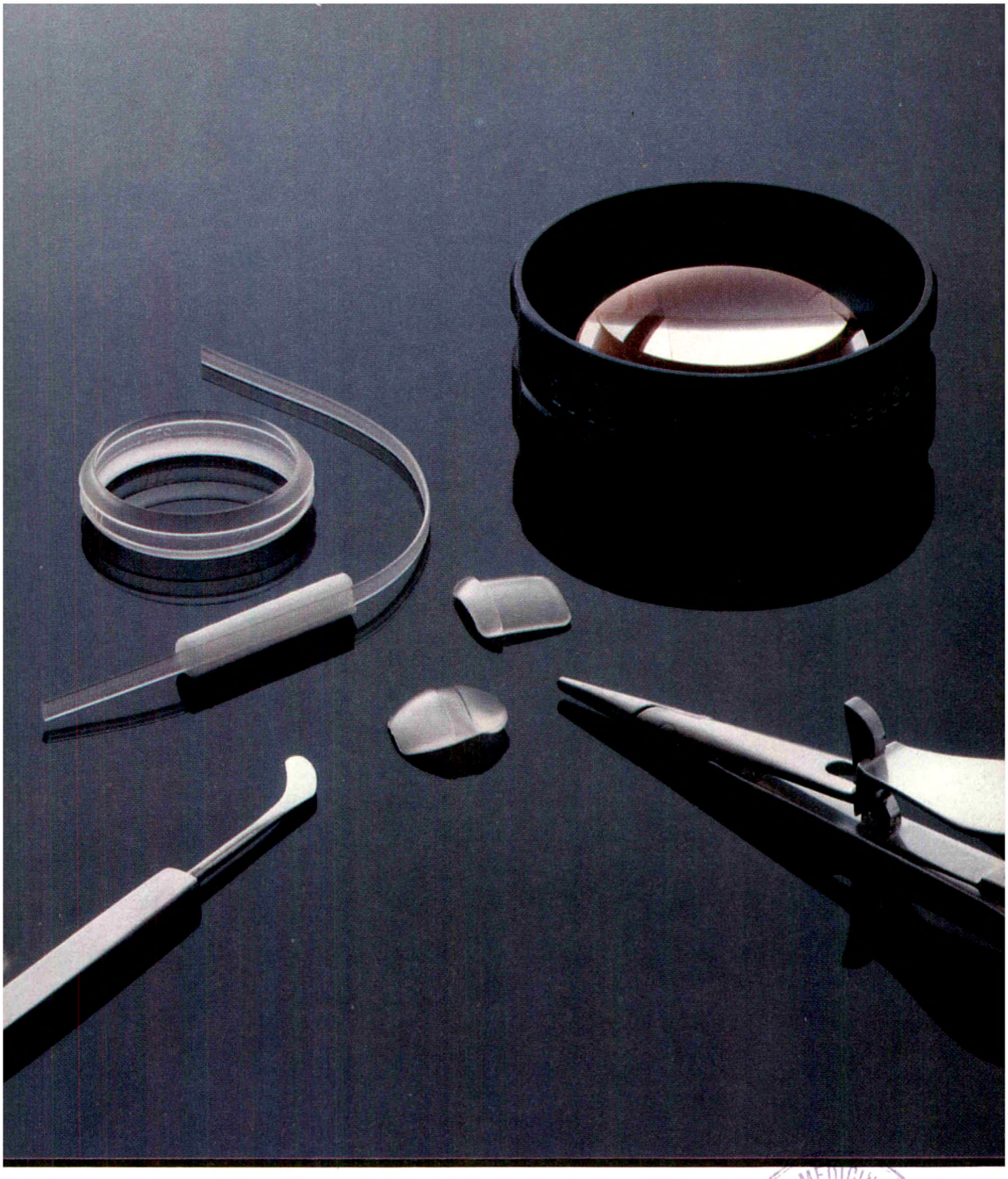
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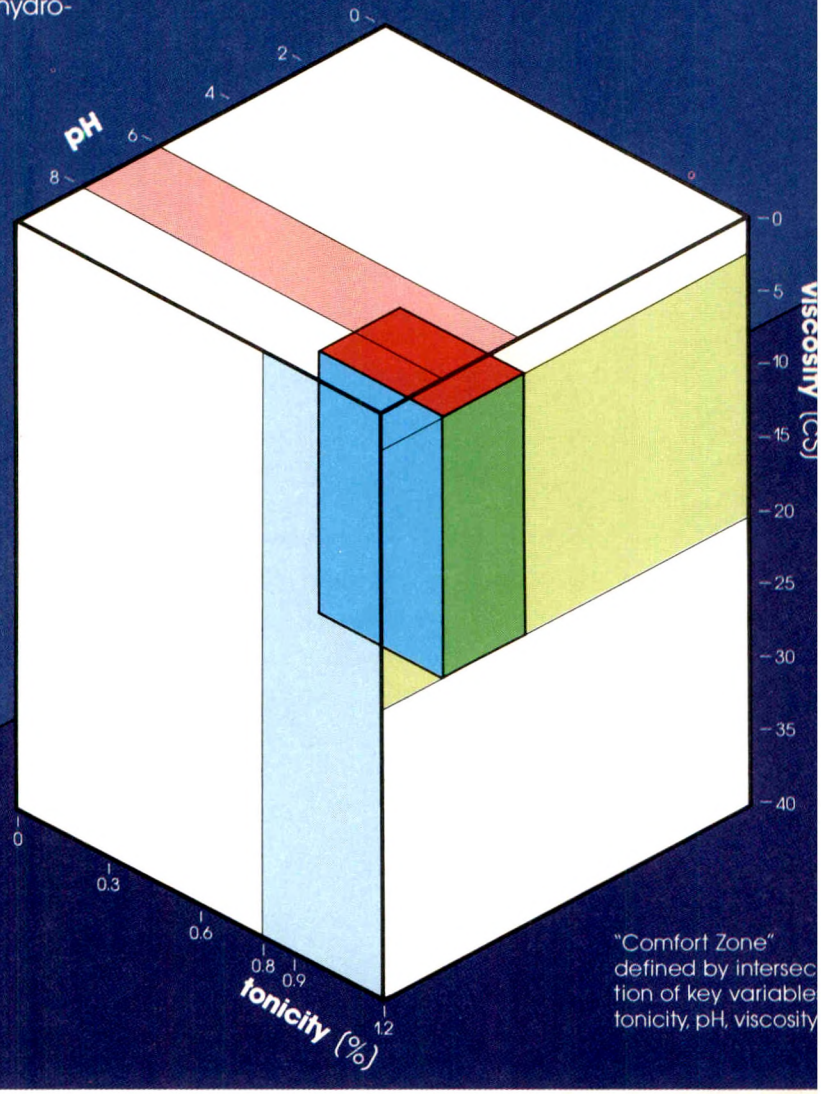
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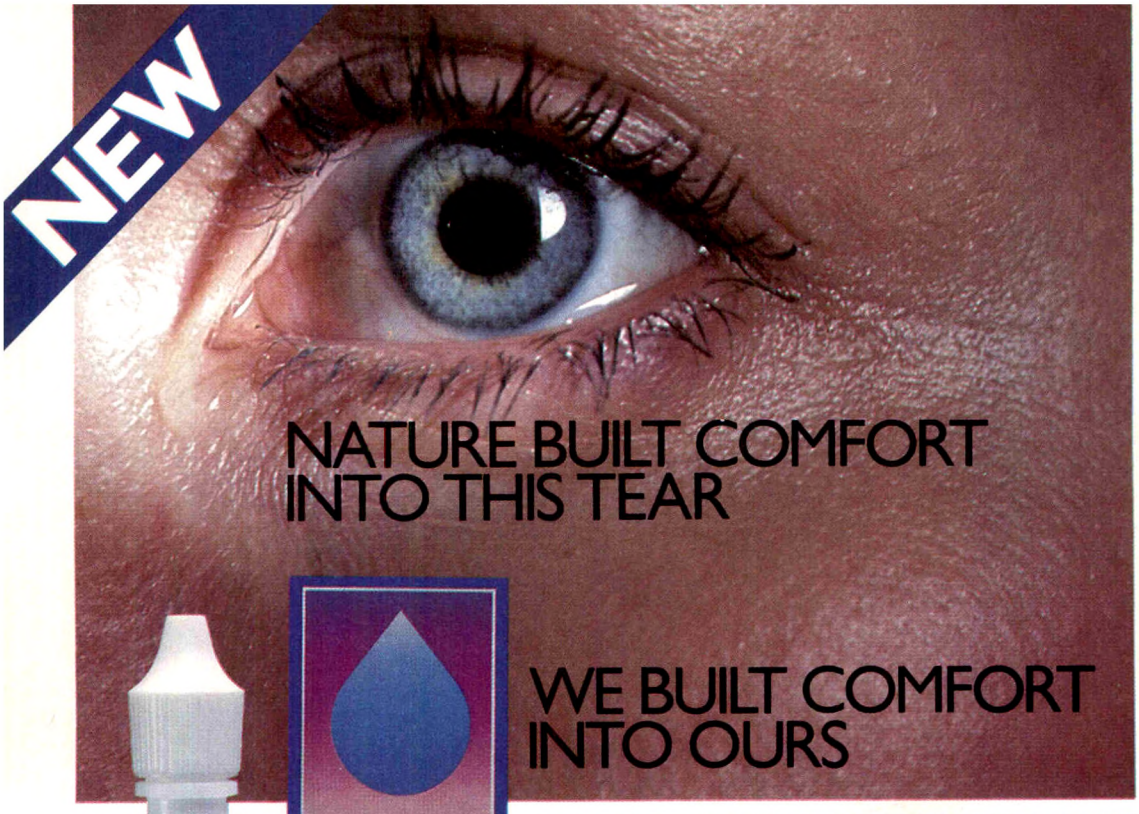
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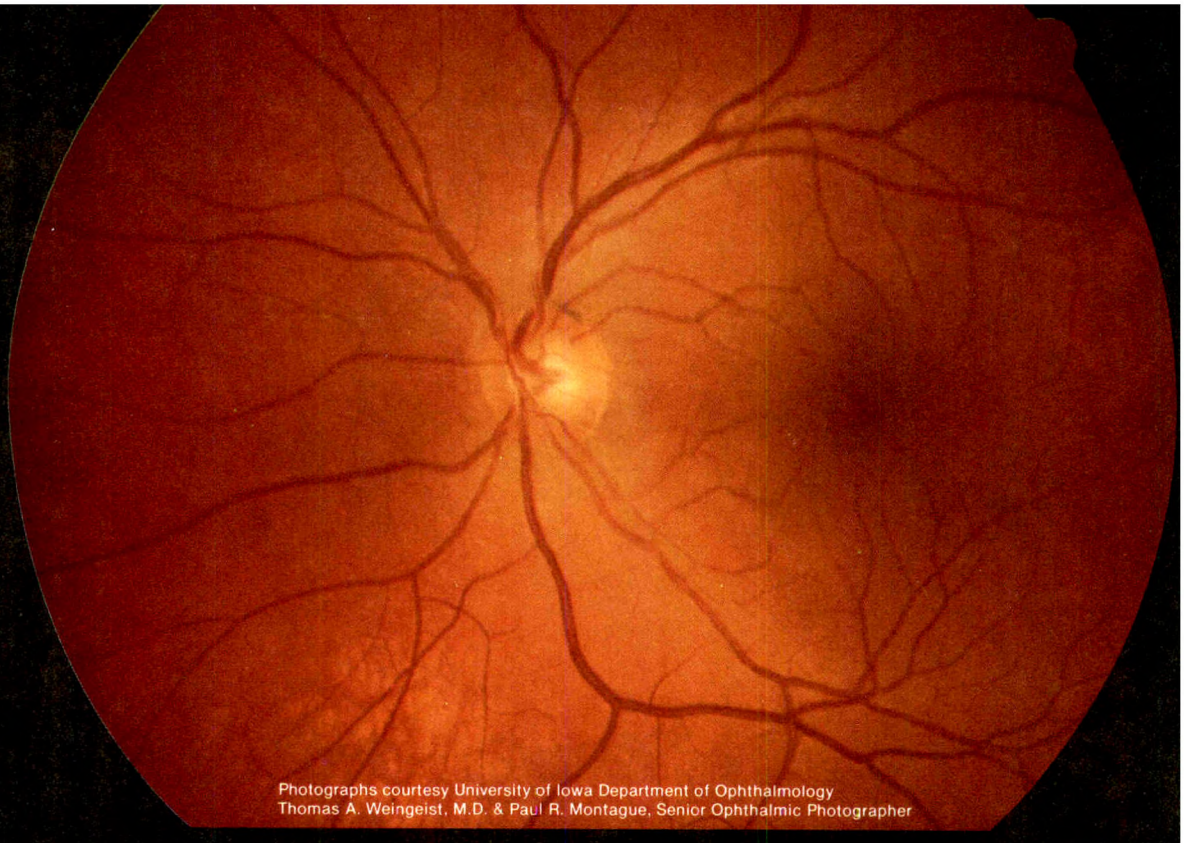
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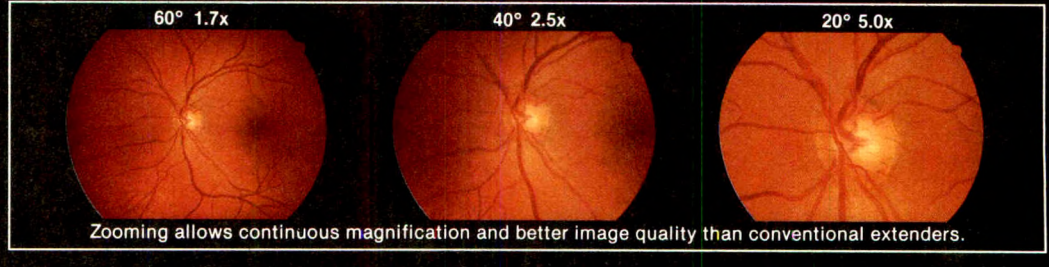
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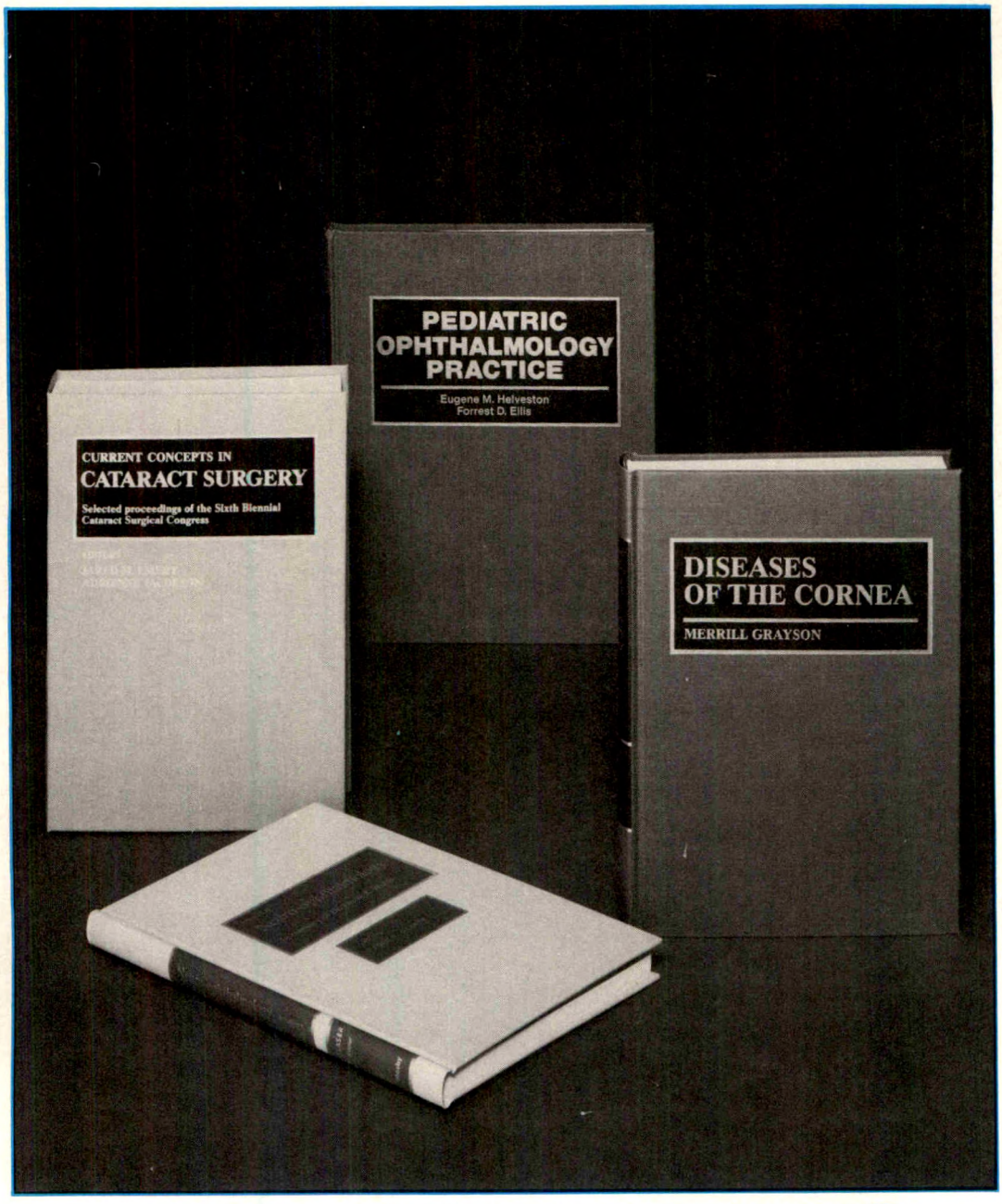
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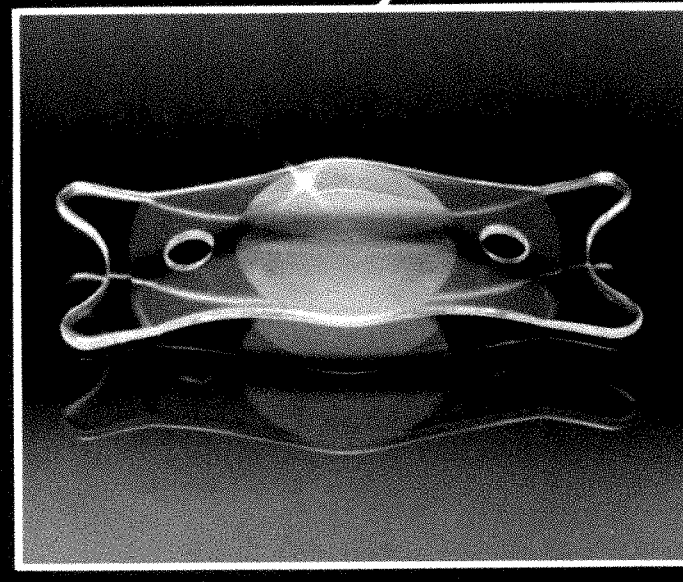
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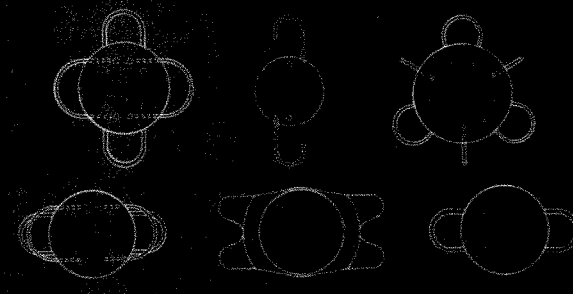
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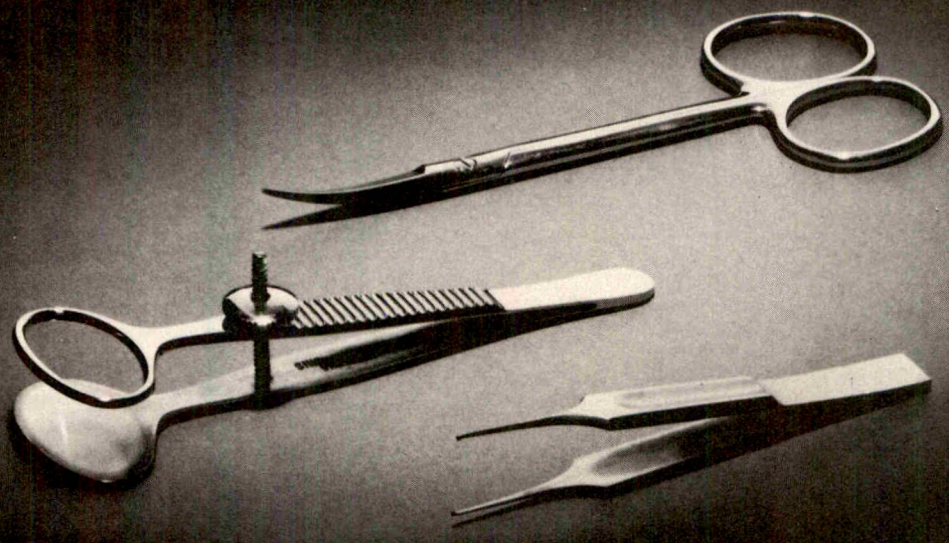


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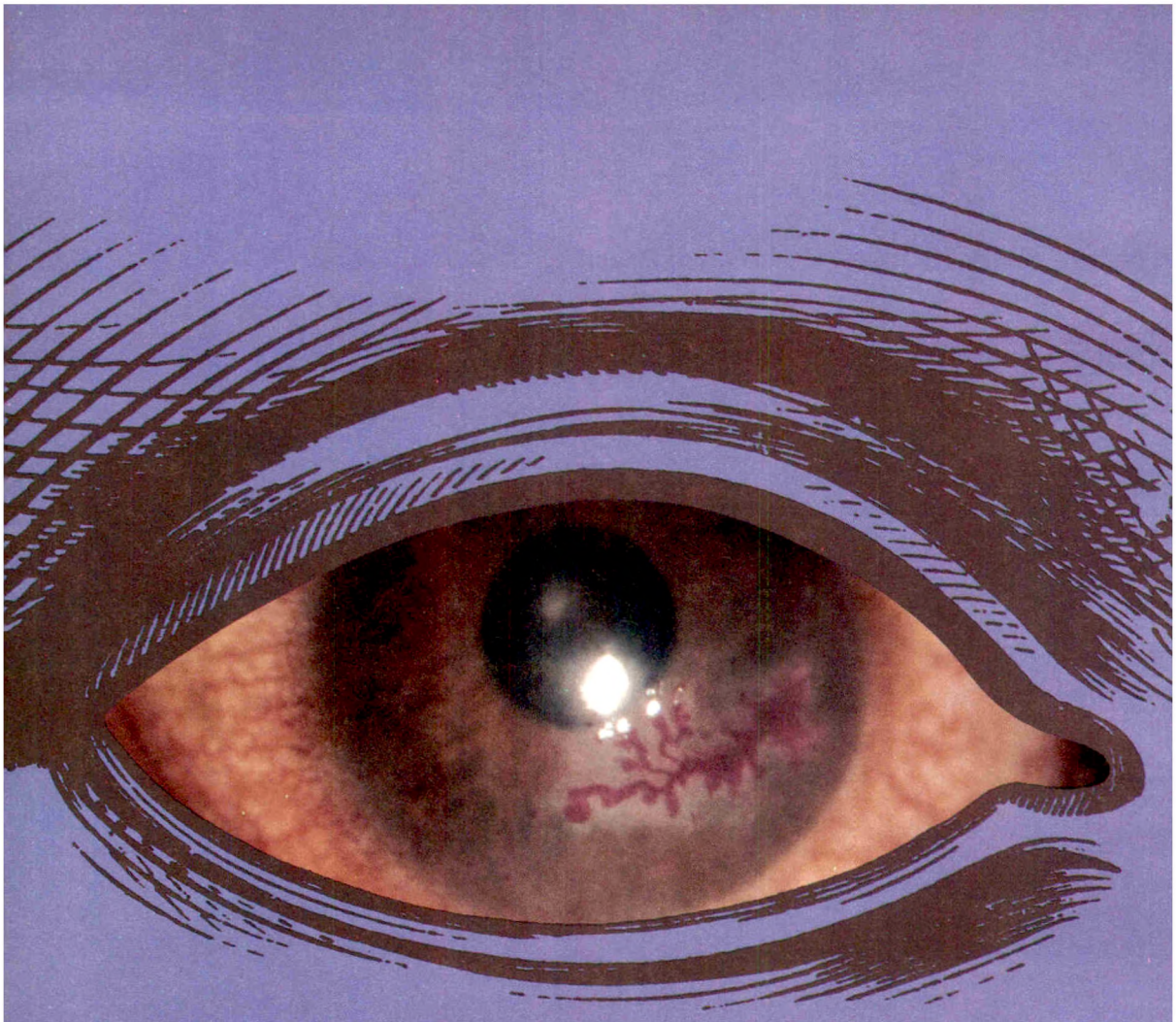
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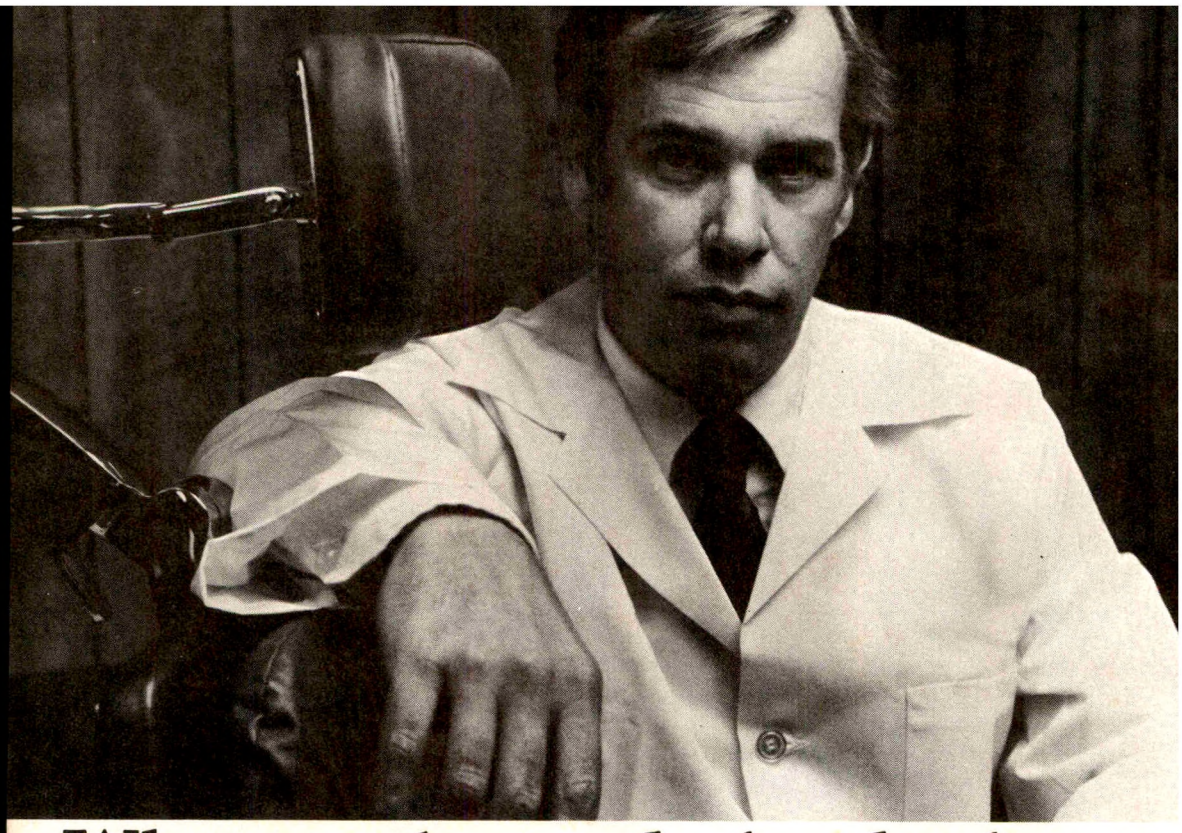
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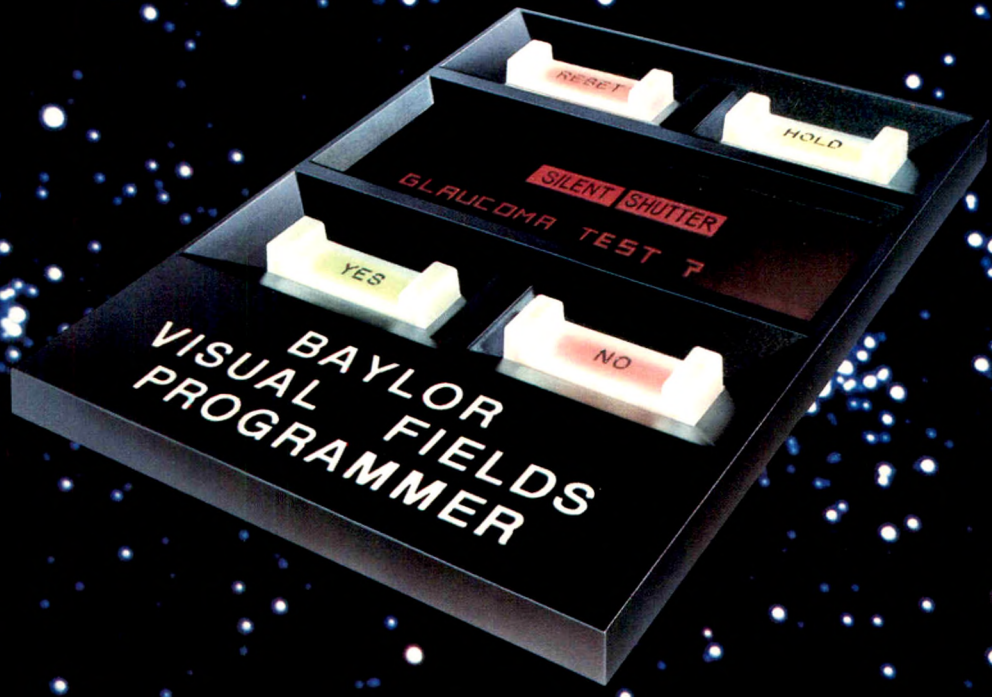
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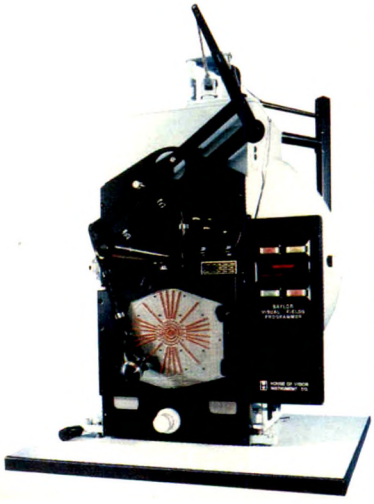
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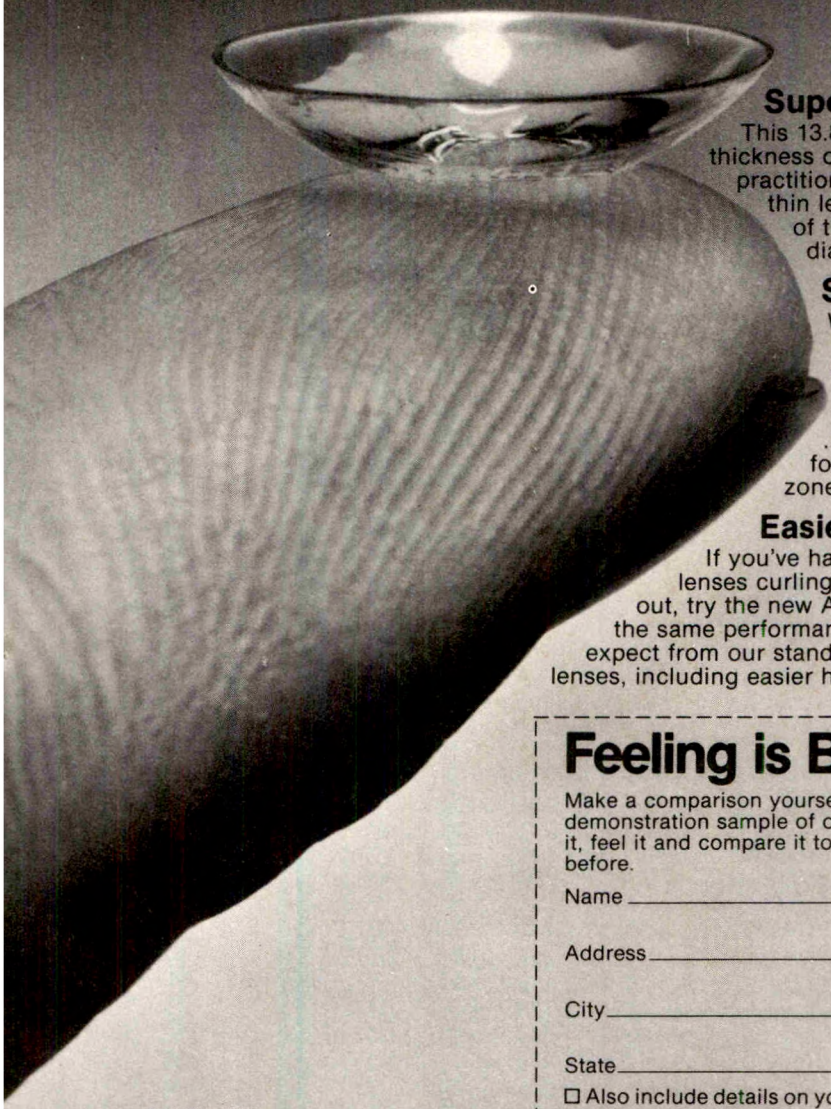
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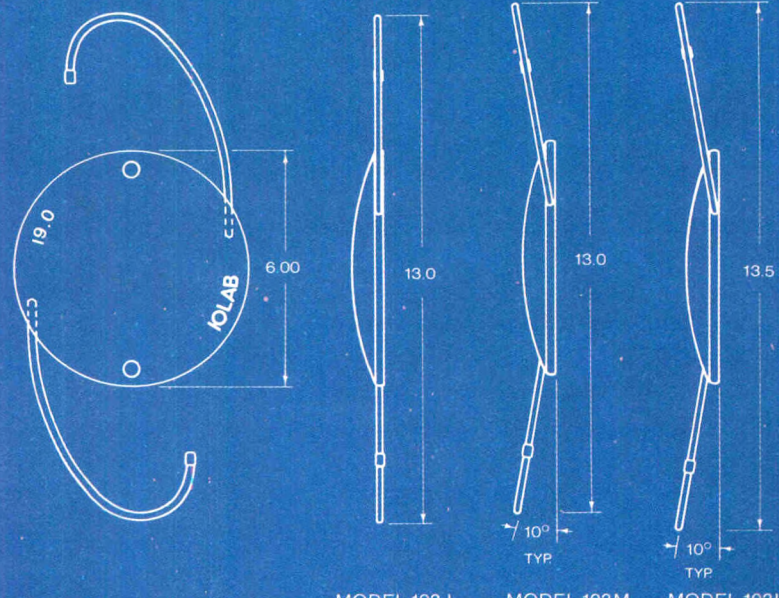
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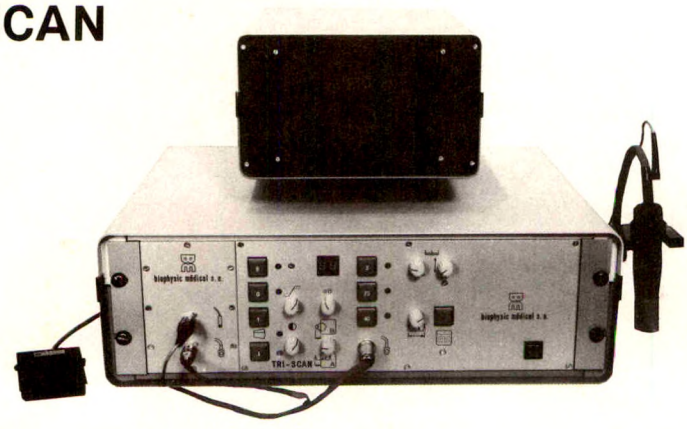
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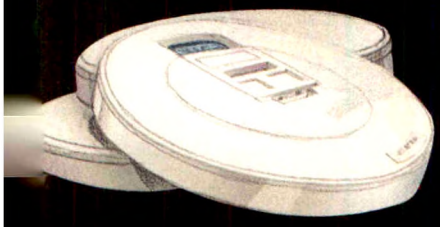
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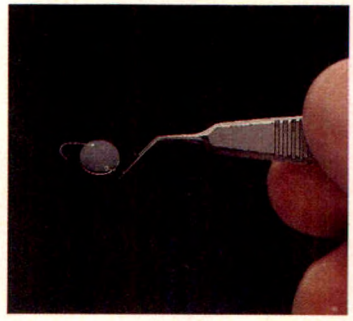
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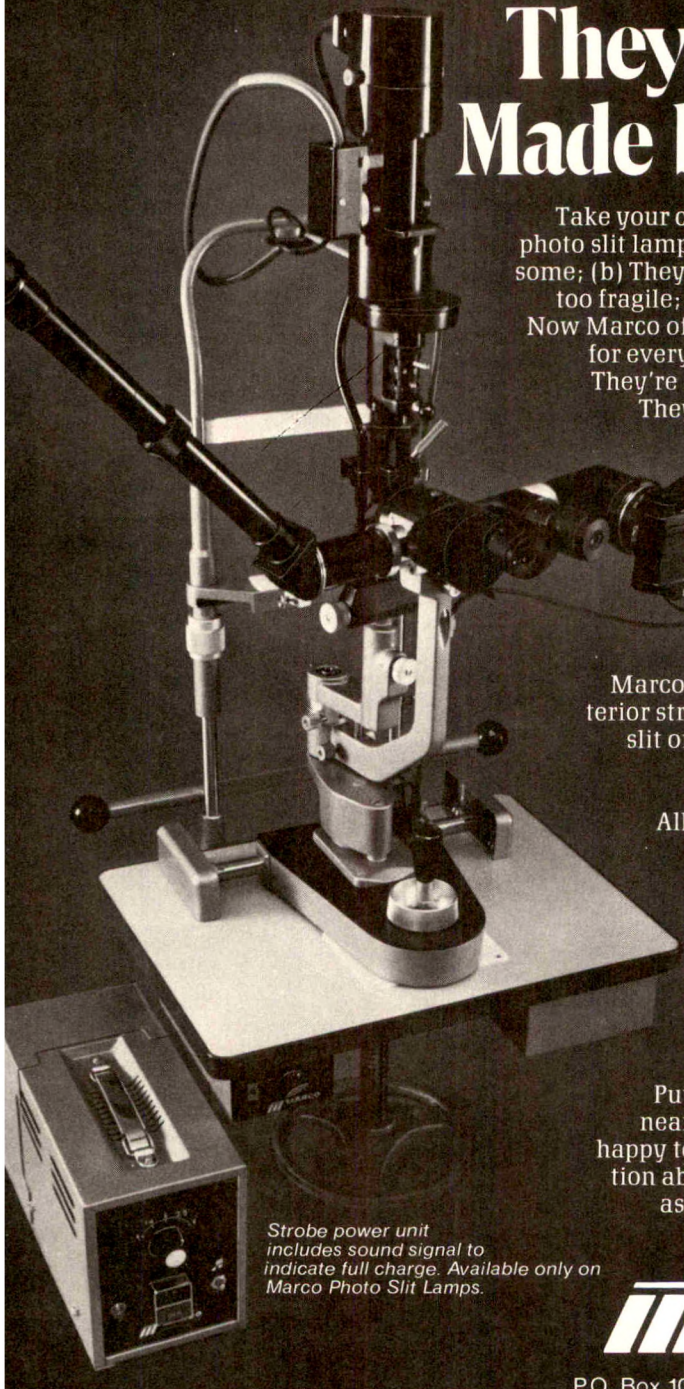
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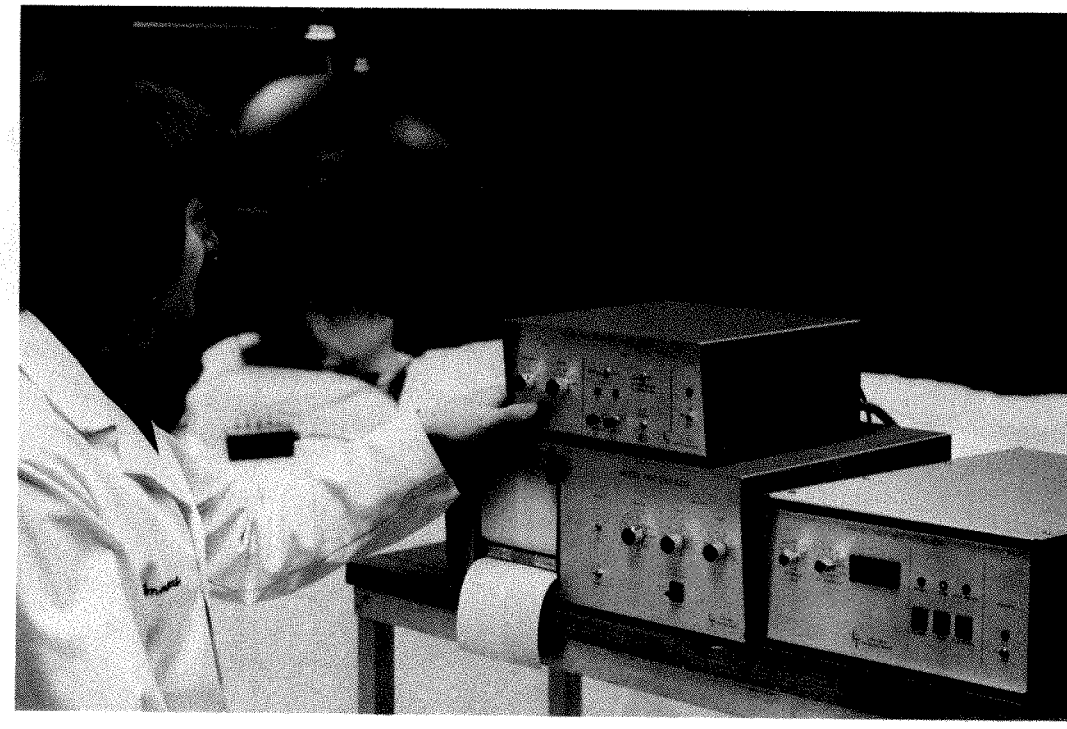
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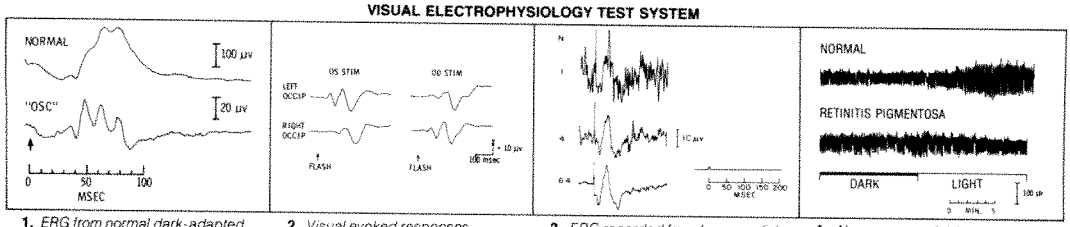
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VISUAL ELECTROPHYSIOLOGY TEST SYSTEM



1. *ERG from normal dark-adapted eye. Above: "Normal" mode; Below: "OSC" mode, gain increased. In "OSC" mode the response is filtered to emphasize the oscillatory potential.*

2. *Visual evoked responses recorded from right and left occiputs (dual channel system). The patient has an optic chiasm lesion.*

3. *ERG recorded from lower eyelid with averaging. "N"s at left of traces indicate the number of responses averaged.*

4. *Above: normal EOG with light-peak /dark-trough ratio of 2.4. Below: abnormal EOG in retinitis pigmentosa with light-peak / dark-trough ratio of 1.0. (EOG mode, 0.5 mm chart speed).*

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Permalens®
(perfilcon A)

Hydrophilic Contact Lens For Extended Wear

DESCRIPTION

The **Permalens®** (perfilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The perfilcon A material is a terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone, and methacrylic acid, with ethyleneglycol dimethacrylate as a crosslinking agent. When fully hydrated in buffered normal saline, the water content is 71%. The refractive index is 1.38 and light transmission is at least 96% for the visible light spectrum. For the minus lenses the base curve ranges from approximately 7.7 mm to 8.6 mm, with a diameter of approximately 14.0 mm. Center thickness ranges from 0.1 mm to 0.24 mm. For the plus lenses the base curve ranges from approximately 8.0 mm to 8.9 mm, with a diameter of approximately 14 mm to 14.5 mm. Center thickness ranges from 0.3 mm to 0.43 mm. The gas permeability of **Permalens®** (perfilcon A) Hydrophilic Contact Lens has been determined to be: 42.0×10^{-11} ($cm^2/sec/ml\ O_2/ml \times mmHg$ at 36°C. The percent equivalent oxygen performance has been determined to be approximately 11.5% for a **Permalens®** (perfilcon A) Hydrophilic Contact Lens of 0.17 mm center thickness.

PERMALENS® (perfilcon A) Hydrophilic Contact Lenses are available in powers from −20.00 to +35.00 diopters.

ACTIONS

In its hydrated state, the **Permalens®** (perfilcon A) Hydrophilic Contact Lens is soft and pliable. When placed on the eye, the hydrated lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS (Uses)

The **Permalens®** (perfilcon A) Hydrophilic Contact Lens is indicated for extended wear use to improve vision in patients with healthy eyes who are not aphakic and who have no more than 2 diopters of corneal astigmatism.

CONTRAINDICATIONS (Reasons not to use)

The **PERMALENS®** Hydrophilic Contact Lens should not be dispensed for vision improvement if the patient has any of the following conditions:

- Acute and subacute inflammation of the anterior segment of the eye.
- Any eye disease which affects the cornea or conjunctiva.
- Any active corneal infection: purulent (pus) bacterial, fungal, or viral infection.
- Insufficiency of lacrimal secretion (dry eyes).
- Corneal hypoesthesia (reduced corneal sensitivity).
- Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.
- Allergy to any ingredient in the solutions necessary for care of the lenses. **Thimerosal,** used as a preservative in many solutions, is one cause of such allergy.

WARNINGS

SERIOUS DAMAGE TO THE EYE AND LOSS OF VISION MAY RESULT FROM PROBLEMS WITH CONTACT LENSES. IMMEDIATELY CONSULT YOUR EYE CARE PRACTITIONER TO IDENTIFY THE CAUSE OF ANY UNEXPLAINED EYE DISCOMFORT, WATERING, VISION CHANGE, OR REDNESS OF THE EYE AND TO BEGIN ANY NECESSARY TREATMENT.

PRECAUTIONS

You must take the following precautions **to prevent damage to your eyes or to your Permalens®** Hydrophilic Contact Lenses:

- Always wash and rinse your hands before handling your lenses. Eye irritation may result if cosmetics, soaps, lotions, creams, or deodorants come in contact with your lenses.
- Avoid using aerosol products such as hair spray while wearing your lenses. If a spray is used, keep your eyes closed until the spray has settled.
- Fluorescein should not be used while the lens is on the eye.
- Always follow the lens care system recommended for your **Permalens®** Hydrophilic Contact Lenses. (See Directions).
- Do not mix or alternate heat (thermal) and chemical (not heat) lens care systems.
- Use **only** the recommended lens care solutions. Do not use saliva or anything other than the approved solutions to wet (hydrate) the lenses. (See Directions).
- Avoid all harmful or irritating vapors and fumes while wearing your lenses.
- Do not swim with your lenses in place.
- Never use tweezers or other implements to remove the lens from the lens container. Pour the lens into your hand.
- Do not touch the lens with your fingernails.

ADVERSE EFFECTS (Possible problems)

The following adverse effects have been reported with the use of soft contact lenses, including **Permalens®** Hydrophilic Contact Lenses:

- Discomfort, or feeling of something in the eye (a foreign body, or an abrasion, or a scraped area)
- Eye infection
- Stinging, burning, itching (irritation)
- Excessive watering of the eye (tearing)
- Redness of the eyes
- Dry eyes
- Reduced sharpness of vision (poor visual acuity)
- Blurred vision
- Sensitivity to light (photophobia)

What to do if a problem occurs:

- First remove the lens.
- If the discomfort or problem stops, then look closely at the lens.
- If the lens is chipped, cracked, or otherwise damaged, **DO NOT** put the lens back on your eye. Return the lens to the storage case. Then contact your eye care practitioner.
- If the lens has dirt, an eyelash, or other foreign body on it, thoroughly clean and disinfect the lens using the method recommended by your eye care practitioner. (See Directions)
- If the problem does not stop, or if it recurs when you replace the lens, contact your eye care practitioner immediately.

DIRECTIONS (How to care for your lenses)

General Instructions

- **BOTH CLEAN AND DISINFECT THE LENSES EACH TIME THEY ARE REMOVED FROM YOUR EYES.**
- First wash and rinse your hands thoroughly.
- Use only the recommended system of lens care, either heat (thermal) or chemical (not heat).
- **DO NOT ALTERNATE OR MIX LENS CARE SYSTEMS.**
- Use the right solutions (see chart of approved solutions).
- After disinfection leave the lenses in the unopened storage case until ready to put the lenses on your eyes.
- Empty all of the solution from the storage case as soon as you remove your lenses.

CLEANING

- Place 3 drops of PREFLEX® Sterile Cleaning Solution on each surface of the lens and thoroughly clean between thumb and forefinger for at least 20 seconds to remove mucus and other deposits.
- Rinse both surfaces thoroughly for at least 10 seconds with a steady stream of PERMASOL™ Rinsing and Storage Solution, UNISOL® Preservative-Free Saline Solution, or FLEX-CARE® Sterile Solution for Rinsing, Storing and Disinfecting, depending upon the disinfection method used. (See Approved Solutions Chart.)

DISINFECTING

Heat (thermal) Lens Care System

- First clean and rinse the lenses. (See Cleaning).
- Place each lens in the correct section (Right or Left) of the storage case or vial and fill with the recommended rinsing solution; be sure the lens is completely covered.
- Close the storage case tightly.
- Place the storage case in the recommended low-heat disinfection unit.
- Disinfect according to the directions for operation of the low-heat disinfection unit.
- After disinfection, store the lenses in the **unopened** storage case until ready to place on the eyes.
- Empty the storage case after removing the lenses and allow the case to air dry.

EMERGENCY (ALTERNATE) METHOD FOR HEAT (THERMAL) LENS CARE SYSTEM

- If a low-heat disinfection unit is not available, place the storage case containing the lenses in a pan of already boiling water for 10 minutes (at altitudes above 7000 feet boil for 15 minutes).
- Remove the pan from the heat and allow to cool for 30 minutes to complete disinfection.
- USE OF THE RECOMMENDED LOW-HEAT DISINFECTION UNIT MUST BE RESUMED AS SOON AS POSSIBLE.

Chemical (not heat) Lens Care System

- First clean and rinse the lenses. (See Cleaning)
- Place each lens in the correct chamber (Right or Left) of your empty storage case.
- Fill the storage case with fresh FLEX-CARE® disinfection solution, completely covering the lens.
- Close the storage case tightly.
- NEVER HEAT THE PERMALENS® IN THE DISINFECTING SOLUTION.
- Leave the lenses in the solution for at least 4 hours.
- Remove each lens separately.
- Rinse thoroughly with FLEX-CARE® before putting the lens on the eye.

Lens Care Solutions

A complete list of solutions approved for use with the **Permalens®** (perfilcon A) Hydrophilic Contact Lens is given in the following table.

APPROVED SOLUTIONS FOR USE WITH PERMALENS® (PERFILCON A) HYDROPHILIC CONTACT LENS

SYSTEM PROCESS	HEAT (THERMAL) DISINFECTION SYSTEM	CHEMICAL (NOT HEAT) DISINFECTION SYSTEM
CLEANING	PREFLEX®	PREFLEX®
RINSING	PERMASOL™ UNISOL®	*FLEX-CARE®
DISINFECTING	PERMASOL™ used with approved low-heat disinfection unit. UNISOL® used with approved low-heat disinfection unit	*FLEX-CARE®
LUBRICATING	CLĒRZ® ADAPETTES®	CLĒRZ® ADAPETTES®

*Chemical solution which disinfects. NOT TO BE USED WITH HEAT. The use of FLEX-CARE® with heat has a harmful effect on the lenses.

Product List:

ADAPETTES®	Sterile Lubricating Solution (Burton, Parsons & Co., Inc.)
CLĒRZ®	Lubricating and Rewetting Eye Drops (CooperVision, Inc.)
FLEX-CARE®	Rinsing, Storing and Disinfecting Solution (Burton, Parsons & Co., Inc.)
PERMA-CASE™	Contact Lens Storage and Carrying Case (CooperVision, Inc.)
PERMASOL™	Rinsing and Storage Solution (CooperVision, Inc.)
PREFLEX®	Sterile Cleaning Solution (Burton, Parsons & Co., Inc.)
UNISOL®	Preservative-Free Saline Solution, for rinsing and storing in the heat system (CooperVision, Inc.)

How to care for a dried out (dehydrated) lens

- Always keep the lens completely covered in the recommended storage solution when the lens is not being worn, to prevent the lens drying out.
- If a **Permalens®** Hydrophilic Contact Lens is left exposed to the air for 30 minutes or longer it will become dry and brittle.

To rewet your lens

- Handle a dried-out lens with care.
- First, place the lens in its storage case, and soak for at least one hour in one of the recommended rinsing and storage solutions: PERMASOL™ or UNISOL®, when using the heat (thermal) system, FLEX-CARE® when using the chemical (not heat) system.
- Next, clean and disinfect the rewetted (rehydrated) lens, using the lens care system recommended by your eye care practitioner. (See Directions)

DOSAGE AND ADMINISTRATION

Fitting Information

In general, the eye care practitioner will use conventional methods to fit the **Permalens®** (perfilcon A) Hydrophilic Contact Lens for vision correction. For a detailed description of the recommended fitting procedure refer to the **Permalens®** Fitting and Information Guide, available from CooperVision, Inc. (address below).

Dosage (Wearing schedule)

NOTE: Not every patient is able to wear the **Permalens®** Hydrophilic Contact Lens on an extended wear basis even if able to wear a lens on a daily wear basis. The eye care practitioner should determine the wearing schedule. Regular checkups, as determined by the eye care practitioner, are extremely important.

The eye care practitioner may recommend a limited wearing time for the first two days. However, clinical studies have established that a wearer's eyes adapt readily to wearing the **Permalens®** Hydrophilic Contact Lens on an extended wear basis, and that limited initial wear is usually not necessary.

The lens should be removed for cleaning and disinfection at least once every two weeks (14 days).

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a sterile buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expiration date of the lens.

CAUTION: FEDERAL LAW PROHIBITS DISPENSING WITHOUT PRESCRIPTION

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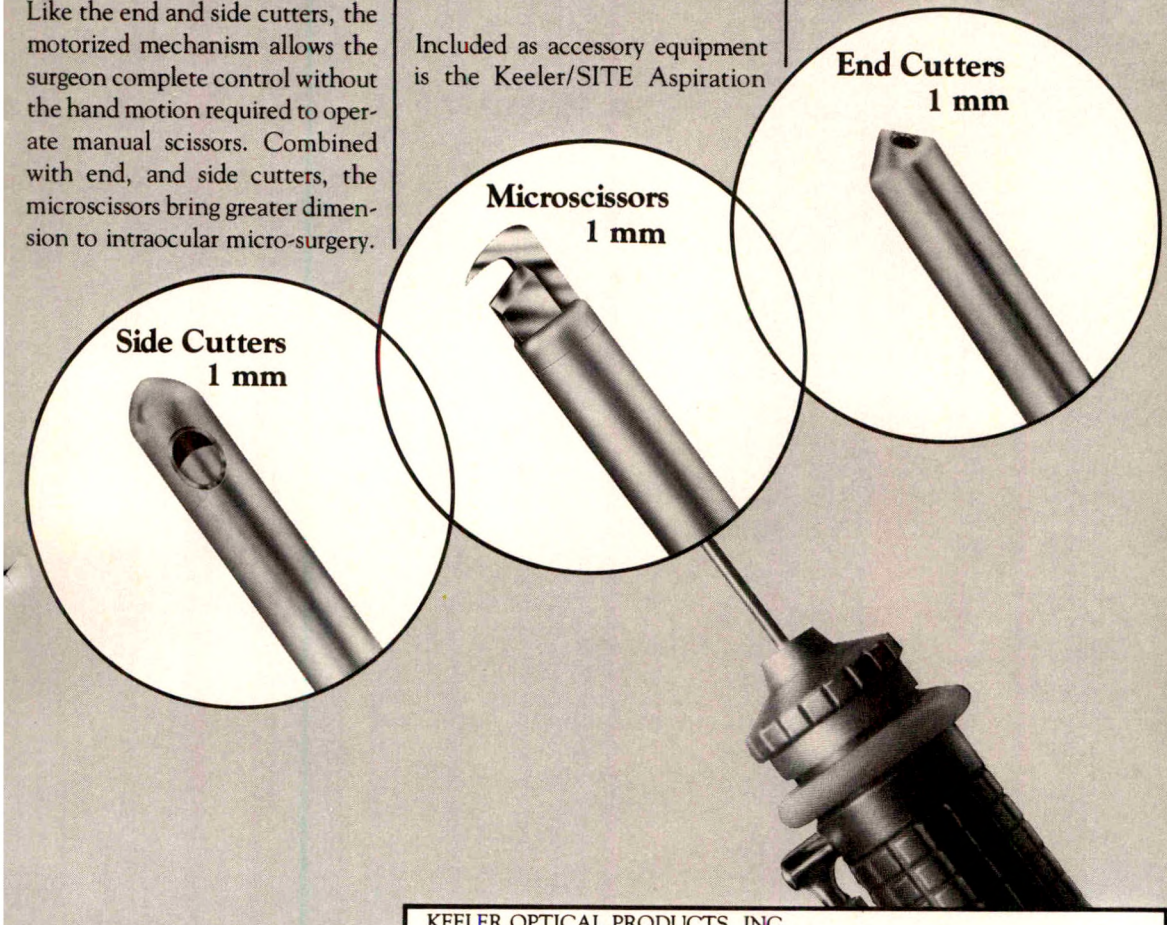
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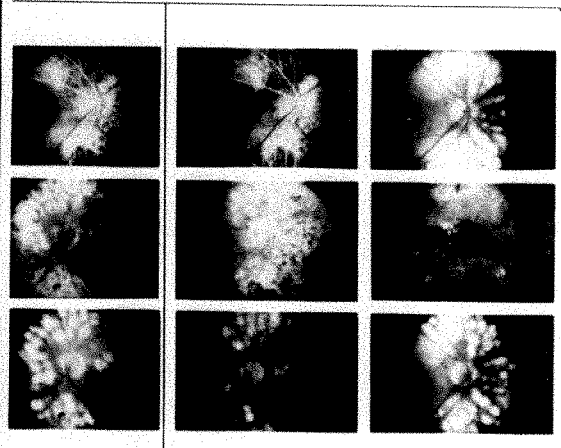
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The Eye of Horus

The origin of today's prescription symbol "Rx" goes back 5,000 years to the picture of the eye of the Egyptian god of healing—Horus.

DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′-deoxyuridine.

VIROPTIC sterile opththalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

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(Trifluridine)

OPHTHALMIC SOLUTION, 1%

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95% of Patients Responded*

During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

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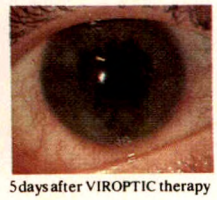
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5 to 9 Drops Daily

One drop every 2 hours while awake until herpetic lesion has completely re-epithelialized, then one drop every 4 hours for 7 days post-re-epithelialization.



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5 days after VIROPTIC therapy

Effective in 138 of 150 patients* Unresponsive or Intolerant to Idoxuridine or Vidarabine

In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

*Data on file, Medical Division, Burroughs Wellcome Co.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

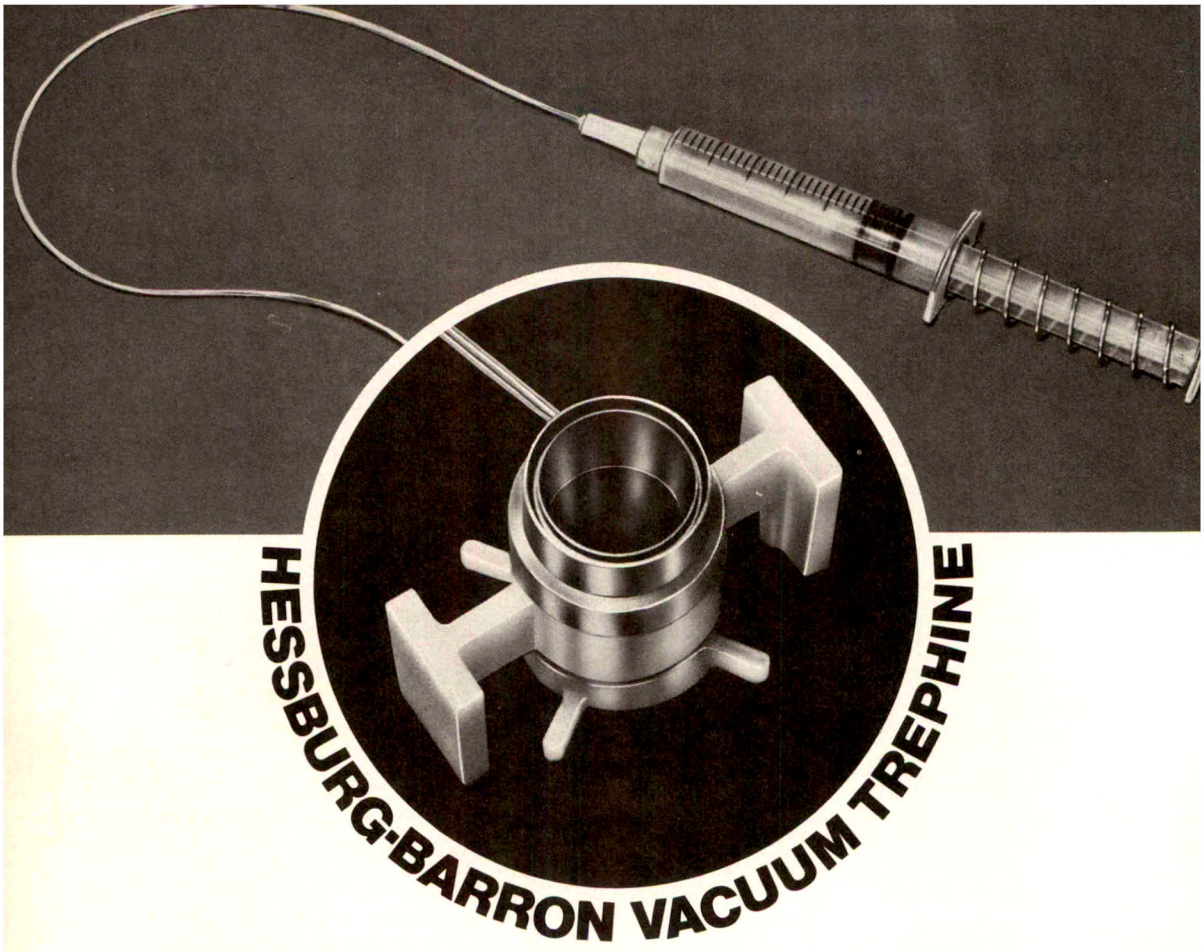
Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml. Store under refrigeration 2° to 8° C (36° to 46° F).

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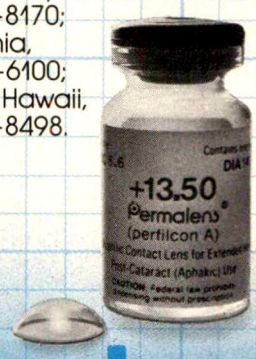
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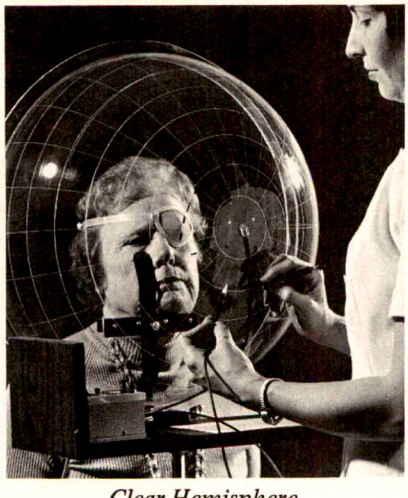
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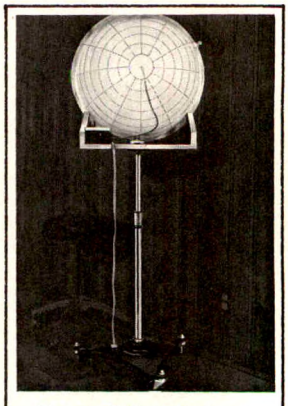
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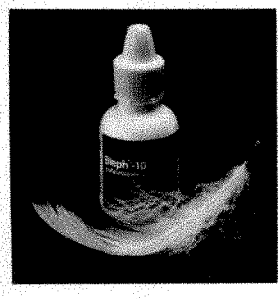
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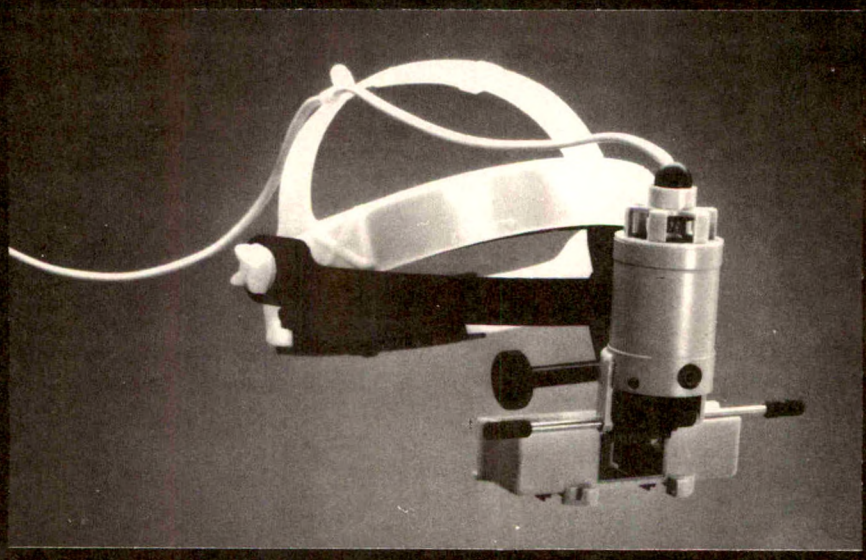


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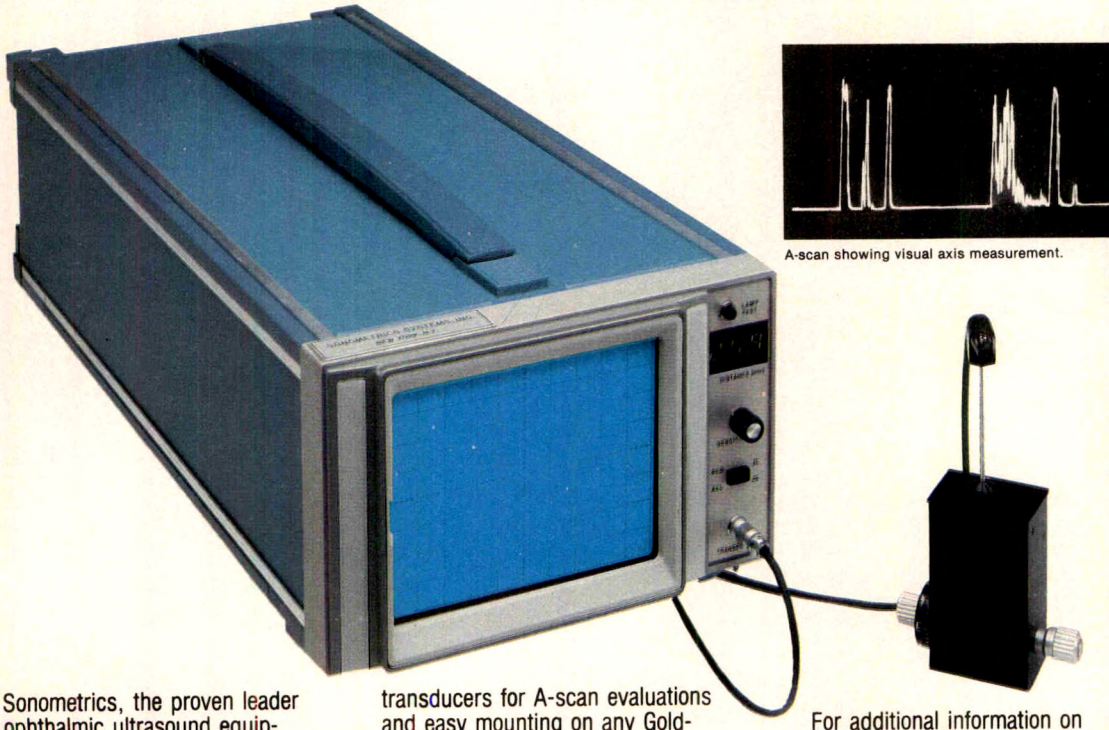
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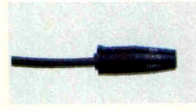
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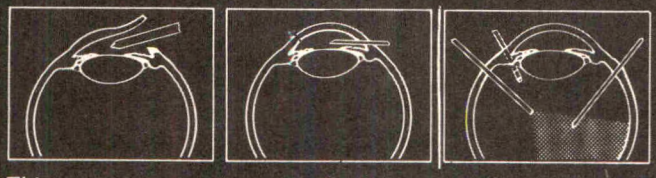
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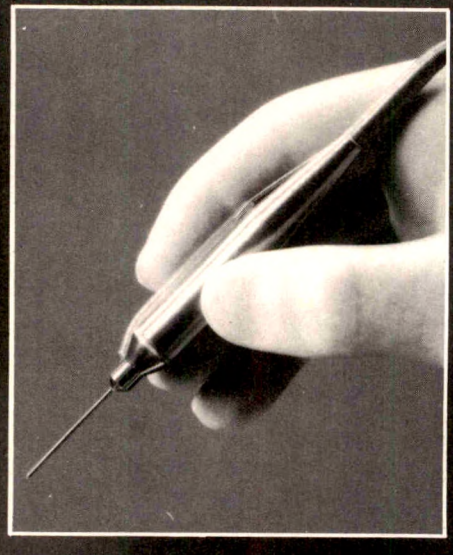
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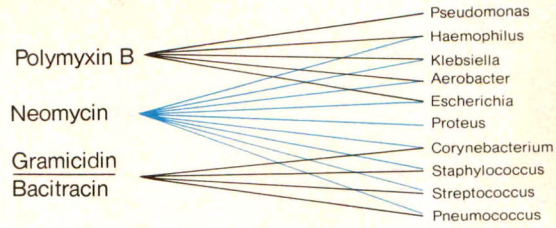
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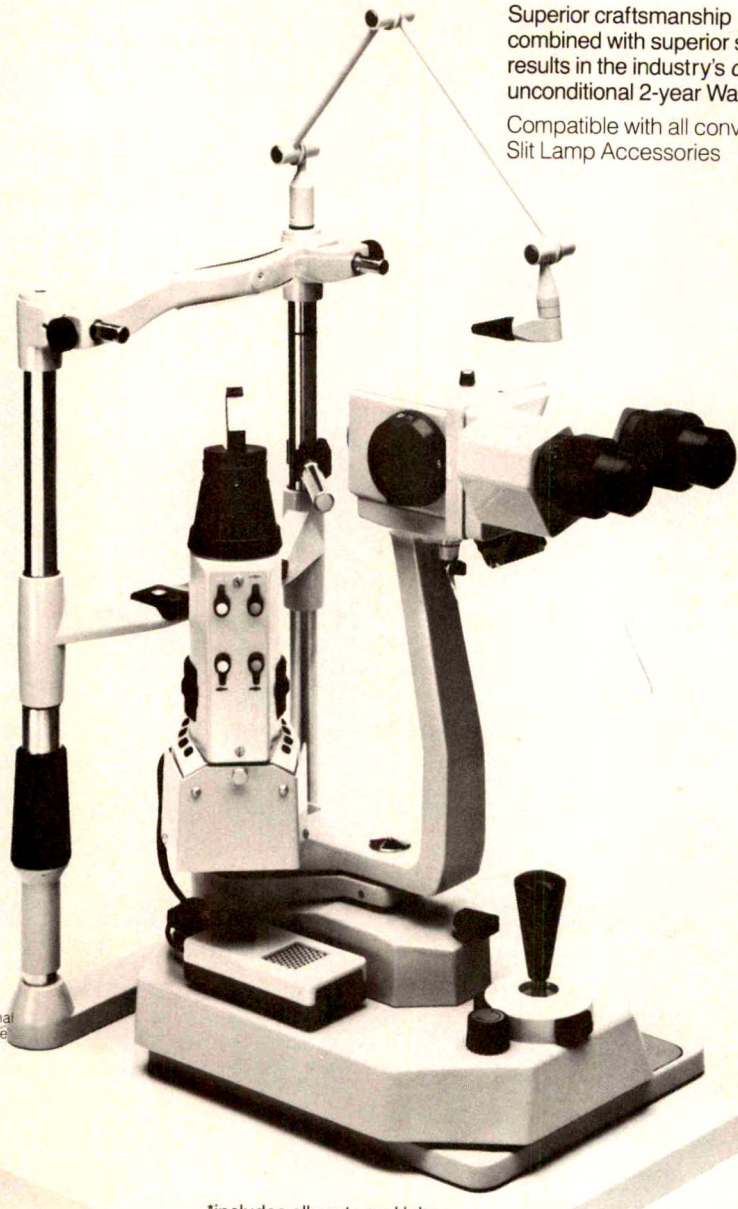
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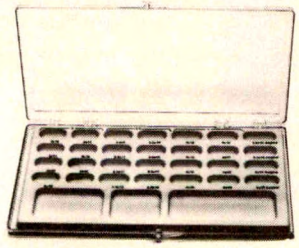
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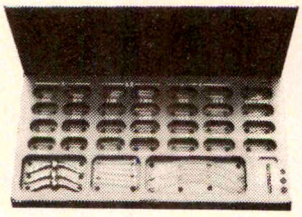
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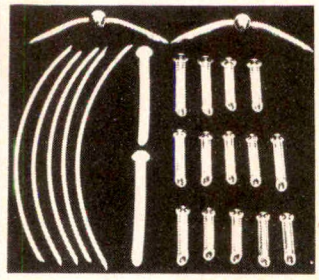
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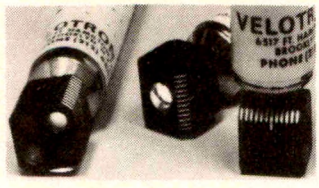
* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
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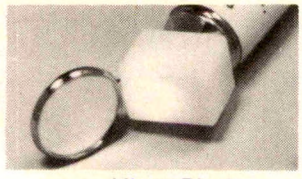
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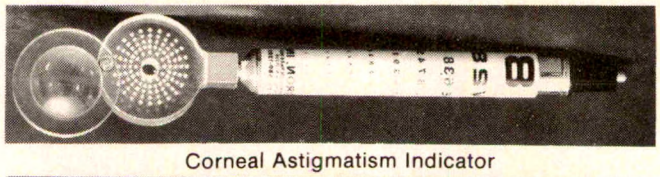
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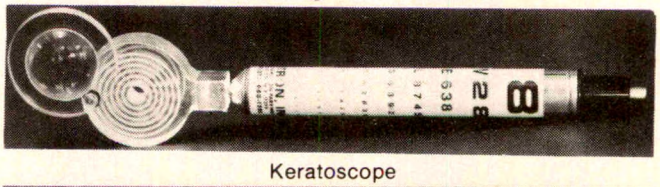
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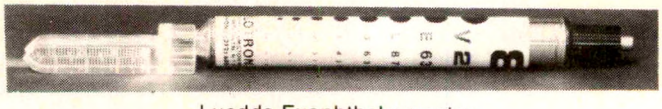
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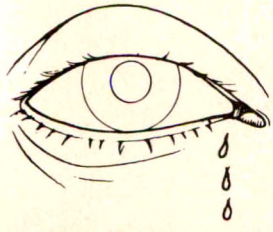
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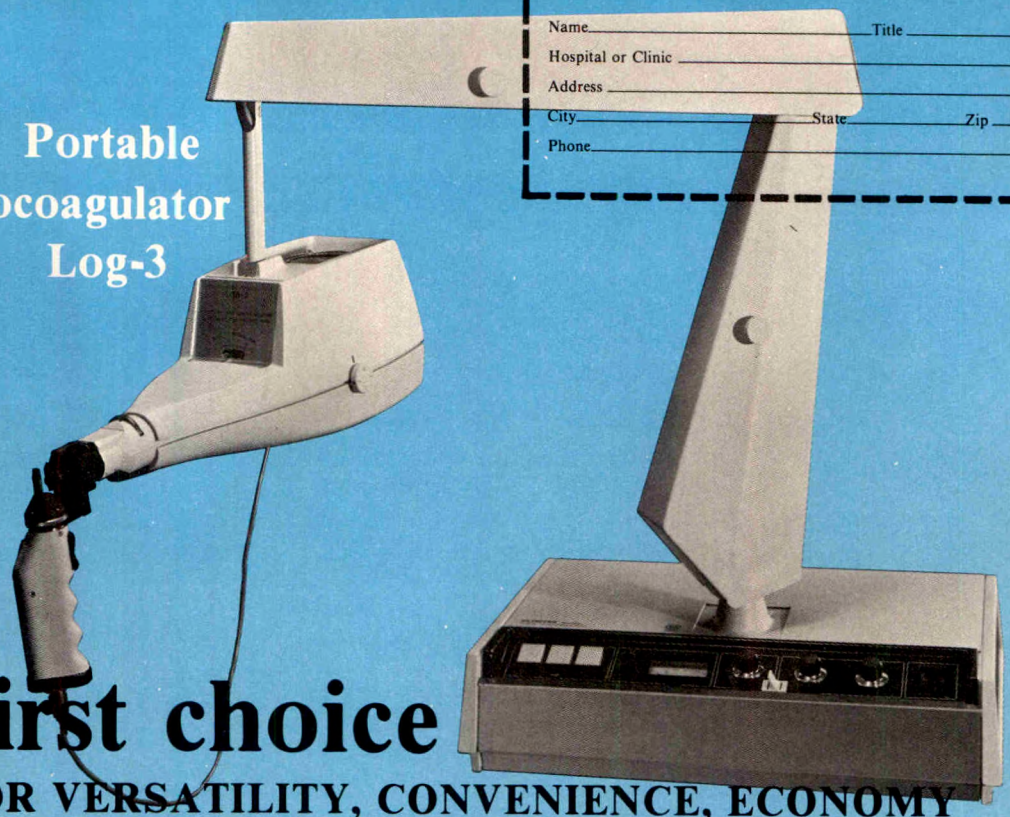
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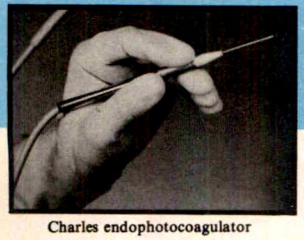
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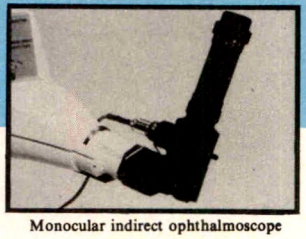
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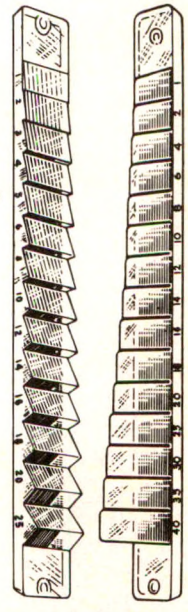
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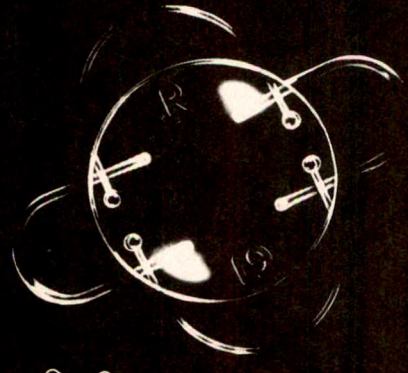
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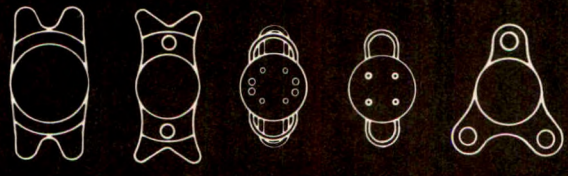
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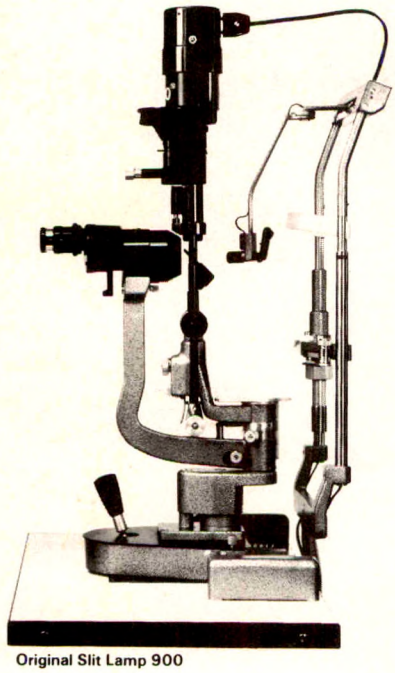
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*INDICATIONS: Based on a review of these drugs by the National Academy of Sciences—National Research Council and/or other information, FDA has classified the indications as follows:

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CONTRAINDICATIONS: These products are contraindicated in acute purulent conjunctivitis and blepharitis; tuberculous, fungal or viral lesions of the skin or eye, including herpes simplex, dendritic keratitis, vaccinia or varicella; and conditions involving the posterior segment of the eye. They are also contraindicated in those individuals who have shown hypersensitivity to any of their components.

PRECAUTIONS: Extended ophthalmic use of topical steroid therapy may result in glaucoma with possible damage of the optic nerves, posterior subcapsular cataract formation, or may aid in the establishment of secondary ocular infections from fungi or viruses liberated from ocular tissues. It is advisable that intraocular pressure be checked frequently. In those diseases causing thinning of the cornea, perforation has been known to occur with the use of topical steroids. As with any antibiotic preparation, prolonged use may result in the overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

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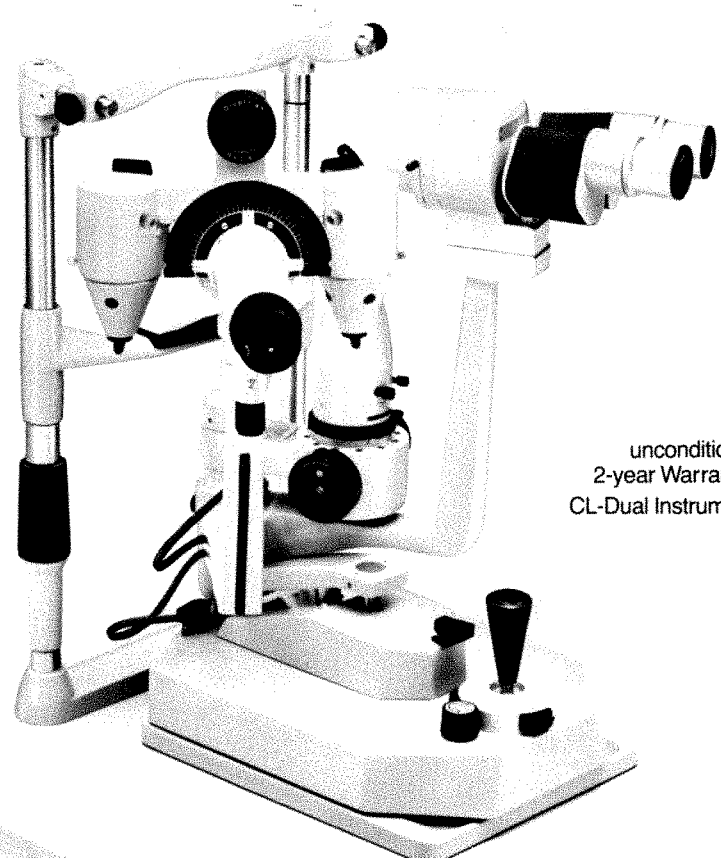
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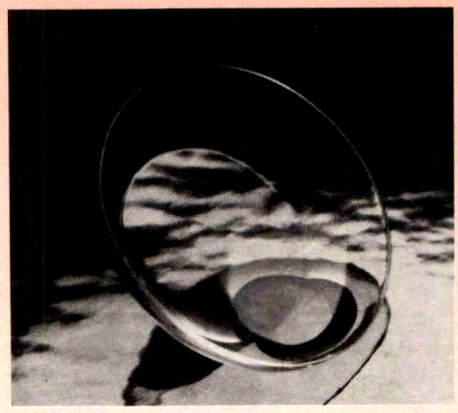
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1. Fatt, I. and Morris, J., "A Survey of Gas-Permeable Contact Lenses." The Optician, November, 1977.

2. Clinical data on file at Heyer-Schulte Medical Optics Center.

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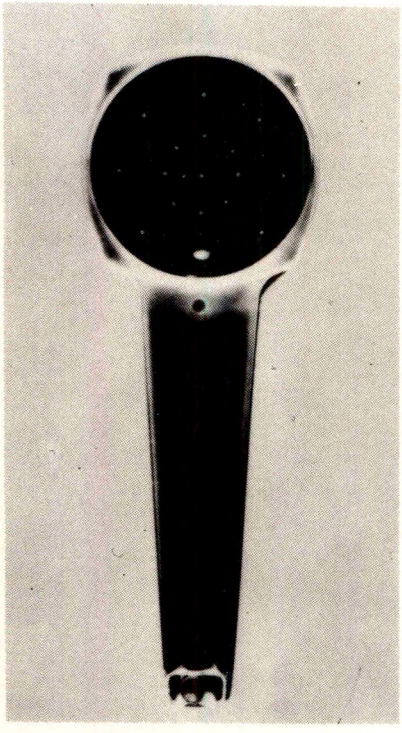
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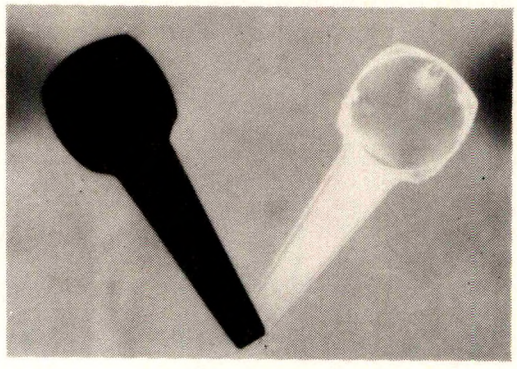
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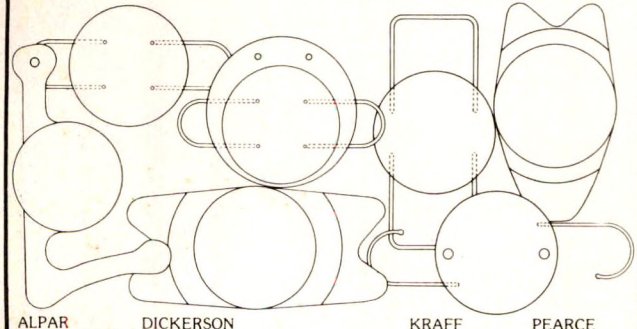
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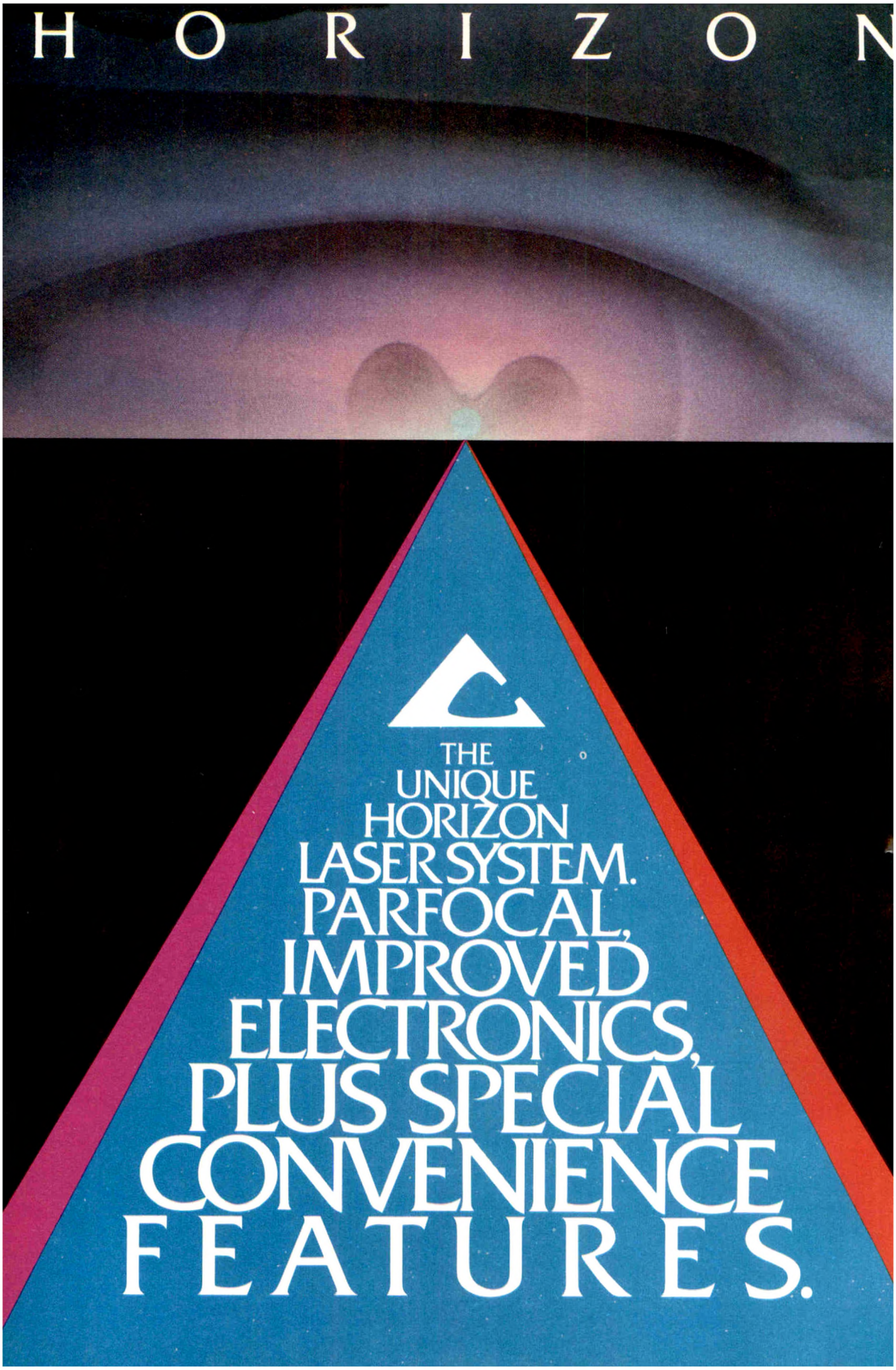
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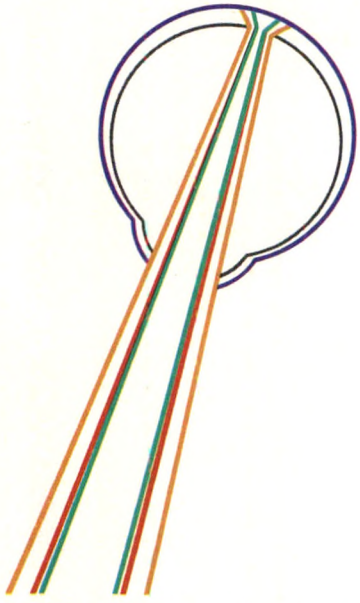
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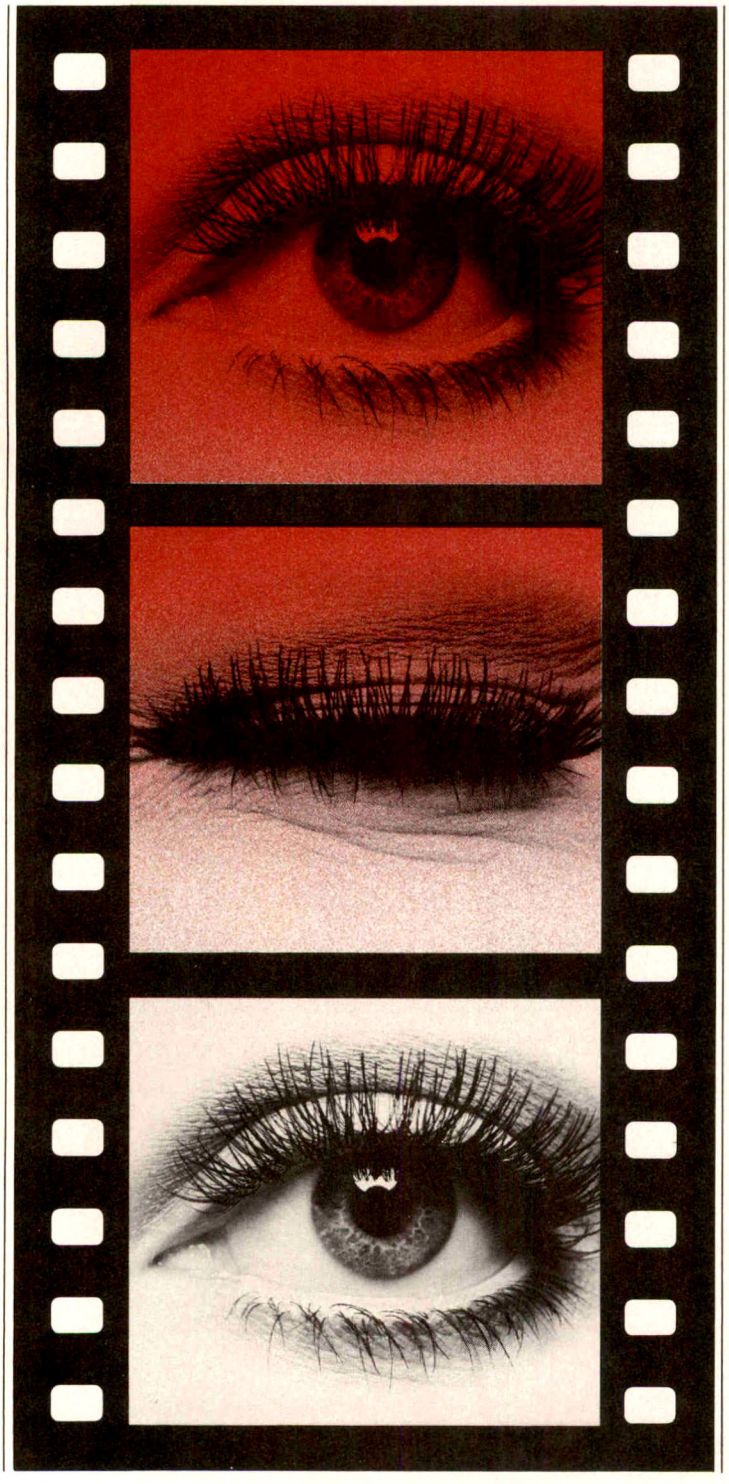
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VOLUME 91 NUMBER 3 MARCH, 1981

MORPHOLOGIC, FLUORESCEIN ANGIOGRAPHIC, AND LIGHT MICROSCOPIC FEATURES OF EXPERIMENTAL CHOROIDAL NEOVASCULARIZATION

D. B. ARCHER, F.R.C.S., AND T. A. GARDINER, F.R.C.S.

Belfast, Northern Ireland

We induced choroidal neovascularization in the rhesus monkey by impoverishing the blood supply to the inner retina and producing defects in Bruch's membrane by photocoagulation. Fourteen of 46 eyes undergoing photocoagulation developed neovascular fronds which were identified and categorized by histopathologic examination and fluorescein angiography. All new vessels gained access to the retina through defects in Bruch's membrane at the site of photocoagulation marks.

In eight eyes the new vessels remained localized to the immediate vicinity of photocoagulation marks. In four eyes neovascular fronds infiltrated the subretinal space for distances up to 6 disk diameters from the point of entry into the retina. In two eyes choroidovitreal neovascular complexes developed but rapidly regressed shortly after gaining the vitreous cavity. Fluorescein angiography demonstrated that all neovascular fronds were grossly incompetent to dye but that formed feeding channels had some degree of integrity. Light microscopic studies showed the proliferating networks to be composed of capillaries with well-formed basement membranes and more mature vessels with the basic structure of choroidal arteries and veins.

The penetration of Bruch's membrane by newly formed choroidal vessels is a common occurrence in aging eyes. Such neovascular complexes extend beneath the retinal pigment epithelium or neuroretina, but most provoke little reaction, remain clinically hidden, and are detected only on routine histopathologic examination.[1,2] Less commonly, infiltrating neovascular networks stimulate the proliferation of fibrous tissue and lead to the accumulation of fluid and blood beneath or within the outer retina.[3,4] This reaction, which commonly occurs at the macula, is known as the disciform re-

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sponse and has serious consequences for central vision. Recent surveys of severe visual impairment have shown that choroidal neovascularization and its sequelae are now major causes of blindness in elderly patients in scientifically developed countries.[5,6]

Choroidal neovascularization may also complicate certain conditions that damage Bruch's membrane and alter the outer retina. Among these are severe myopia, angioid streaks, multifocal inner (presumed histoplasmin) choroiditis, traumatic ruptures of the choroid, and cases where fluid chronically accumulates beneath the retinal pigment epithelium or sensory retina. Choroidal neovascular networks should be suspected when a disciform response occurs. They can be defined accurately by fluorescein angiography, which also demonstrates their characteristic permeability to dye.[3,4,7] Nevertheless, our understanding of the histopathologic process of choroidal neovascularization has been limited by the scarcity of preserved human tissue suitable for detailed analysis. Additionally, no appropriate and reproducible animal model for choroidal neovascularization has been available to help explain the basic pathophysiology of the early disease process.

We induced choroidal neovascularization in the rhesus monkey, examined the fine structure of new choroidal vessels, and developed a hypothesis as to their likely mode of growth.

Material and methods

We used 28 rhesus monkeys, both male and female, aged between 6 months and 2 years and weighing between 2 and 6 kg each, for the investigation. We anesthetized the animals with intravenous sodium pentobarbitol, 10 mg/kg of body weight, and intermittently added sodium pentobarbitol as needed during the course of the experiment.

The pupils were widely dilated with 1% cyclopentolate and 10% phenylephrine. We occluded many branch retinal veins of different sizes (usually second or third order) by photocoagulation. We used small, target-sized, high-intensity burns to occlude selected veins. We initially arrested blood flow with a single, high-intensity burn straddling the selected vein, and later directed one or more supplementary burns to the partially collapsed adjacent segment of the vessel downstream. Vascular patency occasionally reappeared after this treatment and rephotocoagulation was undertaken at a later date. Vitreous hemorrhage occurred at the time of photocoagulation in about one third of the experiments. In these instances photocoagulation was repeated one to two months later when the vitreous hemorrhage had either absorbed or cleared sufficiently to permit good visualization of the fundus.

In 11 monkeys, fellow arteries were also photocoagulated to produce more extensive areas of inner retinal ischemia. This was technically more difficult to accomplish and in most instances we achieved only partial obstruction. The various combinations of vascular obstruction and occlusion are shown in the Table. For control purposes, we placed intense photocoagulation marks around the posterior poles of five monkeys, taking care to avoid major retinal blood vessels.

We observed the monkeys for periods of two weeks to nine months and took serial color photographs and fluorescein angiograms while they were under general anesthesia. We used a fundus camera equipped with interference filters for angiography and injected 0.1 and 0.5 ml of sodium fluorescein via a calf vein. Color photography and fluorescein angiography were carried out in each animal before it was killed.

At the termination of each experiment, we enucleated both eyes, opened them

TABLE

RESULTS OF EXPERIMENTAL CHOROIDAL NEOVASCULARIZATION

Retinal Vessels Photocoagulated	No. of Eyes	Eyes With New Vessels	Eyes With Vitreous Hemorrhage
Superior and inferior temporal veins	28	6	9
Superior and inferior temporal veins; superior temporal artery	7	3	2
Superior and inferior temporal veins; inferior temporal artery	3	1	2
Superior and inferior temporal and inferior nasal veins	1	—	—
Superior and inferior nasal veins	3	—	1
Superior and inferior nasal and inferior temporal veins	1	1	1
Superior and inferior nasal veins; inferior nasal artery	1	—	—
Macular veins	2	2	1
Superior and inferior temporal veins and secondary burns to ischemic retina	2	1	2
Total	48	14	18

via a circumferential pars plana incision, and immediately immersed them in 3% glutaraldehyde in a 0.1 M phosphate buffer solution. After four hours the eyes were transferred to a wash of identical buffer containing a solution of 3% sucrose. We removed the anterior segments of the eyes 24 to 48 hours later and studied each fundus with a stereomicroscope. Areas identified as sites or possible sites of choroidal neovascularization were located and photographed. We used fluorescein angiograms to locate, orientate, and dissect appropriate blocks of tissue for light and electron microscopy. Tissue blocks were postfixed in 2% osmium tetroxide in 0.1 M phosphate buffer and embedded in Araldite for sectioning. We used 1-μ sections stained with toluidine blue for light microscopy.

RESULTS

Alterations in the retina after retinal vascular obstruction—The combined obstruction of a superotemporal and inferotemporal branch vein, with or without photocoagulation of a fellow artery, resulted in widespread temporal retinal venous stasis and ischemia within the distribution of the obstructed vessels. A few venovenous collateral channels formed at the borders of the obstructed venous circulations and diverted blood from obstructed to normal retinal vessels. Despite such channels, the temporal retinal circulation was markedly compromised, causing serous fluid to accumulate within and deep to the retina. Localized serous detachment of the retina was common at the posterior pole of the eye. Within two to three days, hemorrhage, often widespread, occurred within the inner retina, occasionally spreading beneath the internal limiting membrane and reaching the vitreous cavity. Subretinal tracking of blood also occurred occasionally. Focal and generalized zones of inner retinal ischemia were identified within a few days of the vascular occlusion and sheathing of affected major retinal vessels was apparent within one week.

Fluorescein angiography confirmed widespread closure of the retinal microvasculature within the distribution of the obstructed veins and incompetence of

most residual patent veins and venules. The untreated retinal vasculature remained intact except for participating in collateral formation.

By three to four weeks after occlusion, much of the retinal hemorrhage and edema had absorbed, and the residual affected vasculature had regained some degree of competence, although the vessel walls typically stained with fluorescein during angiography. Widespread structural abnormalities of the affected microvasculature were also outlined by fluorescein angiography, particularly microaneurysms, intraretinal neovascular buds, shunt-like vessels, and spike-like regressing precapillary arterioles which commonly bordered areas of nonperfused retina. Occluded and nonfunctioning major vessels appeared as distinct white lines. Striking alterations, principally related to the retinal pigment epithelium, occurred at the outer retina. We found foci of retinal pigment epithelial atrophy, proliferation, and mound formation. Characteristic cicatricial changes, including wrinkling, folds, and detachment, also occurred at the level of the internal limiting membrane. Dehiscences commonly occurred in the ischemic inner retina and frank retinal detachments occurred on two occasions. Periretinal membrane formation was common and typically associated with retinal pigment epithelial cell proliferation and migration into the preretinal space. Vitreous degenerative changes included syneresis, the formation of organized bands, and alterations associated with long-standing vitreous hemorrhage.

The reopening or recanalization of one of the major temporal veins after photocoagulation provoked the development of extensive venovenous collateral systems which communicated across the median raphe. Various degrees of retinal ischemia occurred within the distribution of the occluded vein, particularly toward the equatorial and peripheral retina. Photocoagulation of the superonasal and inferonasal branch retinal veins, with or without fellow arteries, produced acute fundus changes of retinal edema, hemorrhage, and ischemia similar to, if less widespread than, occlusion of two major temporal vessels. At sites of intense photocoagulation, occluded veins and occasional arteries established communication with the choroidal circulation, maintaining the circulatory competence of the area affected (Fig. 1). The occlusion of macular veins precipitated macular edema, inner retinal cystoid degenerative changes, and intraretinal hemorrhage.

At photocoagulation sites there was full-thickness destruction of specialized retinal elements, disruption of Bruch's membrane, and atrophy of the underlying choriocapillaris that continued for a short distance beyond the photocoagulation burn. Fluorescein angiography and histopathologic evaluation of representative areas indicated that the choroidal circulation elsewhere was well perfused, although, less commonly, many contiguous photocoagulation burns placed along the course of a retinal vein or artery compromised the local choroidal circulation to some degree, as shown by slow or nonfilling of vessels on fluorescein angiography.

Choroidal vasoproliferation—Fourteen eyes demonstrated evidence of choroidal vasoproliferation at and beyond sites of photocoagulation. Occasionally the neovascular networks could be observed by ophthalmoscopy or biomicroscopy, but they were best identified by their gross incompetence to dye during fluorescein angiography. Fluorescein angiography also demonstrated that some neovascular complexes filled synchronously with the choroidal circulation and before retinal vascular perfusion had occurred. Serial sections traced well-developed neovas-

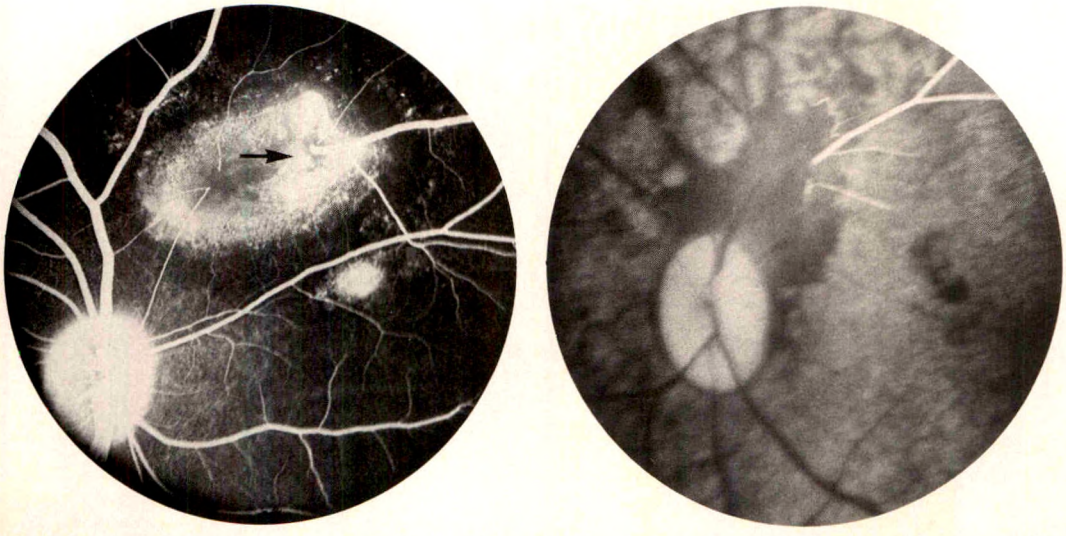


Fig. 1 (Archer and Gardiner). Left, Venous-phase fluorescein angiogram of the superonasal area of the fundus of a left eye after occlusion of the major superonasal retinal vein. A major anastomosis has developed between the distal portion of this vein and the choroidal circulation (arrow). Right, Early-phase fluorescein angiogram of a left fundus. A major left superotemporal artery fills directly from the choroidal circulation, which has perfused in advance of the retinal vasculature.

cular channels from the proliferating neovascular fronds to the choroidal circulation, confirming their origin from the choroidal vasculature. Smaller, less well developed neovascular outgrowths in the immediate vicinity of the photocoagulation burns were more difficult to link directly with the choroidal circulation because the small and tortuous new vessels were often obscured in the profusion of fibroglial tissue that characteristically occurred at photocoagulation scars. Three patterns of choroidal vasoproliferation emerged between one and six months after photocoagulation.

1. The most common neovascular response was local infiltration of the outer retina at and near to the site of photocoagulation (eight eyes) (Fig. 2). Such vascular invasion of the outer retina occurred within two to four weeks of photocoagulation, but the new vessels rarely extended farther than 1 or 2 disk diameters from the margins of the photocoagulation burns. Two eyes in which macular veins had been occluded developed a choroidal neovascular response within the macular region (Fig. 3).

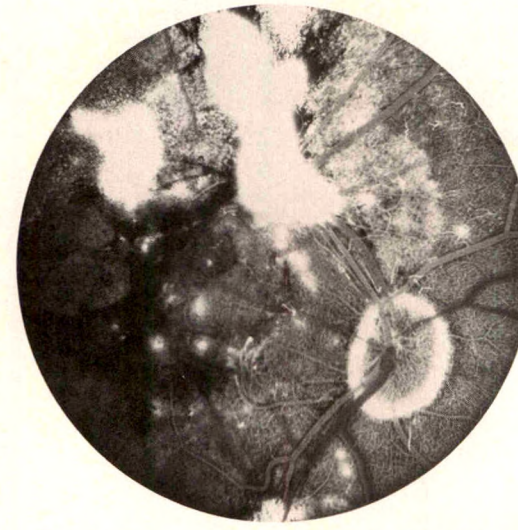


Fig. 2 (Archer and Gardiner). Late-phase fluorescein angiogram of the fundus of a right eye. New, strikingly fluorescent choroidal vessels have formed at the site of photocoagulation marks in the superotemporal fundus.

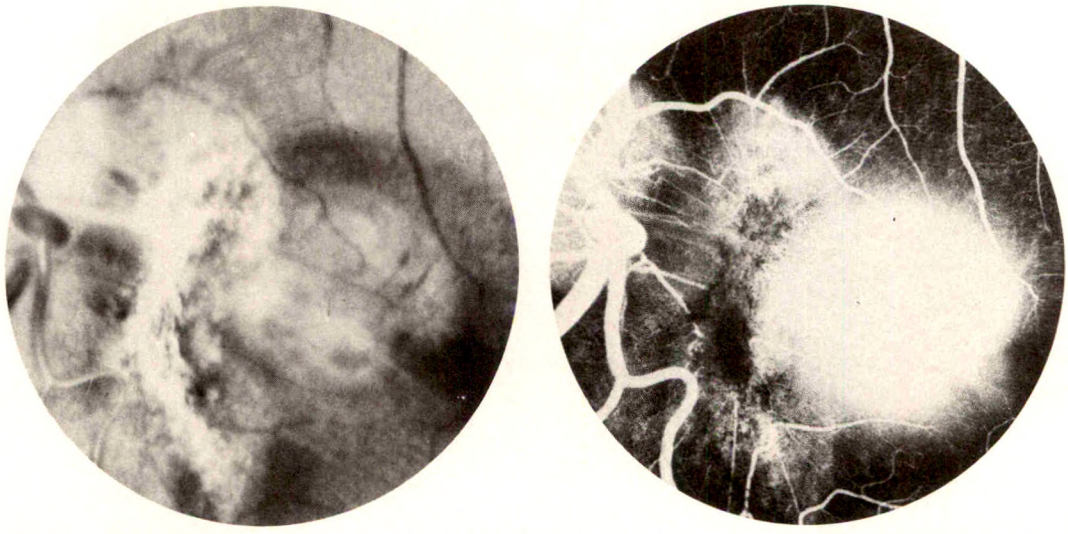


Fig. 3 (Archer and Gardiner). Left, Macular region of a left eye. Photocoagulation scars are present just temporal to the optic disk. A disciform-like lesion has developed temporal to the photocoagulation marks and involves the macular region. Right, Late-phase venous fluorescein angiogram of the same fundus. New choroidal vessels invading the subretinal space at the macula are strikingly incompetent to dye and fluoresce brightly.

Three eyes developed neovascular fronds that infiltrated the retina for some distance beyond the sites of photocoagulation. In two instances well-developed feeding channels evolved to service the neovascular fronds (Fig. 4).

2. Five eyes developed neovascular outgrowths from the choroid which traversed the necrotic retina at photocoagulation sites and gained the vitreous cavity. Such vasoproliferation typically occurred at sites of intense photocoagulation marks (Fig. 5) and where secondary photocoagulation burns had been placed in areas of ischemic inner retina (Fig. 6). Secondary burns characteristically produced operative choroidal hemorrhage with extravasation of blood into the preretinal region immediately anterior to the photocoagulation scars. In the event of two such photocoagulation marks being placed side by side, vasoformative tissues from each site established intervascular (intravitreal) links and anastomoses (Fig. 6). These profuse neovascular outgrowths typically regressed within three to four weeks, although a single well-developed channel connecting separate photocoagulation sites often remained suspended within the vitreous cavity. Fluorescein angiography confirmed that these neovascular outgrowths were grossly incompetent to dye; however, established channels maintained some integrity, although blood flow through such vessels was generally very retarded (Figs. 7 and 8).

Light microscopy—Histopathologic examination of vasoproliferative regions confirmed that most new vessels were located in the subretinal space (Fig. 9). Serial sections in some eyes showed continuity between the new vessels and the choroidal circulation through defects in Bruch's membrane. In other eyes such communications could not be confirmed as the neovascular architecture was obscured by scar tissue and proliferating

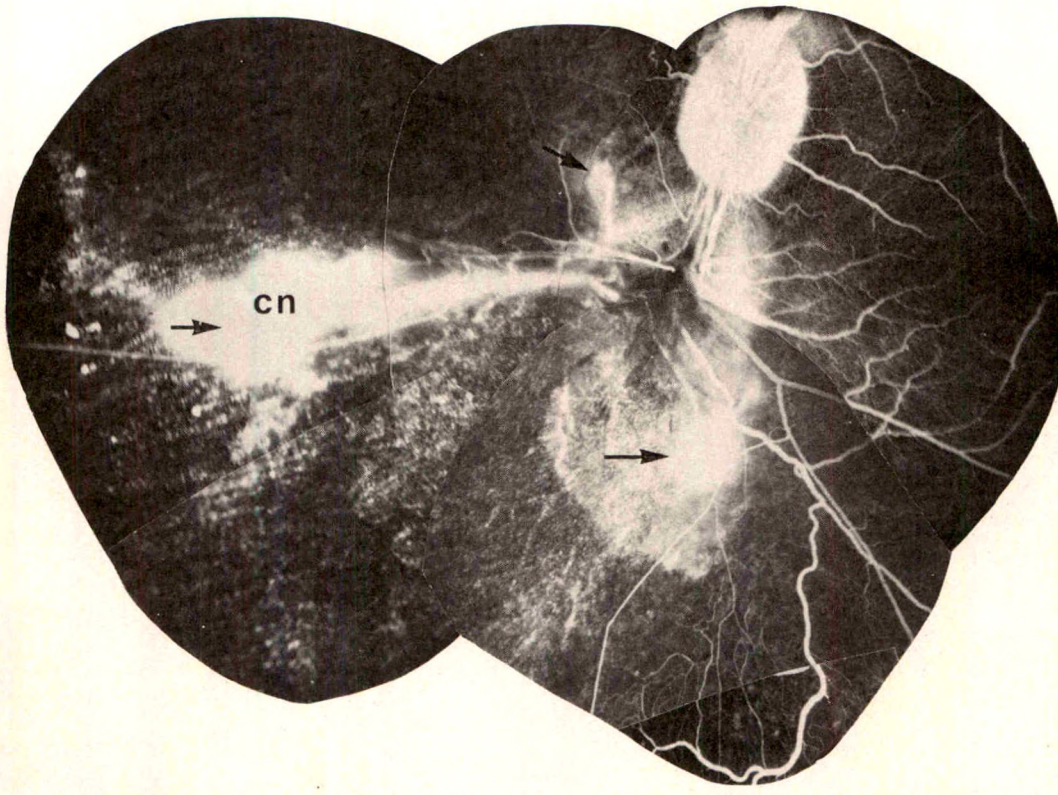


Fig. 4 (Archer and Gardiner). Composite fluorescein angiogram (late-phase) of the fundus of a left eye. Photocoagulation marks have been placed along the course of the inferonasal retinal vessels. Several fronds of subretinal neovascularization have formed at sites of photocoagulation (arrows). One network of subretinal neovascularization has extended some distance from the photocoagulation site towards the nasal equatorial retina (cn). Much of the nasal retina has lost its retinal circulation and occasional collateral branches link obstructed and unobstructed circulation. The neovascular networks are strikingly incompetent at their growing fronts.

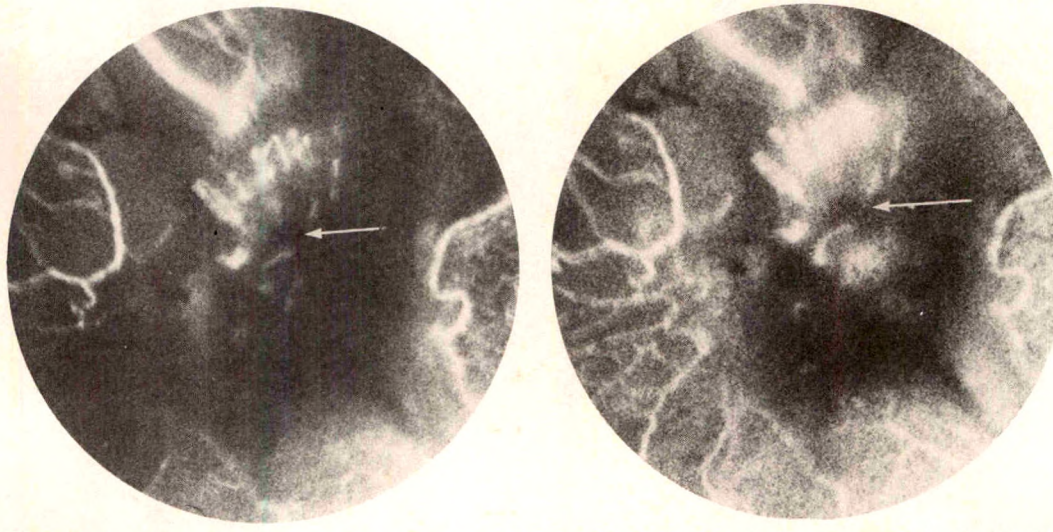


Fig. 5 (Archer and Gardiner). Venous angiograms show filling of choroidovitreal neovascular fronds that have developed at photocoagulation sites (arrows). The loop-like configuration of the new vessels can be identified in the early phases of angiography (left); late-phase angiogram (right) demonstrates their marked incompetence to dye.

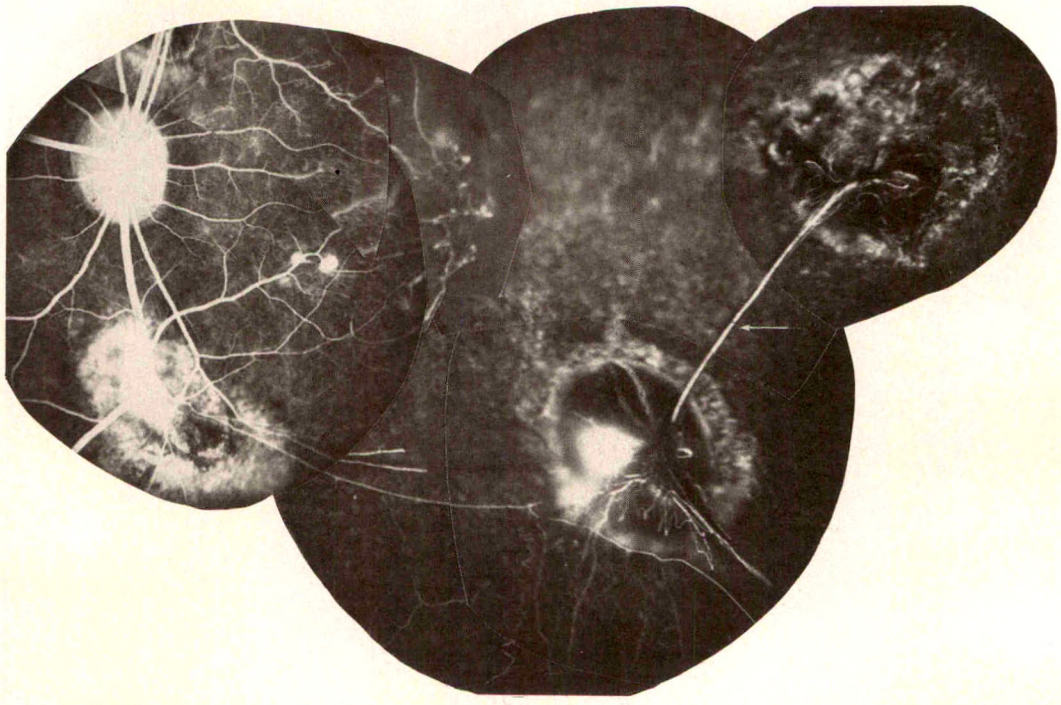


Fig. 6 (Archer and Gardiner). Fluorescein angiographic montage (late-phase) of the fundus of a left eye. Photocoagulation of superotemporal and inferotemporal vessels has resulted in widespread ischemia of the temporal inner retina. Choroidovitreal neovascularization has occurred at secondary photocoagulation marks placed within the ischemic temporal retina. A major intravitreal vascular trunk communicates between the two foci of vasoproliferative tissue (arrow).

retinal pigment epithelial cells at the photocoagulation sites. We found no communications between the vasoproliferative tissue and the retinal or papillary circulation, although, as noted previously, major retinal veins and arteries occasionally communicated with the choroidal circulation.

The retina overlying the neovascular complexes was largely ischemic and necrotic. The internal limiting membrane was typically wrinkled, often detached, and in some regions showed breaches through which Müller cells and astroglial processes had gained access to the preretinal space. Focal accumulations of degenerate red blood cells together with macrophages were frequently associated with the detached limiting membrane. Some macrophages contained partially sequestrated red blood cells, whereas others appeared to debride the internal surface of the detached membrane. Loss of the nervous elements in the inner retina often resulted in vacuolization and retinoschisis (Figs. 10 and 11), although the basic tissue skeleton was usually maintained through a compensatory overgrowth of the glial components. The gliotic tissue contained hypertrophied Müller cells, scattered fibrous astrocytes and, occasionally, in the interstices, lipid-laden macrophages. Embedded in the mosaic of glial processes were numerous remnants of the atrophic retinal vasculature. This material usually consisted of redundant basal lamina and degenerate erythrocytes. Where residual vascular basement tubes remained patent, they persisted as empty ghost vessels, although in many instances these too had been colonized or parasitized by the advancing glia.

The outer retina also showed a host of

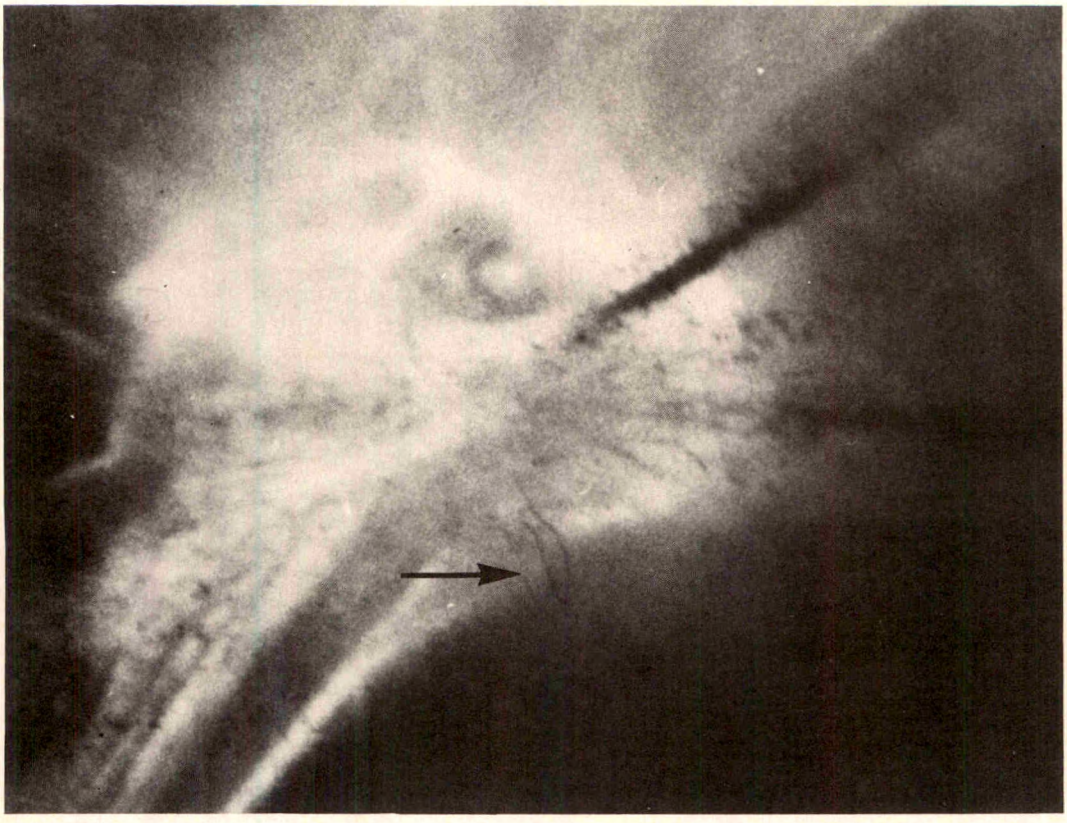


Fig. 7 (Archer and Gardiner). Superior secondary photocoagulation burn shown in Figure 6. Neovascular loops can be identified against the white background of fibroglial tissue (arrow). Linear accumulations of pigmented cells accompany the new vessels and extend into the vitreous.

degenerative changes near choroidal neovascular membranes. The retinal pigment epithelial cells in particular displayed a wide spectrum of alterations, including atrophy, depigmentation, and proliferation (Fig. 9). Proliferative transformations occurred either as reduplication of the existing epithelium or in the formation of subretinal membranes. These membranes were composed of cells that formed orderly palisades, having a basal lamina at one aspect and a microvillous border at another. Wandering phagocyte-like cells that appeared to bud from the pigment epithelium were common both at photocoagulation scars and throughout the adjacent retina. Less frequent additions to the cellular infiltrate of the neovascular lesions were unusual giant cells with ultrastructural features suggesting a pigment epithelial origin.

Bruch's membrane was discontinuous and disorganized at sites of photocoagulation associated with choroidal neovascularization, and small scattered dehiscences were found in the region of choroidal neovascular networks, particularly where advanced retinal pigment epithelial alterations had occurred. The retinal receptor layer was often degenerate or absent in the region of the neovascular complexes, although islets of deformed residual outer receptors persisted in the subretinal space. The choriocapillaris was obliterated at sites of photocoag-

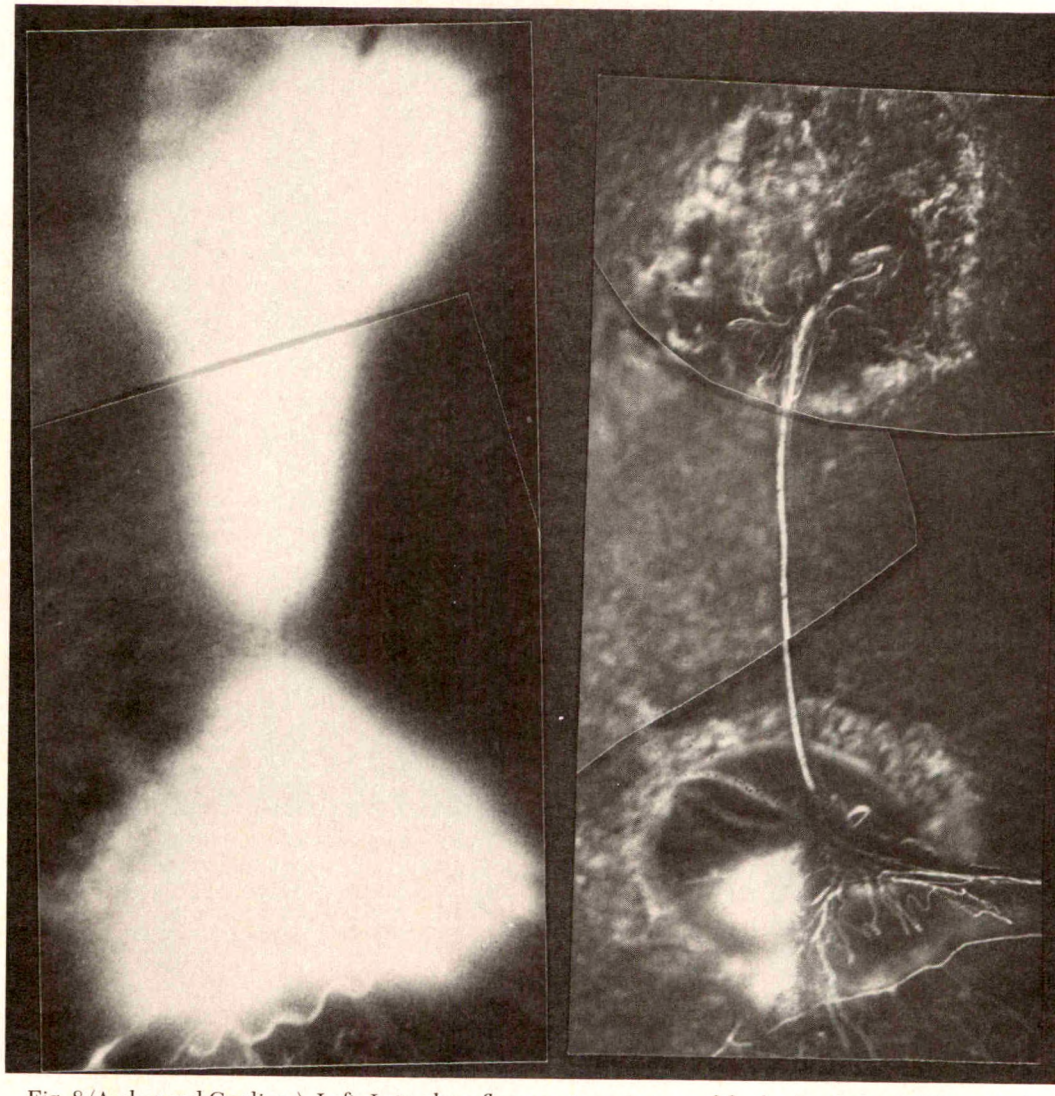


Fig. 8 (Archer and Gardiner). Left, Late-phase fluorescein angiogram of the fundus shown in Figure 6 taken three weeks after photocoagulation of ischemic retina. There is extensive intravitreal fluorescein leakage from incompetent choroidovitreal neovascular networks. Right, Late-phase fluorescein angiogram taken three weeks later shows regression of most choroidovitreous vessels at the superior photocoagulation site and significant resolution of neovascular fronds inferiorly. A residual major vessel links the photocoagulation sites and is competent to dye.

ulation but remained intact and perfused elsewhere.

The neovascular fronds contained conglomerations of irregularly convoluted capillaries enmeshed in a heterogeneous ground substance within the subretinal space (Fig. 8). The capillaries had endothelial and pericyte cell components and well-developed basement membranes. As neovascular fronds matured and infiltrated the outer retina, major vascular trunks occasionally developed to service the growing fronds. These vessels had the features of arteries and veins and were closely surrounded by dense fibrous tissue that included scattered fibroblasts

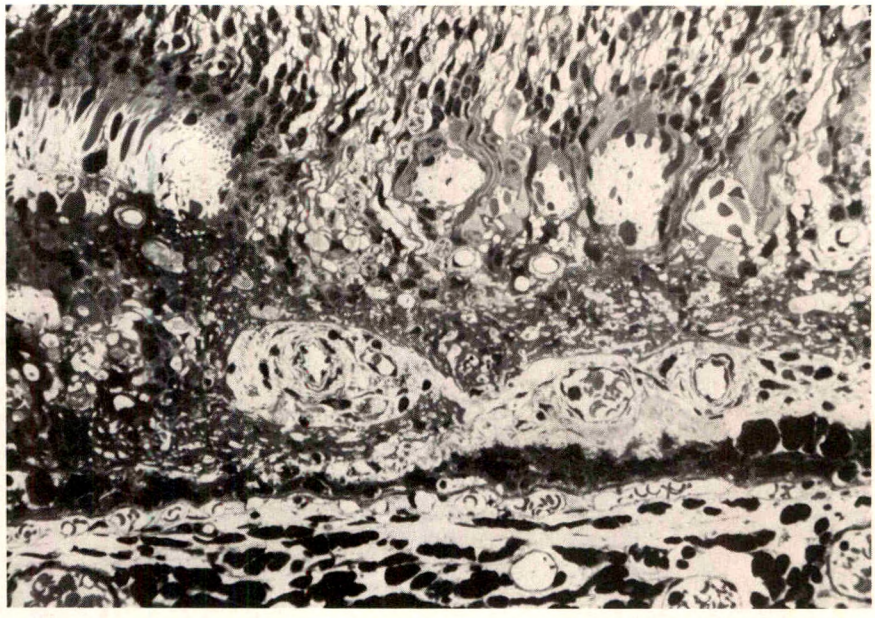


Fig. 9 (Archer and Gardiner). Subretinal neovasuclar frond. New vessels are present in the subretinal space. The overlying retinal receptors are atrophic and stunted and show a wide variety of degenerative changes. The inner retina is atrophic, gliotic, and largely avascular. The retinal pigment epithelium shows proliferation and reduplication. The choroidal vasculature remains intact (×300).

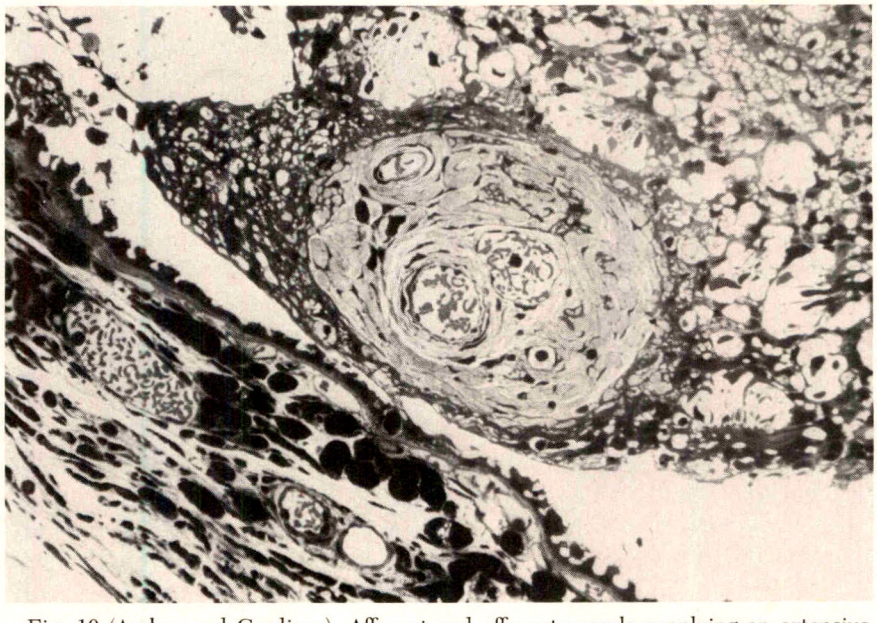


Fig. 10 (Archer and Gardiner). Afferent and efferent vessels supplying an extensive choroidal neovascular frond. The nutrient vessels are encapsulated in a fibrous sheath containing fibroblasts and large bundles of collagen. The vascular bundle lies in the subretinal space and is surrounded by Müller cells and residual dystrophic receptors (×300).

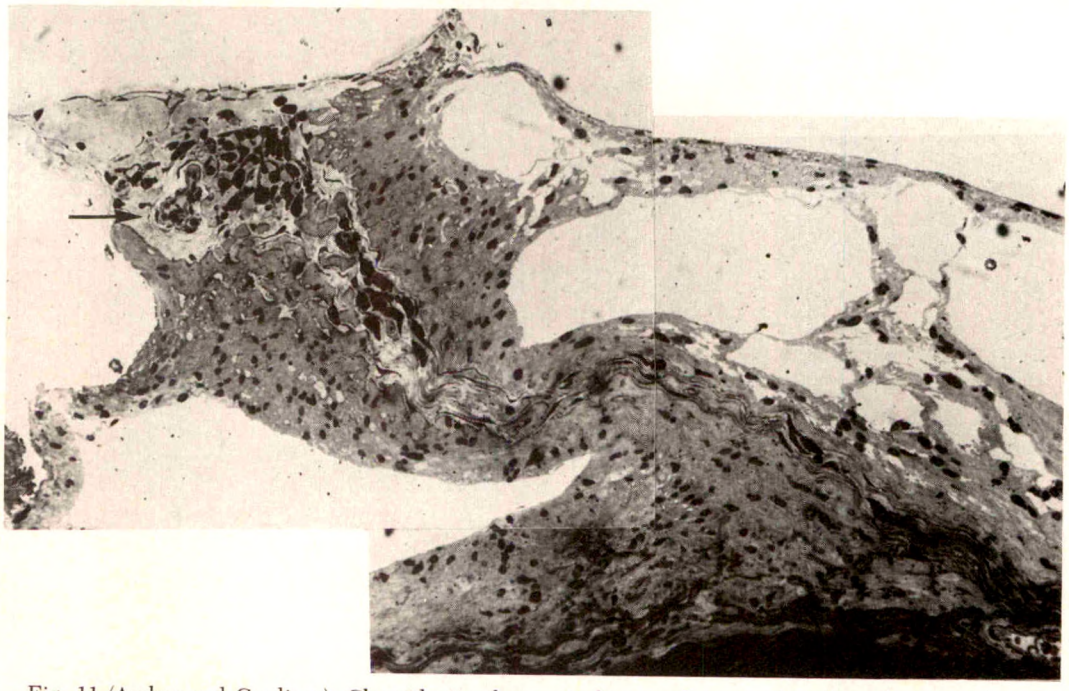


Fig. 11 (Archer and Gardiner). Choroidovitreal neovascular complex extending from the choroid to the preretinal region (arrow). The new vessels are accompanied by fibrous tissue and pigmented cells of probable choroidal origin. The neighboring retina is degenerate with areas of retinoschisis (× 300).

(Fig. 10). This common adventitia was surrounded by a neuroglial layer composed of processes of hypertrophied Müller cells.

New vessels that extended from the choroid through the retina into the vitreous were ensheathed by fibrous tissue and surrounded by numerous pigmented cells, probably of choroidal origin (Fig. 11). Major choroidovitreal channels had the features of choroidal arterioles but the structure of nearby capillary radicals could not be determined at this level of magnification. Major channels that occasionally communicated between two photocoagulation sites had the general features of choroidal arteries.

Discussion

The exact factors that precipitate and sustain choroidal neovascularization in the human eye are not known, although characteristic changes in Bruch's membrane, the retinal pigment epithelium, and outer retina are known to precede and accompany fibrovascular invasion of the subpigment epithelial or subretinal spaces.[1,2] Typical changes include thickening and microfractures of Bruch's membrane and qualitative changes in its collagen and elastic fibers. The deposition of calcium, needle-like crystals, and PAS-positive granular, vesicular, and filamentary material within Bruch's membrane also make it more vulnerable to the ingrowth of new vessels from the choroid. Separation of the retinal pigment epithelium from Bruch's membrane, either by drusen material or serous fluid, also appears to be a key event leading to choroidal neovascularization; indeed, some investigators consider it a necessary prerequisite. Most of these predisposing changes are age-related and probably

account for the absence of spontaneous choroidal neovascularization in the healthy young animal and the difficulties experienced in inducing experimental new vessel growth into the outer retina.

In this experiment, choroidal neovascularization was induced in the rhesus monkey by impoverishing the blood supply to the inner retina and producing defects or damage to Bruch's membrane by intense photocoagulation. Neovascular networks were observed in 14 of 46 eyes after photocoagulation; these occurred predominantly in eyes where intense repeated photocoagulation marks had produced extensive inner retinal ischemia and disruption of Bruch's membrane. No new vessels formed where retinal vascular patency was re-established and where the retinal microvasculature had reperfused.

The choroidal origin of the new vessels was confirmed histologically in four instances, although most of the communicating vessels identified were of larger order, joining major vascular radicals in the outer choroid. These vessels were from well-established fronds and showed few of the early vasoformative changes that might have originated from the choriocapillaris layer.

Fluorescein angiography showed that well-established neovascular networks perfused synchronously with the choroidal circulation and independently of the retinal vasculature that was characteristically attenuated or absent in the region of neovascularization. All neovascular networks were grossly incompetent to dye during angiography, particularly at their growing tip, and could be easily identified by the rapid outpouring of dye from their walls into nearby retina or vitreous during the early phase of angiography. This profuse leakage of dye was distinguishable from the hyperfluorescence that occurred at retinal pigment epithelial defects or from zones of exposed choriocapillaris that bordered photocoagulation marks. The major arterial and venous trunks that served actively growing neovascular fronds were more competent to dye, although in the late phases of angiography fluorescein leaked for a short distance into the neighboring retina. It is likely that their thickness (three or four layers) and the surrounding condensation of fibrous tissue had an important bearing on their degree of integrity and competence to fluorescein.

Most neovascular complexes infiltrated the subretinal space only in the immediate vicinity of photocoagulation marks for a short distance beyond the confines of photocoagulation scars. In several instances, there was evidence of regression six to eight weeks after photocoagulation. These vessels may have represented a healing response of the choroidal vasculature that waned after a scar was formed. Conversely, other neovascular fronds invaded the subretinal space for distances of 5 or 6 disk diameters and developed well-defined supporting vessels. The rate of growth of these fronds was typically slow, barely exceeding 2 to 3 mm per month. Occasionally, we noted small hemorrhages at or adjacent to the advancing front of the neovascular frond. The factors determining the direction and rate of neovascular propagation were not clear; however, new vessel formation was characteristically confined to regions where overlying retina was ischemic and necrotic, and in no instance did new vessels invade normal retina.

Choroidovitreal neovascularization fronds occurred at sites of intense photocoagulation, particularly where a second photocoagulation mark had been placed within an area of ischemic inner retina. Such secondary burns were often associated with choroidal rupture and extravasation of blood through the degenerate retina into the vitreous cavity. Extensive fronds of choroidovitreal neovascularia-

tion developed at such sites within three or four weeks after photocoagulation. When two such complexes developed nearby, the vasoformative fronds from each showed a tendency to establish intervascular links and anastomoses. After resolution of vitreous hemorrhage, the neovascular fronds rapidly regressed and in some instances disappeared entirely, leaving either a formed vascular channel spanning the photocoagulation scars or a fibrous band in the wake of the neovascular tissue. Why such actively proliferating fibrovascular channels should regress so rapidly upon gaining the vitreous cavity is not known, although there is evidence that the vitreous may have an inhibitory effect on vasoproliferation.[9,10] Established vitreous channels seemed more resistant and persisted for many months after the main vasoproliferative outgrowth had receded. These major channels communicated with the choroidal circulation at each extremity and were clearly under low perfusion pressure as blood flow within them was strikingly retarded.

The proliferating neovascular networks were composed of capillaries with well-defined lumina of approximately the same dimensions as the choriocapillaris (10 to 30 μ). The new vessels had well-developed endothelia and basement membranes and possessed pericytes in significantly greater numbers than is common in normal choriocapillaris tissue. Each growing frond was composed of many primitive and proliferating capillaries cut at many angles within each section. Neovascular channels proximal to the growing front had significantly wider lumina surrounded by well-formed endothelial cells and one to four layers of smooth muscle cells. These vessels predominantly occupied the subretinal space and, as they increased in size, were closely surrounded by fibrous tissue and neuroglia. The proliferating capillaries and larger channels typically infiltrated the subretinal space which contained loosely arranged amorphous material, chronic inflammatory cells, and phagocytes scattered about the outer retina. The phagocytes, however, were not particularly concentrated about the advancing edge of the neovascular tissue and were not a feature of the vasoproliferative process. Conversely, retinal pigment epithelial cells in various guises (migratory or giant cells, for example), were always closely associated with neovascular growth.[8]

The two factors that particularly influenced new choroidal vessel formation in this experiment were dehiscences in Bruch's membrane and alterations in the cellular structure and architecture of the outer retinal parenchyma which appeared to provide a suitable environment for vasoproliferation. Choroidal neovascularization developed only where breaks in Bruch's membrane had occurred. However, defects in Bruch's membrane alone are insufficient to provoke subretinal neovascularization. Breaks produced in normal retina by intense photocoagulation only result in a limited growth of fibroglial tissue from such sites toward the vitreous cavity without significant or detectable vasoproliferation.

The special conditions favoring vasoproliferation after retinal vascular occlusion are not clear; however, vein occlusion may produce widespread necrosis of the inner retina and open the subretinal space after the seepage and dispersion of fluid and blood from obstructed inner retinal vessels. Convenient low-resistance pathways are thus available for the extension of neovascular channels beneath the retina. Dense and compact tissue resists infiltration by blood vessels, as shown by the resistance of normal corneal stroma to invasion by corneo-

scleral limbal neovascular fronds and the role of corneal edema in the development of neovascularization.[11,12]

In this experiment no new choroidal vessels invaded the subpigment epithelial region. In this respect our experimental model differs from the clinical situation where the subpigment epithelial ingrowth of vessels is the most common preliminary to vascularization of the subretinal space and initiation of the disciform response. The failure of new choroidal vessels to invade the subpigment epithelial region in this experiment may be related to the application of intense photocoagulation marks which may have produced a firm attachment of the retinal pigment epithelium to Bruch's membrane, thus closing the potential subpigment epithelial space.

This experiment provides no firm description of the precise stimulus to vasoproliferation, although, theoretically, filterable vasoformative factors could be liberated by degenerating retinal cells or from the abnormally proliferating pigment epithelial cells that attend the neovascular fronds. It is unlikely that choroidal ischemia has any major role in the formation of new vessels, as the choriocapillaris in this model remained well perfused except in the immediate vicinity of the photocoagulation burns. We do not know whether retinal hypoxia has any bearing on the formation and maintenance of choroidal neovascularization because we did not make direct measurements of oxygen tension in the vicinity of new vessels. However, as the choriocapillaris was normally perfused and the inner retina largely necrotic, containing only a few viable and presumably actively metabolizing cells, it would seem that hypoxia was not a major factor. Ernest and Archer[13] measured oxygen tension in a similar situation in monkeys where superior and inferior temporal branch retinal veins had been occluded to produce extensive inner retinal ischemia. They found little detectable difference in oxygen tensions between ischemic inner retina and adjacent normal retina, and it is likely that the findings would be similar in this experiment.

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BILATERAL JUXTAPAPILLARY SUBRETINAL NEOVASCULARIZATION ASSOCIATED WITH PSEUDOTUMOR CEREBRI

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A 32-year-old obese woman with hypertension and a three-year history of pseudotumor cerebri developed bilateral juxtapapillary subretinal neovascular membranes. To our knowledge, this is the first reported case of bilateral subretinal neovascular membranes complicating the course of this disease. The subretinal neovascular membrane in the left eye spontaneously involuted, but because the membrane in the right eye threatened the foveola, the patient underwent argon-laser photocoagulation. The subretinal fluid and hemorrhage progressively resolved, the membrane was replaced by fibrous tissue, and visual acuity improved.

The pathogenesis of the subretinal neovascular membranes was presumably secondary to pressure deformity of the border of Bruch's membrane at the optic disk, creating a discontinuity of normal anatomic apposition of the chorioretinal layers. This anatomic dehiscence, coupled with hypoxia created by axonal tissue swelling and resultant impaired vascular perfusion of the tissues, led to the development of subretinal neovascular membranes.

The list of ocular diseases in which subretinal neovascular membranes have been observed is constantly expanding. To date, these diseases include senile macular degeneration, presumed ocular histoplasmosis syndrome, idiopathic subretinal neovascularization in both old and young patients, severe myopia, healed disseminated chorioretinitis of unknown origin, drusen of the optic nerve head, hereditary drusen of Bruch's membrane, angioid streaks, toxoplasmosis, toxocariasis, sarcoidosis,[1-4] pseudotumor cerebri,[5,6] choroidal folds,[7] Best's vitelliruptive macular degeneration,[8] rubella,[9,10] choroidal nevi, choroidal melanomas, choroidal hemangiomas, hamartomas of the retinal pigment epithelium, metastatic tumors,[11] coccidiomycosis, Harada's disease, acute multifocal posterior placoid pigment epitheliopathy, Behçet's disease,[12] geographic helicoid (serpiginous) choroiditis,[13] and choroidal rupture.[14,15] They also occur after excessive photocoagulation.

To the best of our knowledge, this is the first report of bilateral juxtapapillary subretinal neovascular membranes in a woman with pseudotumor cerebri. The two previous cases of unilateral disease occurred in men.[5,6] In our patient, one eye underwent a spontaneous involution of the subretinal neovascular membrane without treatment, whereas the fellow

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eye required argon-laser photocoagulation because of progressive enlargement of the subretinal neovascular membrane that threatened the foveola.

Case report

A 32-year-old obese woman was examined here in the fall of 1976. She had had right frontal headaches and intermittent dizziness throughout the previous year. During the previous six months she had noticed episodes of blurred vision lasting 20 to 30 minutes. These were so severe that she was unable to see anything clearly. She had a history of hypertension and rheumatic fever as a child. She was being treated with a mixture of spironolactone and hydrochlorothiazide (Aldactazide) and a potassium supplement.

Results of a physical examination were unremarkable. Her blood pressure was 130/96 mm Hg. Results of a neurologic examination were within normal limits, except for a slight nystagmus on right lateral gaze.

Ophthalmic examination disclosed an uncorrected visual acuity of 6/6 (20/20) and 4 point in both eyes. Intraocular pressure was 14 mm Hg in each eye by applanation tonometry. Results of anterior segment and slit-lamp examinations were within normal limits. On ophthalmoscopy, both optic disks showed some hyperemia with slight elevation and blurred margins. There was a mild retinal venous dilation, with no spontaneous venous pulsation on either optic disk, but this was easily elicited by gentle pressure on the globe. Peripheral visual fields were within normal limits.

Results of computed axial tomography of the orbits and brain with iodine infusion and the sodium pertechnetate Tc 99m brain scan were within normal limits. The VDRL test was nonreactive. Lumbar puncture gave an opening pressure of 320 mm H_2O, with clear fluid and no cells; protein level was 29 mg/100 ml and glucose level was 58 mg/100 ml. The diagnosis was pseudotumor cerebri and the patient began taking acetazolamide, 250 mg every six hours, in addition to spironolactone and hydrochlorothiazide.

The patient's symptoms improved, and she had only an occasional mild headache over the ensuing months. In early 1977 treatment with acetazolamide was discontinued.

Periodic follow-up examinations disclosed no change until July 17, 1979. At that time her uncorrected visual acuity was still 6/6 (20/20) and 4 point in both eyes. The bilateral papilledema was more pronounced and we found no spontaneous retinal venous pulsations on the optic disks. There was a small hemorrhage inferotemporal to the optic disk in the left eye. The Goldmann visual fields showed enlarged blind spots in both eyes with a small superonasal visual field defect in the left eye.

The patient was admitted to the hospital on July 23, 1979. Her blood pressure was 132/85 mm Hg. A lumbar puncture, performed July 24, gave an opening pressure of 270 mm H_2O; cerebrospinal fluid was clear and without cells; the protein level was 33 mg/100 ml and the glucose level was 56 mg/100 ml. Results of cytologic studies of the cerebrospinal fluid and immunoglobulin tests were unremarkable. A second computed axial tomographic scan showed small ventricles consistent with the diagnosis of pseudotumor cerebri.

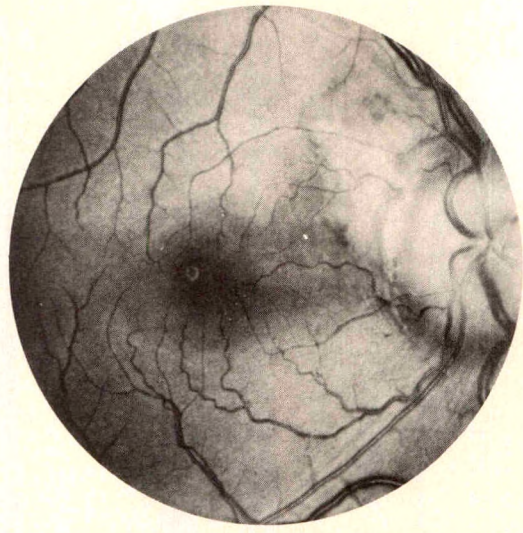


Fig. 1 (Morse and associates). Right eye shows marked papilledema. The juxtapapillary subretinal neovascular membrane is the light-colored, elevated area superotemporal to the optic disk. There are a few small hemorrhages at the margin of this lesion.

On ophthalmic examination Aug. 31, the patient's best corrected visual acuity was R.E.: 6/6 (20/20) and 3 point and L.E.: 6/60 (20/200) and 0 point. The optic disk in the right eye was elevated with capillary dilation giving a slightly hyperemic appearance. There was a light-colored elevation of the retina and retinal pigment epithelium superotemporal to the optic disk (Fig. 1). The left eye showed an elevated optic disk with dilation of the capillaries. There was a light-colored elevation of the retina inferotemporal to the optic disk with retinal edema, hard yellow exudate, some subretinal fluid, and subsensory retinal hemorrhage with retinal striae extending to the foveola (Fig. 2).

On Nov. 30, 1979, her uncorrected visual acuity was R.E.: 6/4.5 (20/15) and 3 point and L.E.: 6/18+2 (20/60+2) and 4 point. Ophthalmoscopy showed some resorption of the inferotemporal peripapillary hemorrhage in the left eye.

By Dec. 28, 1979, the patient had noted an increased blurring of vision in her right eye, but her uncorrected visual acuity was 6/6−2 (20/20−2) and 4 point. On ophthalmoscopy, the right eye showed further superotemporal peripapillary retinal elevation, retinal edema, and subsensory retinal hemorrhage. The left eye showed temporal and inferotemporal retinal pigment epithelial atrophy, nodular

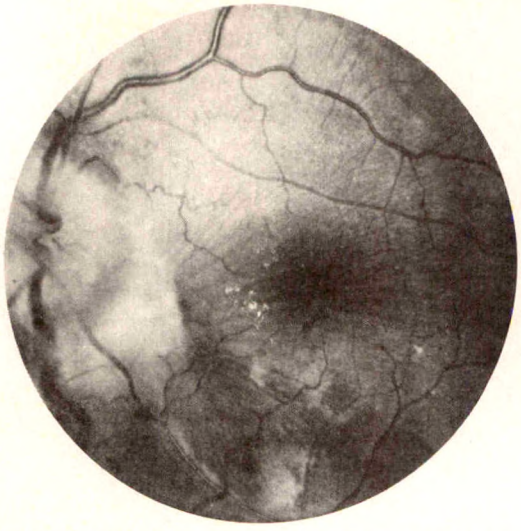


Fig. 2 (Morse and associates). Left eye at time first visual difficulty was noted. The optic disk is elevated and the margins are blurred. Inferotemporal to the border of the optic disk there is a light-colored area beneath which lies the subretinal neovascular membrane. There is surrounding hemorrhage, retinal traction striae, and some hard yellow exudate.

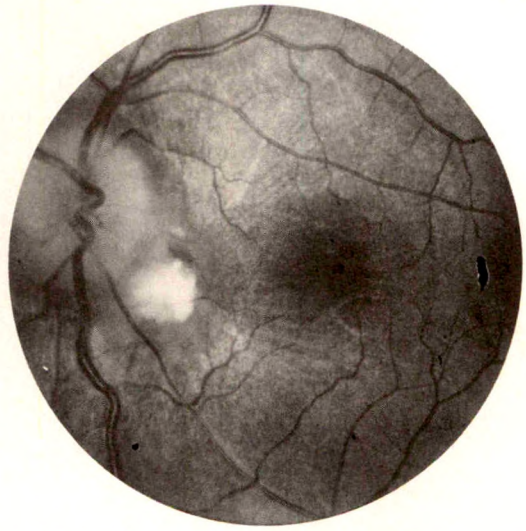


Fig. 3 (Morse and associates). Left eye after spontaneous involution of the subretinal neovascular membrane. A small collar of subretinal fluid not involving the foveola remains. There are some faint lines from subretinal fibrosis and the striae have become less prominent.

subretinal fibrosis, and striae of the retina extending in a superotemporal direction involving the foveola (Fig. 3). Fluorescein angiography demonstrated the staining of the subretinal fibrous nodule and surrounding speckled hypopigmentation and hyperpigmentation as a result of the disruption of the retinal pigment epithelium (Fig. 4).

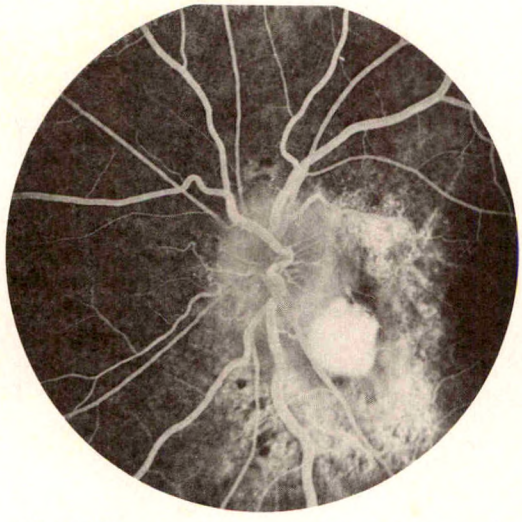


Fig. 4 (Morse and associates). Left eye. Fluorescein angiogram shows the residual subretinal fibrous nodule at the site of the subretinal neovascular membrane. There is a surrounding area of mottled disruption of the retinal pigment epithelium.

On May 19, 1980, the patient complained of decreasing visual acuity in her right eye. Best corrected visual acuity was R.E.: 6/12−1 (20/40−1) and 12 point and L.E.: 6/12+3 (20/40+3) and 4 point. The right eye showed a large light-colored area, delimiting the superotemporal juxtapapillary subretinal neovascular membrane with an increased amount of subretinal fluid and subsensory retinal hemorrhage (Fig. 5). The subretinal neovascular membrane was confirmed and localized by fluorescein angiography (Fig. 6).

On May 30, 1980, the patient's visual acuity was R.E.: 6/18 (20/60) and L.E.: 6/9 (20/30). Because the enlargement of the subretinal neovascular membrane in the right eye threatened the foveola, the patient underwent argon-laser photocoagulation (Fig. 7).

At a follow-up examination on June 20, 1980, visual acuity in the right eye was 6/90 (20/300) and 18 point. There was more subretinal serous transudate visible but the subretinal hemorrhage showed partial resorption. By July 11, 1980, visual acuity had improved to 6/18+2 (20/60+2) and 4 point with progressive resorption of the subretinal fluid and hemorrhage.

By Aug. 15, corrected visual acuity in both eyes was 6/7.5 (20/25) with −0.75 sphere; visual acuity was 4 point at near without correction. The area of the temporal subretinal neovascular membrane adjacent to the optic disk in the right eye was occupied by subretinal fibrous tissue. There were small retinal folds or striae, created by the fibrous membrane exerting traction on the retina, radiating toward the macula (Fig. 8).

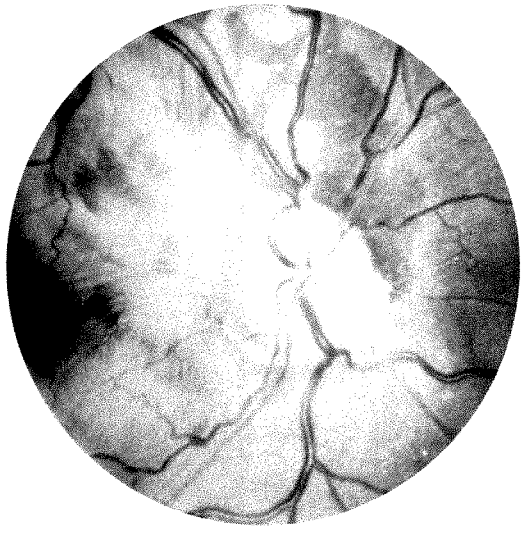


Fig. 5 (Morse and associates). Right eye shows a marked enlargement of the subretinal neovascular membrane superotemporal to the optic disk, with subretinal hemorrhage, serous subretinal fluid, and striae of the retina.



Fig. 6 (Morse and associates). Right eye. Late-stage fluorescein angiogram demonstrates greater intensity of fluorescein staining of the right subretinal neovascular membrane and hyperfluorescence of the optic disk.

Discussion

This patient was, to the best of our knowledge, the first observed with bilateral juxtapapillary subretinal neovascular membranes in pseudotumor cerebri. Although this disease predominantly occurs in women, the two previously reported cases of unilateral subretinal neovascular membranes involved men 31 and 54 years old.[5,6] In our patient, the subretinal neovascular membrane in the left eye underwent a spontaneous involution without

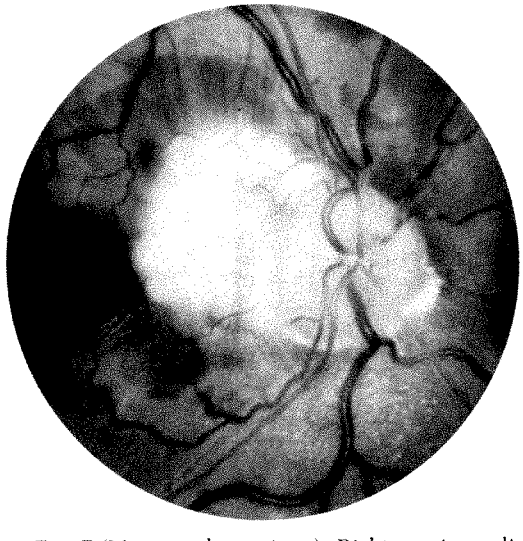


Fig. 7 (Morse and associates). Right eye immediately after argon-laser photocoagulation of the area of the subretinal neovascular membrane.

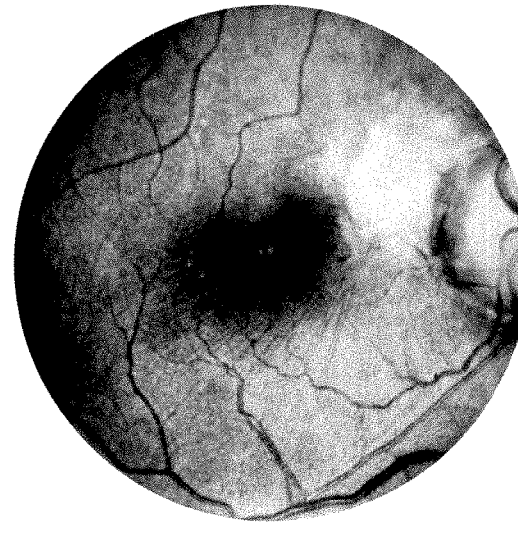


Fig. 8 (Morse and associates). Right eye, 2½ months after photocoagulation of the subretinal neovascular membrane. There is residual subretinal fibrosis and small retinal striae created by traction.

significant damage to central vision. However, in her right eye the subretinal neovascular membrane showed signs of fulminant enlargement, threatening the foveola and papillomacular bundle, and was treated with the argon laser.

The formation of subretinal neovascular membranes is still not fully understood. Normal eyes possess connections between the posterior ciliary and retinal blood vessels at the optic nerve head.[16] The physical deformation of the normal anatomic apposition of peripapillary tissues by papilledema may create the required pathway for the ingrowth of neovascularization beneath the retinal pigment epithelium and sensory retina. The axons of the optic nerve head are bordered by rigid tissue circumferentially and posteriorly and therefore the prelaminar tissue pressure is increased by the swollen axons seen in papilledema. Monocular, inferior, altitudinal visual field defects secondary to ischemia, confirmed by fluorescein angiography, have been documented in a patient with chronic papilledema.[17] Atrophy of the optic nerve head also occurs in patients with chronic papilledema. Evidence points to a compromise of a prelaminar blood supply, the adequacy of which depends upon intraocular pressure, optic disk tissue pressure, and arterial perfusion pressure.[18]

Subretinal neovascular membranes are not usually symptomatic unless they compromise the patient's central vision or visual field. Any patient with a juxtapapillary hemorrhage should be carefully observed because this sign may be the earliest indication of a subretinal neovascular membrane, appearing before it enlarged enough to become visible beneath the elevated retinal pigment epithelium.

The rate of enlargement of subretinal neovascular membranes, their ultimate size, and the time period during which this occurs vary widely. Spontaneous involution without marked damage to the central vision does occur, but any subretinal neovascular membrane in a temporal juxtapapillary location or any subretinal neovascular membrane within the temporal major retinal vascular arcades is a potential threat to central vision. Subretinal neovascular membranes may be stable for weeks to months, and then either involute or undergo sudden rapid enlargement. These lesions must undergo photocoagulation before it becomes unsafe. Once the lesions spread beneath the capillary free zone, photocoagulation is no longer safe. The topographical location of the subretinal neovascular membrane is important. Central vision may be transiently reduced by serous transudate or hemorrhage from a subretinal neovascular membrane which is distant from the foveola and which may undergo photocoagulation safely. After resorption of subretinal fluid and hemorrhage, previous visual acuity is restored. However, any portion of a subretinal neovascular membrane beneath the capillary free zone and foveola cannot undergo photocoagulation safely.

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Fifty years ago this month in The Journal

Among the many excellent plans for increasing the usefulness of "The Academy," none has met with greater favor than that of the Section on Instruction, devoted to "tabloid" courses of postgraduate instruction, splendidly arranged and most acceptably given to groups small enough to lend an intimate and informal character to each period. The most recent addition to this course is the intensive work in practical pathology and pathologic histology, a course necessarily limited to a small number, and which was over subscribed for, practically by return mail, when the notices of it were issued.

Nor should we forget the part the Academy played, in conjunction with the other national special societies, in founding the examining boards for ophthalmology and otolarynology, which have functioned so successfully and which have contributed greatly to a sort of standarization of the preparation for special work.

E. C. Ellett: The Academy. *Am. J. Ophthalmol.* 14:256, 1931

CHOROIDAL NEOVASCULARIZATION AS A LATE COMPLICATION OF OCULAR TOXOPLASMOSIS

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Macular detachment caused by choroidal neovascularization in eyes with previous toxoplasmic infections is one possible explanation of sudden loss of visual acuity. Three patients with quiescent chorioretinal scars, presumed to be toxoplasmic in origin, developed choroidal neovascular membranes that caused sudden loss of visual acuity. In none of the three cases was there any ophthalmoscopic evidence of intraocular inflammation. In two patients, the choroidal neovascularization was subfoveal and, therefore, not suitable for photocoagulation. However, visual acuity in each of the affected eyes was only partially impaired (6/15 [20/50] in one patient and 6/30 [20/100] in the other). In the third patient, the choroidal neovascular membrane was treated with argon-laser photocoagulation. Two months after treatment, visual acuity in that eye was 6/9 (20/30), and there was angiographic evidence that the neovascular membrane had closed.

Among the common features of ocular toxoplasmosis are anterior uveitis, retinochoroiditis, vitreitis, or some combination of these.[1-3] We studied three cases in which visual loss was ascribed to a macular detachment secondary to a choroidal neovascular membrane adjacent to an inactive retinochoroidal scar. In one case, the neovascular membrane was outside the fovea and was treated successfully with argon-laser photocoagulation.

CASE REPORTS

Case 1—An asymptomatic 13-year-old girl had a visual acuity of 6/6 (20/20) recorded during a routine ocular examination in February 1976 and again in November 1977. On each occasion, fundus photographs were taken of a yellow spot beneath the right macula and some perivascular pigmentation below each macula. In March 1978, she noted visual loss in the right eye for the first time. Visual acuity was reduced to 6/7.5 (20/25) in the right eye, and there was an area of retinal inflammation with overlying vitreous cells below the right macula. Photographs were not taken.

A Sabin-Feldman dye test, positive at a titer of 1:128, confirmed the clinical suspicion of toxoplasmic retinochoroiditis, and she was treated with pyrimethamine, sulfadiazine, and systemic corticosteroids.

In May 1978, her visual acuity worsened to 6/24 (20/80) in the right eye, but by June 1978 it had improved to 6/12 (20/40). In August 1978 her visual acuity was 6/9 (20/30) in the right eye and there was no evidence of anterior or posterior segment inflammation. There was a yellow chorioretinal scar below the right fovea (Fig. 1, left). Therapy was discontinued because the lesion appeared to be quiescent.

The patient was examined periodically. On April 25, 1979, her visual acuity was 6/18 (20/60) in her right eye. There was a hemorrhagic lesion above the chorioretinal scar in the right macula. At that time the patient was referred here.

On May 22, 1979, visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/6 (20/20). The media were clear in both eyes. Ophthalmoscopy showed a chorioretinal scar below the fovea in the right eye and a green-gray choroidal neovascular membrane causing a serous detachment of the macula (Fig. 1, right). Subretinal blood was present nasal to the lesion. A fluorescein

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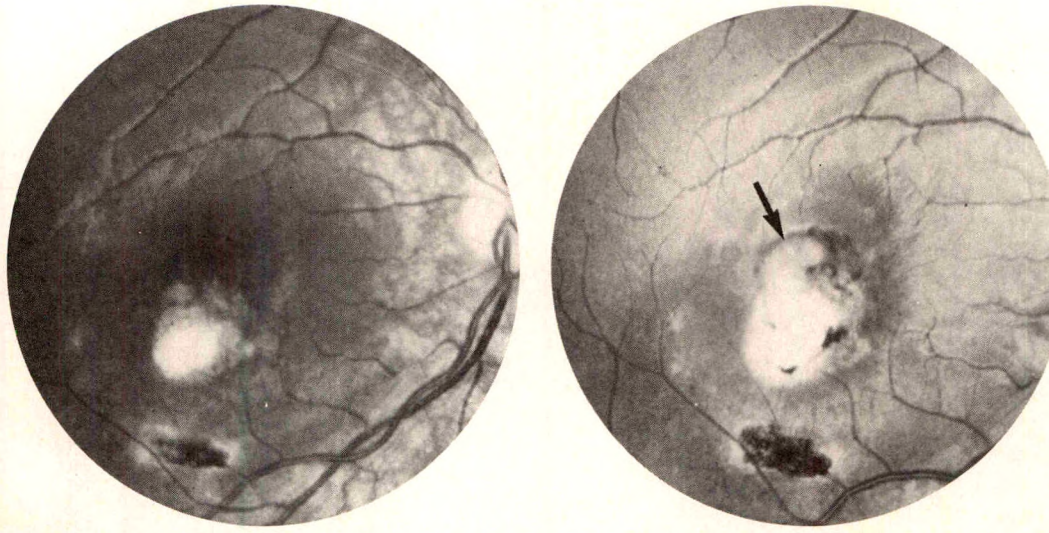


Fig. 1 (Fine and associates). Case 1. Left, Right macula, August 1978, shows chorioretinal scar inferior to fovea. Visual acuity was 6/9 (20/30). Right, Right macula in May 1979. Arrow points to new vessel membrane above chorioretinal scar. Note hemorrhage in fovea. Visual acuity was still 6/9 (20/30).

angiogram showed the neovascular membrane to be subfoveal in position and no treatment was recommended (Fig. 2).

In November 1979, visual acuity in the right eye was reduced to 6/60 (20/200). By February 1980, however, visual acuity had improved to 6/15 (20/50) despite a persistent subfoveal neovascular membrane (Fig. 3).

Case 2—A 53-year-old woman had had bilateral macular chorioretinal scars since she was 12 years of age. These had been ascribed to congenital toxoplasmosis. Visual acuity in the right eye had always been poor, but that in the left eye had been good until July 16, 1979, when it was recorded as 6/12 (20/40). At that time she was referred here.

Four days later, on July 20, 1979, visual acuity was

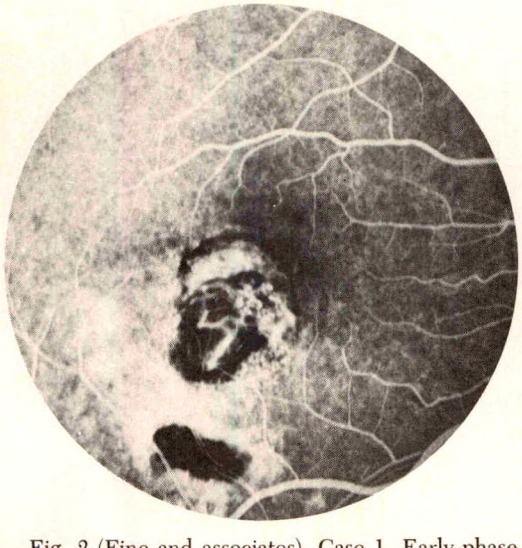


Fig. 2 (Fine and associates). Case 1. Early-phase angiogram shows neovascular membrane under foveal avascular zone.

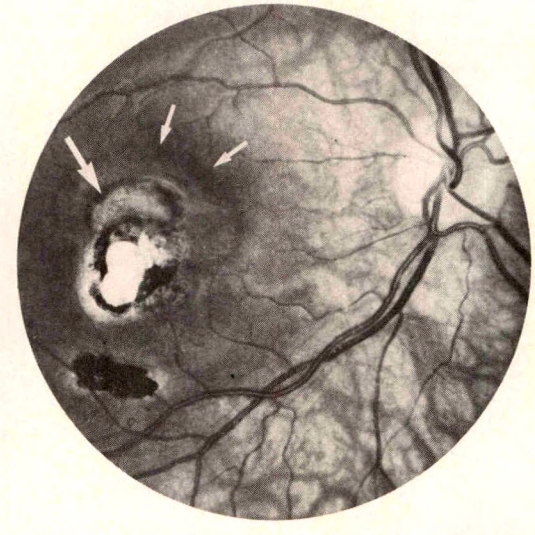


Fig. 3 (Fine and associates). Case 1. Right macula, February 1979, shows persistent subfoveal neovascularization (large arrow) and persistent subretinal fluid (small arrows). Visual acuity was still 6/9 (20/30).

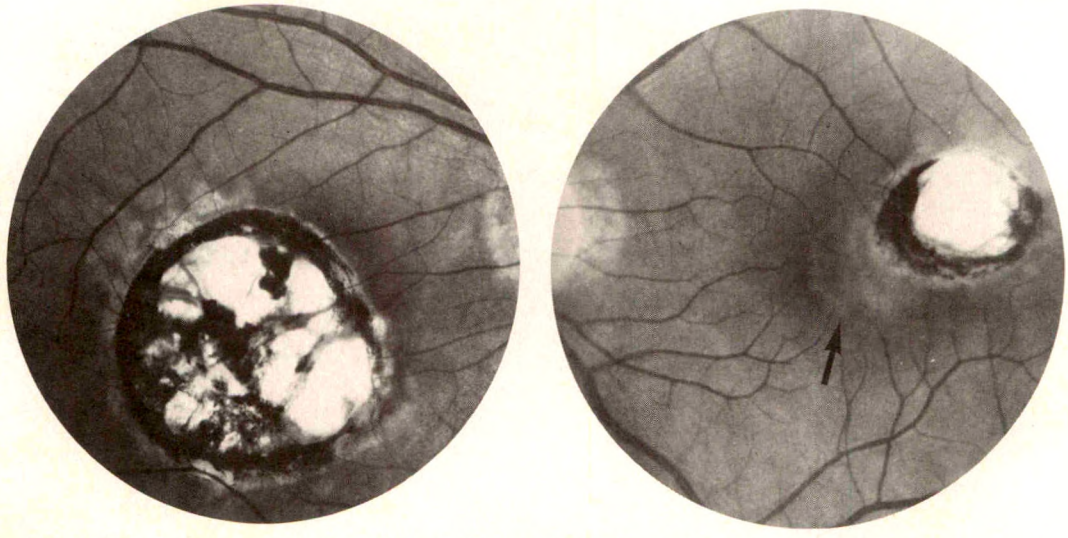


Fig. 4 (Fine and associates). Case 2. Left, Excavated chorioretinal scar involving the right macula of a 53-year-old woman. Right, Chorioretinal scar with serous detachment (arrow) nasal to scar in the left eye.

finger counting in the right eye and 6/12 (20/40) in the left eye. The media were clear in both eyes. Ophthalmoscopy disclosed a large excavated chorioretinal scar in the right eye (Fig. 4, left), and a smaller but similar scar in the left eye temporal to the fovea with a serous detachment of the sensory retina nasal to the scar (Fig. 4, right). The patient was treated with antibiotics and systemic corticosteroids.

When visual acuity failed to improve after three weeks, a fluorescein angiogram was performed. The angiogram showed a choroidal neovascular membrane nasal to the scar with late-phase leakage under the sensory retina (Fig. 5). Because visual acuity was threatened by a neovascular membrane outside the fovea, argon-laser photocoagulation was applied after retrobulbar anesthesia had been administered (Fig. 6, left).

Two months later, in October 1979, visual acuity was 6/9 (20/30) and the sensory retina was flat (Fig. 6, right). Fluorescein angiography at this time showed no further leakage of dye.

Case 3—An 18-year-old man had had a punched-out, sharply defined lesion just inferior to the left macula since he was 5 years old. Results of ophthalmoscopic and slit-lamp examinations were otherwise normal. The lesion was two thirds of a disk diameter in size with fine pigment clumping in the temporal portion (Fig. 7). Corrected visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/9+ (20/30+). The lesion was considered to be secondary to a previous infection with *Toxoplasma gondii*, although it was less heavily pigmented than the typical toxoplasmosis scar.

The patient had been examined at two-year intervals since he was 5 years old. The macular lesion was inactive until March 1, 1977, when the patient complained of difficulty in reading with the left eye of one day's duration. Corrected visual acuity was 6/6 (20/20) in both eyes. The media were clear. A slit-lamp examination showed no evidence of intraocular inflammation. The right fundus was normal. The left macula had extensive sensory retinal detachment, retinal striae, and hemorrhage temporal to the fovea. Fluorescein angiography demonstrated a choroidal neovascular membrane under the center of the fovea (Fig. 8, left). No treatment was recommended. In June 1979, more than two years later, a central

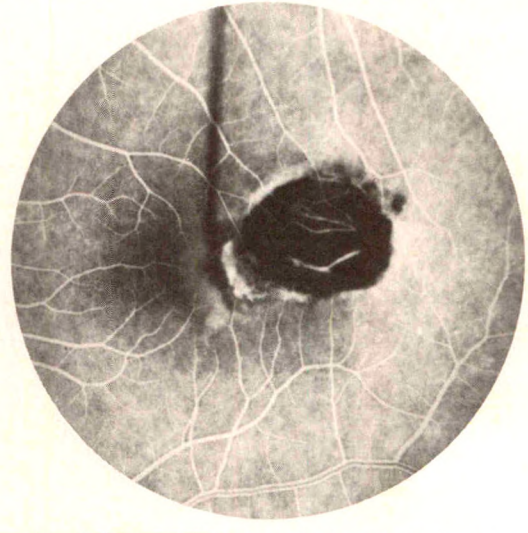


Fig. 5 (Fine and associates) Case 2. Early-phase angiogram shows neovascular membrane adjacent to scar but outside foveal avascular zone.

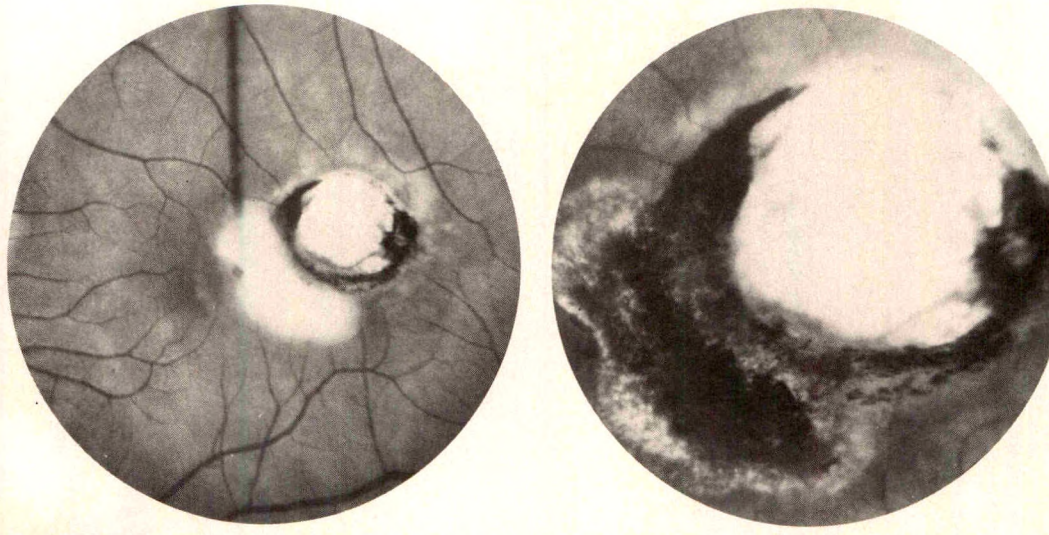


Fig. 6 (Fine and associates). Case 2. Left, One day after photocoagulation. Right, Closer view two months after photocoagulation; visual acuity was 6/9 (20/30) (×2).

disciform nodule was present in the macula (Fig. 8, right). Visual acuity at that time was 6/30 (20/100).

Discussion

Ocular toxoplasmosis is caused by *T. gondii*, an obligate intracellular parasite whose primary host is the cat.[4,5] Recent studies have suggested that virtually all ocular toxoplasmosis in humans represents a recurrence of congenital infection acquired in utero from a mother who acquired the infection during the first trimester of pregnancy.[6] The ocular disease is characterized by a focal necrotizing retinochoroiditis.[7] Involvement of the anterior retina usually produces vitreous opacities. The clinical pattern includes anterior and posterior segment inflammation. Recurrences typically occur adjacent to old scars years after the initial infection.

Retinal vascular lesions previously reported in eyes with toxoplasmosis include branch artery obstruction of a vessel passing through an area of acute retinitis, periphlebitis with or without venous insufficiency, concomitant periphlebitis and periarteritis, segmental periarteritis, and choroidal neovascularization during the acute stage of retinochoroiditis.[8-12] Gass[13] included an illustration of an eye with a neovascular membrane adjacent to a toxoplasmosis scar and provided a limited amount of clinical information about the patient.

The three cases reported here repre-

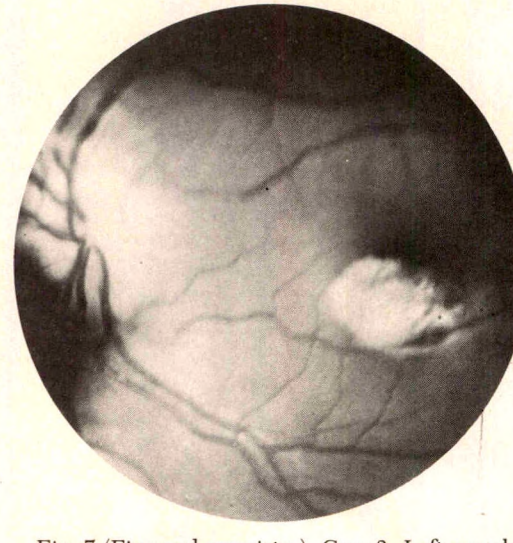


Fig. 7 (Fine and associates). Case 3. Left macula, 1967, shows asymptomatic chorioretinal scar.

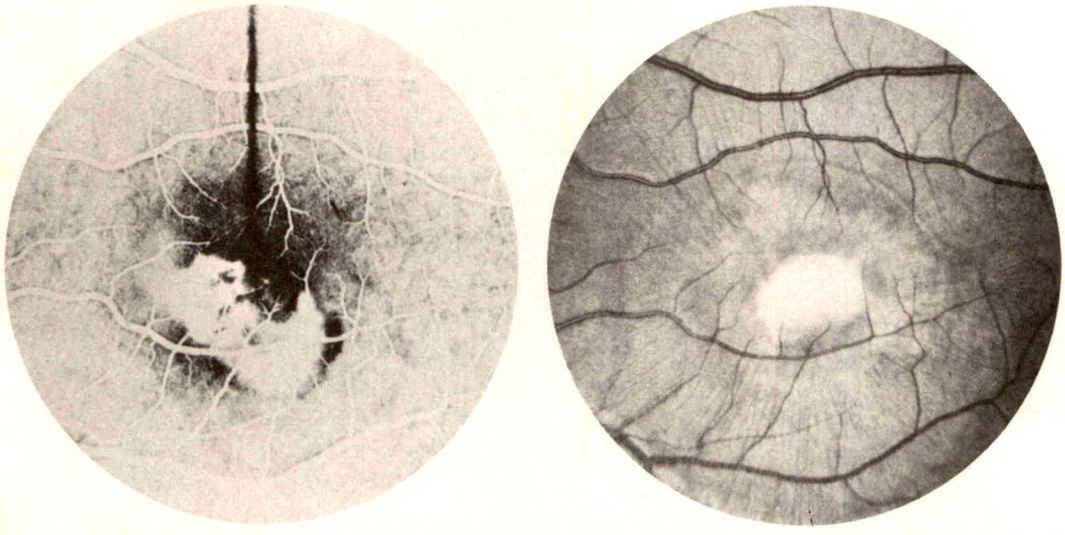


Fig. 8 (Fine and associates). Case 3. Left, Angiogram, March 1977, shows neovascular membrane under fovea. Right, Left macula, July 1979, shows disciform scar. Visual acuity was 6/30 (20/100).

sent a series in which choroidal neovascular membrane formation developed adjacent to a quiescent chorioretinal scar in an eye with no ophthalmoscopic evidence of active intraocular inflammation. In all three cases, there was a presumptive diagnosis of previous toxoplasmic infection. In the second case, photocoagulation was used to treat a choroidal neovascular membrane adjacent to a toxoplasmosis scar, and the short-term visual outcome was excellent. In the first and third cases, the subfoveal position of the neovascular membrane precluded photocoagulation. Visual acuity in these two eyes, however, has been only partially impaired to date. It is important to consider macular detachment secondary to choroidal neovascularization as a cause of sudden loss of visual acuity in patients with inactive toxoplasmosis scars.

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ACYCLOVIR AND DEBRIDEMENT IN THE TREATMENT OF ULCERATIVE HERPETIC KERATITIS

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We treated 25 patients with dendritic keratitis with debridement and 3% acyclovir ointment and another 25 with acyclovir alone. The patients were randomly assigned to the two treatment groups. There were only minimal adverse effects in both groups. The combination of debridement and acyclovir produced a significantly more rapid healing rate than did acyclovir alone. Factors associated with prolonged healing time were duration of symptoms (longer than one week), length of the epithelial defect (>4 mm), proximity of the defect to the corneoscleral limbus (<2 mm), and the presence of stromal inflammation.

Mechanical debridement and topical antiviral therapy are widely used, effective treatments for ulcerative herpetic keratitis. Studies have demonstrated the efficacy of minimal debridement as a precise and nondamaging physical method.[1,2] However, although debridement leads to more rapid healing of an epithelial ulcer than an antiviral medication alone, focal lesions recur in up to 60% of patients, necessitating additional antiviral therapy.[3] Previous clinical trials of the use of antiviral medications in the treatment of herpetic corneal ulceration have shown acyclovir (9-[2-hydroxymethoxymethyl] guanine) to be highly effective with low toxicity.[4,5] Because the ideal treatment should promote rapid, uneventful healing with a minimum of adverse effects, we have conducted a randomized clinical trial comparing the combination of debridement and 3% acyclovir ointment with acyclovir without debridement in the treatment of dendritic keratitis.

SUBJECTS AND METHODS

We included 50 consecutive patients with typical dendritic corneal ulceration in the study; 36 were male and 14 female. The average age was 48 ± 16 years (range, 7 to 74 years). We excluded patients who had visual acuities of 6/24 (20/80) or worse in the fellow eye, women of child-bearing age, and those who were not able to return for follow-up examinations. All patients who entered the study completed the treatment course and follow-up.

At the initial visit, a drawing was made and accurate measurements were taken of each ulcer with Bengal rose 1% and fluorescein stains. We classified the patients by the total length of the dendritic ulceration (shorter or longer than 4 mm) and by the degree of keratouveitis (quantified by a scoring system[6]). Factors included in the total score were the area of stromal inflammation, indicated by the number of quadrants involved (0 to 4); the density of the opacity, indicated by the visibility

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of iris detail (0 to 3); the extent of active corneal blood vessels (0 to 3); and the intensity of anterior chamber inflammation (0 to 3).

We randomly assigned patients within these categories to one of two treatment groups. Group 1 received initial debridement followed by 3% acyclovir five times daily and 1% atropine eyedrops twice daily. Group 2 received 3% acyclovir ointment five times daily and 1% atropine eyedrops twice daily.

Debridement was performed with 1% benoxinate solution and a sterile cotton-tipped applicator to remove the loosened diseased corneal epithelial cells at the ulcer edge (Fig. 1).

We examined each patient daily until the epithelial defect had healed, as indicated by the disappearance of specific Bengal-rose staining of the precise site of the healing dendritic ulceration. We did not consider fine microscopic punctate staining diffusely arranged on the epithelial surface when judging healing time. We recorded all symptoms and signs of a local adverse effect of the therapy. When the ulcer was judged to be healed, the atropine was discontinued and the dosage of the antiviral medication was reduced to three times daily for five days and then discontinued. The patients stopped using all other topical medications during the period of the trial.

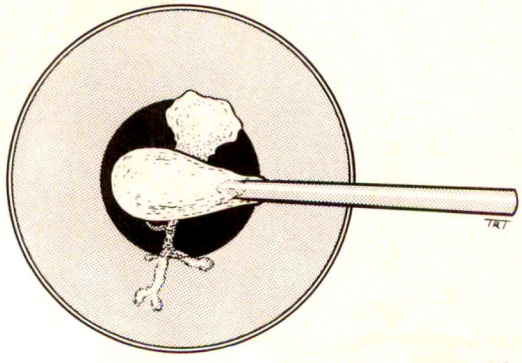


Fig. 1 (Wilhelmus, Coster, and Jones). Minimal debridement of a dendritic ulceration performed with a cotton-tipped applicator.

We examined each patient seven to ten days after the discontinuation of the acyclovir ointment. Each was advised to return three and six months later for follow-up evaluation and earlier if there was any ocular complaint. We recorded the occurrence of any herpetic epithelial ulceration, stromal keratouveitis, or recurrent epithelial erosions during the six-month observation period.

We compared the healing times of the two treatment groups by constructing actuarial survival curves. We used the log-rank test[7] to assess the statistical difference between the treatment groups and to determine which of the pretreatment factors were associated with the rate of healing.

Results

Each group contained 25 patients. Cumulative frequency curves of healing times showed that those patients treated with the combination of mechanical debridement and 3% acyclovir ointment healed more rapidly than those treated with acyclovir alone (Fig. 2). A log-rank analysis demonstrated that the difference between the two groups was statistically significant ($\chi^2 = 5.93$; d.f. = 1; $P < .025$). No recrudescence of viral epithelial disease occurred in either treatment group.

By constructing "life tables" of pretreatment variables from patients of both treatment groups and subjecting the data to a log-rank test, we identified the prognostic factors related to the rate of healing in these 50 patients. Healing was significantly slower with dendritic ulcers which were (1) symptomatically present for more than one week, (2) longer than 4 mm, (3) associated with underlying stromal inflammation, or (4) located within 2 mm of the corneoscleral limbus (Table 1).

Mild to moderate discomfort that re-

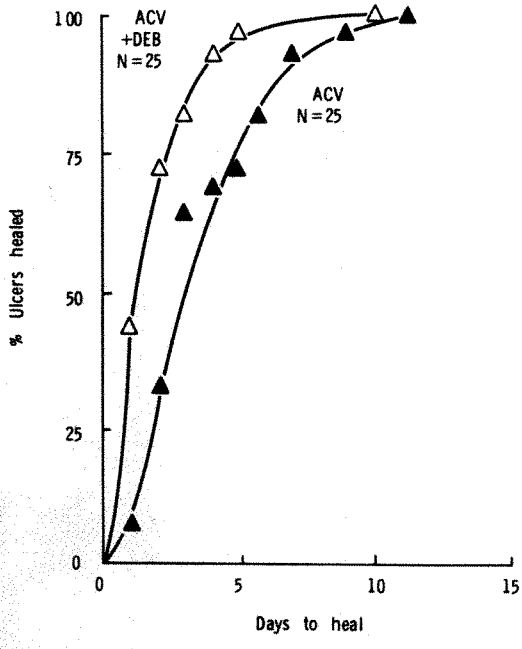


Fig. 2 (Wilhelmus, Coster, and Jones). Cumulative frequency distribution of rates of healing of dendritic ulceration treated with either 3% acyclovir ointment and debridement (ACV + DEB) or acyclovir alone (ACV).

solved within 12 to 24 hours occurred after debridement. Whether debridement was performed or not, the only adverse symptom related to topical acyclovir was a mild transient stinging sensation immediately after application of the antiviral ointment in nine (18%) patients. We found mild punctate epithelial staining with Bengal rose of the inferior bulbar conjunctiva and corneoscleral limbus remote from the original lesion in eight (16%) patients; this occurred, on the average, 14 days after treatment (Fig. 3). This punctate staining was trivial and disappeared within three days after discontinuation of the antiviral medication.

During the subsequent six-month observation period, recurrent dendritic ulceration occurred in three patients; one had been treated with both debridement and acyclovir and two had received acyclovir alone (Table 2). Symptomatic recurrent epithelial erosions were present in three patients, two of whom had received debridement and acyclovir. Herpetic stromal keratouveitis occurred in six patients during the follow-up period. Disciform keratouveitis was present in five patients and irregular stromal infiltrates in one patient. Table 2 shows the distribution of keratouveitis.

Discussion

This study established that a combination of debridement and 3% acyclovir ointment results in more rapid healing of dendritic ulceration than treatment with antiviral chemotherapy alone. Epithelial healing brings about reduction of the patient's symptoms and re-establishes a protective barrier against secondary infection and should be a principal objective in the treatment of ulcerative herpetic keratitis.

A number of ways of treating herpetic

TABLE 1

Factors associated with prolonged healing time

Pretreatment Data	No. of Patients	χ^2	d.f.	P
Symptoms present > 1 wk	17	59.11	1	< .0005
Epithelial defect > 4 mm	20	70.10	1	< .0005
Stromal inflammation present	14	28.03	1	< .0005
Defect < 2 mm from corneoscleral limbus	14	46.61	1	< .0005

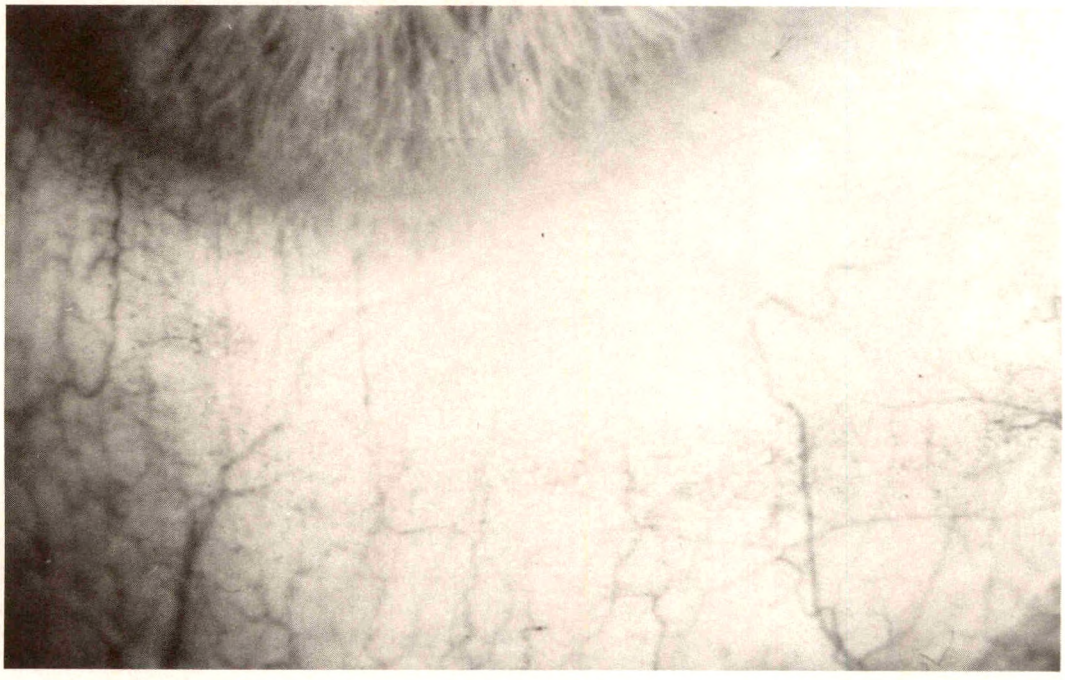


Fig. 3 (Wilhelmus, Coster, and Jones). Linear punctate staining of the inferior bulbar conjunctiva after 14 days of topical 3% acyclovir ointment.

corneal ulceration had been investigated before the introduction of antiviral chemotherapy. Because chemical cauterization and cryotherapy can damage Bowman's membrane and result in scar formation or possibly damage to endothelial cells, minimal debridement has been considered a safe and convenient method of treating dendritic ulcers.[1,2] Wiping the corneal surface with a sterile cotton-tipped applicator easily removes the visibly abnormal epithelial cells at the edge of an active herpetic ulcer. The apparent disruption of intercellular bridges with reduced cellular adherence that may be associated with active viral replication[8] facilitates the selective removal of the virus-infected cells. The ulcer usually heals rapidly, but focal microscopic epithelial lesions recur in up to 60% of

TABLE 2

NO. OF PATIENTS WITH CORNEAL DISEASE DURING SIX-MONTH OBSERVATION PERIOD

	Treatment Group		Total	
Corneal Disease	Debridement + Acyclovir (No. = 25)	Acyclovir Alone (No. = 25)	No.	%
Dendrite	1	2	3	6
Recurrent erosions	2	1	3	6
Stromal keratouveitis	1*	5†	6	12

*Occurred on day 40.
†Occurred on days 10, 11, 42, 66, and 67.

patients within seven days.[3] Thus, additional antiviral therapy is needed to suppress the development of these herpetic recrudescences after debridement.

Acyclovir, an acyclic analogue of 2-deoxyguanosine, has a potent antiviral action against human herpesviruses types 1 and 2. This antiviral effect occurs by selective inhibition of virus-coded DNA polymerase after highly selective phosphorylation of the drug by virus-specified thymidine kinase.[9] Moreover, the compound has good intraocular penetration and is minimally metabolized in humans. This high antiviral specificity and low host toxicity make acyclovir a promising drug for the treatment of herpetic keratitis.[4,5]

In the present study, all ulcers healed within 11 days. More rapid healing resulted from the combination of acyclovir and debridement than from acyclovir therapy alone. Only minimal adverse effects were associated with either mode of treatment. Except for occasional mild discomfort, no complications were related to debridement. Care must be taken during the procedure to avoid damage to the epithelial basement membrane in order to minimize recurrent epithelial erosions. Nine patients (18%) noted a mild stinging sensation, apparently caused by delayed solubilization of the ointment in the precorneal tear film. A transient punctate staining of the inferior bulbar conjunctiva and corneoscleral limbus occurred after 14 days of antiviral use in eight (16%) of the patients.

The ocular inflammatory response appeared to be affected by the treatment of the initial ulceration. During the six-month observation period, six patients developed keratouveitis; five had not been treated with debridement and one patient had been treated with both debridement and acyclovir. The decreased amount of viral antigen, caused by debridement, and the physical removal of infected epithelial cells may have reduced stromal inflammation and subsequent subepithelial scarring.

ACKNOWLEDGMENT

The Wellcome Research Laboratories, Kent, supplied the 3% acyclovir ointment.

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STATISTICAL ANALYSIS OF THE RATE OF RECURRENCE OF HERPESVIRUS OCULAR EPITHELIAL DISEASE

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We studied prospectively the rate of recurrence of epithelial herpetic disease in patients with at least two previously documented episodes of herpes. The estimate of probable recurrence was 24.5% ± 4.8% within 12 months and 32.9% ± 6.5% within 24 months. We found no significant correlation between rate of recurrence and either the age or the sex of the patient. However, the length of time between the two attacks suffered before the study was significantly associated with the length of the interval between entering the study and the next recurrence. Short intervals between previous attacks tended to be associated with short intervals between future attacks, and, therefore, with a higher recurrence rate. By eliminating a given patient from the study after one recurrence, we avoided serious biasing.

Little information is available concerning the rate of recurrence of ocular epithelial herpesvirus disease. In 1936, Gundersen[1] described 221 patients with dendritic keratitis. This group included every patient with corneal infection seen at one institution during an unspecified period of time. On the basis of patient histories, Gundersen found a total of 466 episodes of corneal infection in these 221 patients, an incidence of approximately 2.1 attacks per person. However, only 89 of these patients were being seen for a primary episode; 132 reported previous episodes. Gundersen gave no information about the intervals between clinically diagnosed attacks of herpes infection.

In 1956, Thygeson, Kimura, and Hogan[2] reported 200 cases of herpetic keratitis and keratoconjunctivitis; this group of patients was drawn from an outpatient clinic, referrals to a keratitis research unit, and the private practices of the authors. The authors gave no criteria for the selection of these cases; however, they noted that the patients referred to the research unit tended to have unusually severe or complicated disease. Of the 200 patients, 120 were seen for initial attacks and 38 for second attacks; 42 reported having had two or more previ-

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ous episodes. Many of the patients in this study were seen only once. Some were observed throughout the healing course of the attack, but none were followed specifically from one attack to the next recurrence.

Carroll and associates[3] examined the records of a Boston physician and reported a 26% recurrence rate within two years of the primary attack of dendritic keratitis and a 43% recurrence rate after a second attack. However, this study was retrospective and unstandardized with respect to follow-up times and severity of disease; no virus cultures were done, even in uncertain cases. Additionally, these patients were being treated with idoxuridine or iodine cauterization at the time of the study, although neither treatment appeared to have had an effect on the rate of recurrence.

We examined prospectively a series of patients with at least two previous attacks of ocular epithelial herpes to determine the rate of recurrence and the possibility of a relationship between this rate and the age or sex of the patient or the length of time between successive attacks. Although these patients were being treated with human leukocyte interferon in an attempt to prevent recurrent herpes, we now know that the low-titer interferon used had no effect on the natural recurrence rate.[4]

Because a large number of the patients had to travel long distances for their treatment, some were lost to follow-up. Patients who stopped treatment because of suspected toxicity were followed further for recurrence. Hence, losses to follow-up can be attributed primarily to nonmedical reasons. In applying life-table methods, we assumed that withdrawal had a negligible correlation with the patient's health.

Subjects and Methods

We studied 119 patients at two institutions to determine the rate of recurrence of herpetic keratitis. Each patient had a history of two previous, clinically diagnosed attacks of ocular herpes, and each was free of ocular herpes when entering this study. We examined each patient at monthly intervals until there was a recurrence. The patient was then eliminated from the study.

We estimated the prospective recurrence times, that is, time from entry into the study until the time of recurrence of epithelial herpes, by the life-table methods of Cutler and Ederer[5] (Table 1) and correlated them with age, sex, and time interval between the last two recurrences before the study started with Gehan[6] ranks.

Statistical analysis—The statistical analyses were not the usual parametric tests, in that the variable, prospective recurrence time was censored (Table 2). This led us to use the calculations of Gehan ranks by the method of Mantel.[7]

TABLE 1

Estimates of prospective recurrence rates in 119 patients

Months of Study	Recurrence Rate Estimate (%)	Standard Error (%)	Effective Sample Size	Lost to Follow-up
6	14.4	±3.6	96	20
12	24.5	±4.8	81	13
18	29.1	±5.5	68	3
24	32.9	±6.5	52	1

TABLE 2

STATISTICAL SIGNIFICANCE OF CORRELATIONS WITH PROSPECTIVE RECURRENCE TIMES

Correlation Variable	P	Statistical Method
Age	> .20	Correlation, Gehan ranks
Sex	> .20	Gehan test
Time between last two attacks before study	.03	Correlation, Gehan ranks

We obtained the correlation coefficient between age and Gehan rank and ran a two-tailed test. Similarly, we correlated time between the last two attacks with the Gehan rank, but here we used a one-tailed test because the only logical association was a positive one.

Finally, we used the two-tailed Gehan test[6] for comparing two groups to compare females to males with respect to prospective recurrence rates.

RESULTS

After 1,279 months of follow-up on 119 patients, we evaluated the estimated prospective recurrence rates for the target population with at least two previous documented attacks of ocular herpes by the Cutler-Ederer method[5] at 6, 12, 18, and 24 months (Table 1). For example, we estimated that the probability of a randomly selected patient who was free of herpes at that time and who had had at least two documented attacks experiencing a recurrence within 12 months was 24.5%. The standard error for this estimate is 4.8%. Losses to follow-up for each interval are given in Table 1.

We analyzed the relationships between recurrence times and age, sex, and time between the last two recurrences before the patients entered this study. We could not use standard parametric methods because of the censored nature of the recurrence times. Not every patient had a recurrence during the study. The actual values were replaced by the Gehan ranks. Neither age nor sex correlated significantly with recurrence rate; however, a short time interval between the last two recurrences before the study tended to be associated significantly with future short recurrence times (Table 2).

The patients who were at risk for at least six months and who had no recurrence during the study averaged 27 months (standard error, five months) between the last two documented attacks. Patients who had a recurrence during the study averaged 13 months (standard error, four months) between the prospective recurrence and the last documented retrospective attack. Stating this association another way, of the 26 patients who (1) were prospectively at risk for at least six months and (2) had their last two retrospective attacks less than one year apart, 12 (46%) had a prospective recurrence. All but one of these recurrences occurred within 15 months of entry. This information, when compared with that in Table 1, yields a quantitative picture of the association.

DISCUSSION

To evaluate the effects of various kinds of therapy on the recurrence rate of ocular herpes, it is necessary to compare the recurrence rates seen in treated patients to a baseline recurrence rate representing untreated controls. Published data on recurrence rates have been derived for the most part from studies designed to examine other aspects of herpetic ocular disease, and, as a result, patient selection, disease states, and other factors have been unstandardized and poorly defined. In our study, we attempted to examine patients with epithelial herpes prospectively with regard to possible relationships between recurrence rate and age, sex, and time be-

tween the last two attacks before the study.

Although neither age nor sex appeared to have a significant influence on recurrence rate, the significant ($P = .03$) correlation between the length of time between the last two attacks and the length of time between entering the study and the next attack demonstrated that the length of the interval between attacks should be considered in the design of future studies of recurrence in ocular herpes. Either treatment groups should be balanced with respect to this variable (stratification), or this variable should be adjusted for retrospectively during data analysis.

Precise criteria for the selection of patients in studies of recurrence rates must be described, but probably have only a minor impact in a well-designed study, particularly if each patient is used only once, that is, for the first prospective recurrence interval.

Repeated use of the same patient results in a serious bias in the direction of overestimation of recurrence rate, because short intervals between previous recurrences tend to correlate with short intervals between future recurrences. A patient with a short recurrence time might have more than one recurrence during the period of the study, and would be counted as a separate individual for each separate recurrence. A patient who tended to have long intervals between recurrences might be counted only once. Thus, the study would be biased in favor of patients with a higher rate of recurrence.

It may be that the results of this study still overestimate the recurrence rate of ocular herpes. Because patients with shorter intervals between recurrences may be more likely to seek medical attention and may be more likely to be referred to a university clinic, the population we studied may have been skewed toward shorter recurrence times and, therefore, higher recurrence rates. This bias is much less important, however, than the bias that would be introduced by reuse data for individual patients.

The data for recurrence rate obtained in this study were reassuringly similar to the data derived from previous studies. These recurrence rates permit a more secure calculation of the sample size needed in the design of studies of drugs and other substances being tested for their ability to prevent herpes recurrences. Additionally, the physician can estimate the risk of recurrence, at least for this type of patient, and plan the management of this disease with greater certainty.

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VOGT-KOYANAGI-HARADA SYNDROME WITH FOCAL NEUROLOGIC SIGNS

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The Vogt-Koyanagi-Harada syndrome includes inflammation of the uveal tract, retina, and meninges. Integumentary and auditory signs are common. Neurologic involvement has been reported.

We studied two patients who manifested many of the varied clinical signs of Vogt-Koyanagi-Harada syndrome, but who also showed focal neurologic involvement, including acute transverse myelitis and ciliary ganglionitis. Both patients responded well to corticosteroid therapy.

The findings in these patients and the results of previous studies suggest that Vogt-Koyanagi-Harada syndrome is a cell-mediated autoimmune disorder in which a component of myelin acts as an inciting antigen.

The Vogt-Koyanagi-Harada syndrome, a disorder of unknown origin, is primarily manifested by inflammation of the uveal tract, retina, and meninges. Signs of integumentary (vitiligo, poliosis, and alopecia) and auditory involvement (dysacousia and tinnitus) are common. Neurologic findings have been reported in the Vogt-Koyanagi-Harada syndrome,[1-5] but most of the symptoms have been directly attributed to cerebrospinal fluid pleocytosis, inflammatory arachnoiditis, or the resulting subarachnoid adhesions. Focal neurologic signs in the Vogt-Koyanagi-Harada syndrome have not been stressed in published reports. These signs may prove helpful in determining the cause of this perplexing symptom complex. We studied two cases of Vogt-Koyanagi-Harada syndrome with focal neurologic signs.

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CASE REPORTS

Case 1—A 22-year-old woman noted sudden onset of blurred vision in both eyes. Five days later, persistent frontal headaches and vertigo began. Medical and family histories were noncontributory except for migraine.

On Oct. 25, 1973, one week after the onset of symptoms, her visual acuity was counting fingers bilaterally. She could not distinguish colors. Pupils were normally reactive to light. Anterior segments were clear; no cell or flare was noted. The pupil dilated widely with mydriatics, showing minimal vitreous reaction. Both optic disks were hyperemic. Ophthalmoscopic examination of the right eye disclosed an exudative macular detachment with wrinkling of the internal limiting membrane and a highly elevated bullous detachment located more temporally (Fig. 1). The left fundus had a high bullous detachment just below the optic disk and macula, and irregular, flat detachment of the macula with some overlying vitreous reaction (Fig. 2).

Fluorescein angiography showed early hyperfluorescence of the optic disk and peripapillary retina, with punctate, stippled hyperfluorescence at the level of the retinal pigment epithelium. There were posterior polar foci of blocked fluorescence (Fig. 3, left). Late-phase films showed marked hyperfluorescence of the optic disk and fluorescein staining indicated subretinal pigment epithelial fluid (Fig. 3, right).

The patient began taking 120 mg of prednisone

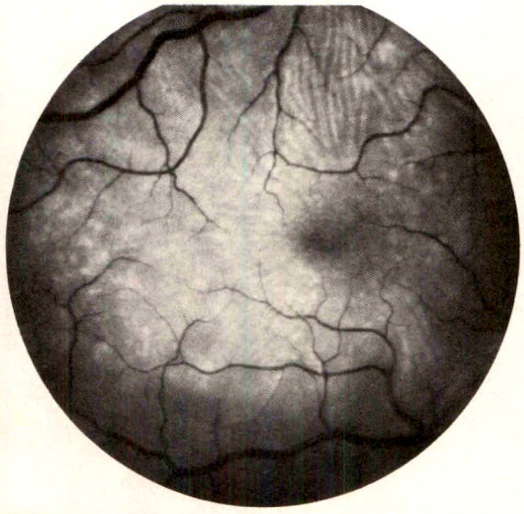


Fig. 1 (Lubin, Loewenstein, and Frederick). Case 1. Right eye. There is a shallow exudative macular detachment with wrinkling of the internal limiting membrane and a highly elevated bullous detachment more temporally (October 1973).

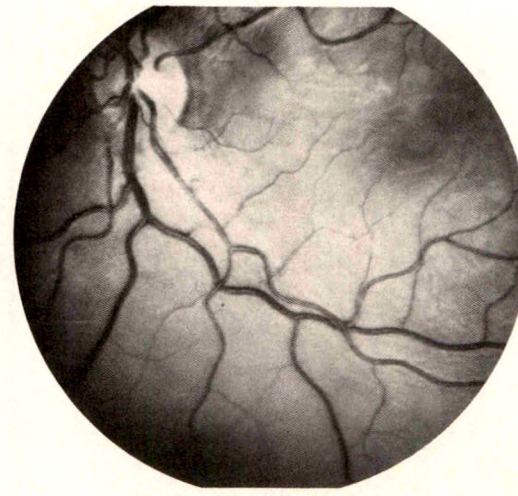


Fig. 2 (Lubin, Loewenstein, and Frederick). Case 1. Left eye. There is an irregular flat detachment of the macula and a bullous detachment just below the optic disk and macula (October 1973).

orally per day, but her visual acuity continued to deteriorate and vertigo made walking impossible. Her headaches persisted. She was admitted to the hospital and treated with 30 mg of dexamethasone per day and intravenously administered mannitol, 2 g/kg of a 20% solution (or 500 ml of a 20% solution). On this regimen, her visual acuity improved to R.E.: 6/30 (20/100) and L.E.: 6/60 (20/200).

A neurologic consultant found mild dysmetria on finger-to-nose testing; this was more marked on the left. She had distinctive bilateral dysmetria in the lower extremities on the heel-to-shin test. Deep tendon reflexes were normal. She had subjective symptoms of vertigo without nystagmus.

Laboratory tests disclosed the following values: hemoglobin, 14.7 mg/100 ml; white blood cell count, 13,800/mm^3; and erythrocyte sedimentation rate,

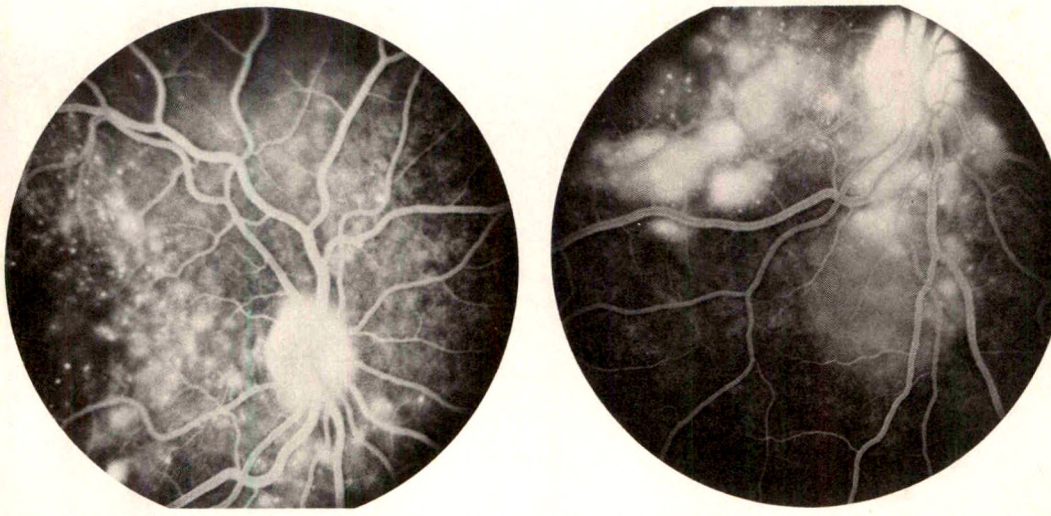


Fig. 3 (Lubin, Loewenstein, and Frederick). Case 1. Right eye. Left, Fluorescein angiogram shows hyperfluorescence of the optic disk and peripapillary retina, with stippled hyperfluorescence at the level of the retinal pigment epithelium. Right, Later in the sequence, fluorescein staining indicates pooled subretinal pigment epithelial fluid (October 1973).

9 mm/hr. Results of serum electrolyte tests, urinalysis, and liver function tests were normal. A lumbar puncture performed on Nov. 19, 1973, after the patient had received corticosteroid therapy for one week, showed an opening pressure of 90 mm H_2O. The cerebrospinal fluid was clear and contained no cells. The protein level was 8 mg/100 ml and the glucose level was 80 mg/100 ml. Immunoglobulin G measured 2.4 mg/100 ml (simultaneous serum IgG was 760 mg/100 ml).

Despite treatment, the headaches and dizziness persisted. On Nov. 24, 1973, the patient complained of back pain. Two days later, she reported increasing pain and weakness of the legs. An examination elicited little voluntary movement of the legs and feet. Knee jerks were 4+ bilaterally and ankle reflexes 3+ with unsustained clonus, but there was no Babinski sign. Position sense was reduced. All sensation was reduced in the legs.

A second lumbar puncture on Nov. 26, 1973, showed that the protein level had increased to 128 mg/100 ml but the cerebrospinal fluid was otherwise normal. Transverse myelitis was diagnosed. Marked pain and weakness of the lower extremities continued for several weeks and then slowly improved.

An ophthalmic examination six months later (April 1974) showed a visual acuity of 6/18 (20/60) in both eyes. Anterior segments were clear and the optic disks were normal. The vitreous was clear. The retinas were flat in both eyes and the macular reflexes had returned, but appeared broader, with pigment stippling of the macula in both eyes. (Fig. 4). A fluorescein angiogram showed a stippled moth-eaten retinal pigment epithelium, most marked in the foveal and parafoveal areas during the arteriovenous phase. There were transmission defects, but no late leakage of fluorescein. Optic disk leakage had resolved (Fig. 5).

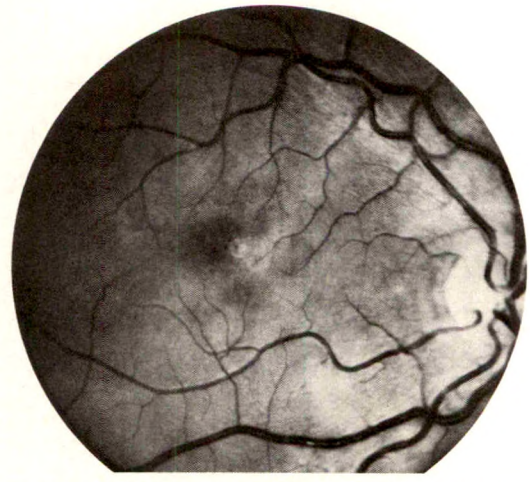


Fig. 4 (Lubin, Loewenstein, and Frederick). Case 1. Right eye. Six months after the initial event the retina has flattened, but pigment mottling has occurred in the macula (April 1974).

Case 2—A 21-year-old man had had one day of rhinorrhea two weeks before admission. Four days before admission, he awoke with a severe right temporal headache which persisted for two days. He

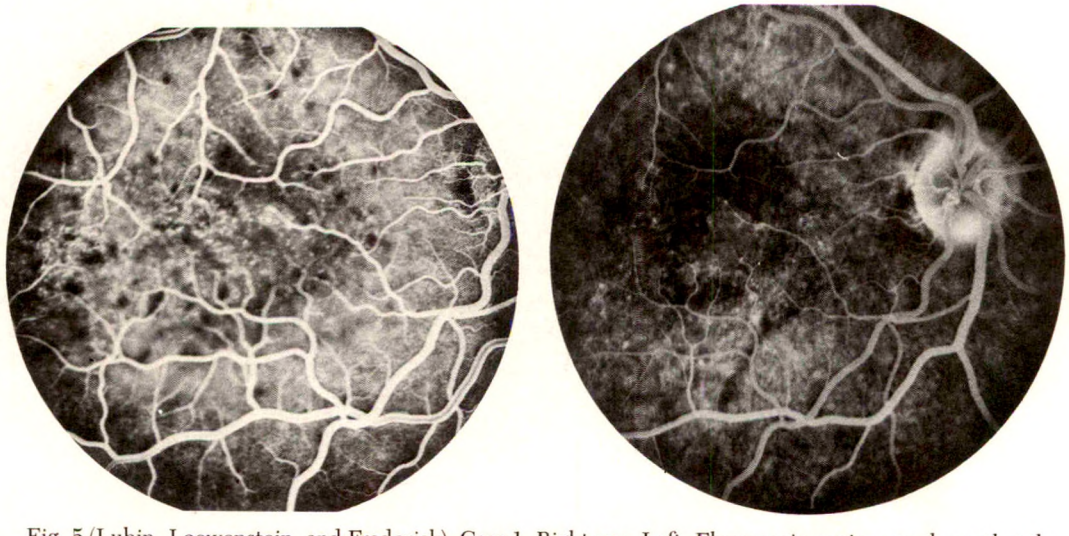


Fig. 5 (Lubin, Loewenstein, and Frederick). Case 1. Right eye. Left, Fluorescein angiogram shows that the retinal pigment epithelium appears stippled and moth-eaten; this is most marked in the foveal and parafoveal areas during the arteriovenous phase. Right, Some transmission defects are present, but there is no leakage of fluorescein (April 1974).

then noted photophobia and blurred vision in both eyes. In the emergency room, he complained of a stiff neck, nausea, and nightsweats of two days' duration. He had recently experienced ataxia and urinary retention. He had had poliomyelitis at the age of 2 years; the disease had caused weakness and decreased muscle bulk of the left leg.

On examination, his temperature was 37.8 C. He had nuchal rigidity. The chest was clear to percussion and auscultation. A cardiac examination disclosed no murmurs. His abdomen was soft and nontender. There was no organomegaly. His skin and hair showed no vitiligo, poliosis, or alopecia. Neurologic findings included an ataxic gait and positive Romberg sign.

On admission, his visual acuity was R.E.: 6/15 (20/50) and L.E.: 6/12 (20/40). Extraocular movements were full. Applanation tensions were R.E.: 13 mm Hg and L.E.: 9 mm Hg. Ishihara plates showed no color vision. He named only the malingering plate. His pupils were 6 mm in diameter in both eyes and were nonreactive to light or near. Corneal sensation was decreased bilaterally. Anterior segment examination disclosed trace flare and occasional cells in the chambers, with fine pigmented keratic precipitates in both eyes. Goldmann lens examination of the vitreous showed 1+ cells. Ophthalmoscopic examination disclosed bilateral optic disk edema, with dilated peripapillary vessels. There was no sheathing of retinal vessels. Cream-colored, irregularly shaped, flat, subretinal lesions were scattered throughout both fundi (Fig. 6). No retinal detachment or subretinal fluid was present.

Blood tests performed when the patient was admitted to the hospital gave the following values: white blood cell count, 7,200/mm^3 with 70 polymorphonuclear leukocytes, 9 band cells, 16 lymphocytes, 3 monocytes, 1 basophil and 1 atypical lymphocyte; hematocrit, 53.1%; hemoglobin, 17.6 mg/100 ml; erythrocyte sedimentation rate, 2 mm/hr; and glucose, 99 mg/100 ml. Results of urinalysis and serum electrolyte tests were within normal limits.

A computed axial tomographic scan of the brain was normal. A lumbar puncture showed an opening pressure of 210 mm H_2O. There were 500 red blood cells and 100 white blood cells (1 polymorphonuclear leukocyte and the rest mononuclear cells). There was no xanthochromia. Protein level was 68 mg/100 ml. Glucose level was normal. Cultures and stains for acid-fast bacilli were negative.

The next morning, the patient's visual acuity had decreased to 6/60 (20/200) in both eyes. There was 3+ flare in the anterior chambers. The pupils remained fixed and dilated to 6 mm in both eyes but constricted to 2 mm after instillation of 0.16% pilocarpine.

Visual field examination on a 2-meter tangent screen with a 10-mm white object showed Bjerrum scotomas in both eyes. At this time, the patient began taking 100 mg of prednisone per day. Visual acuity gradually improved to R.E.: 6/15 (20/50) and L.E.: 6/12 (20/40).

A fluorescein angiographic study showed early-phase blocking of background choroidal blush and there was mild leakage from the optic disk. At 20 seconds, there was patchy hyperfluorescence in the peripapillary choroid and some spots more peripherally (Fig. 7). Hyperfluorescence and staining from these window defects persisted late in the transit (Fig. 8). The stain did not correspond precisely with the lesions visible in the fundus. There was late-phase leakage from the optic disk.

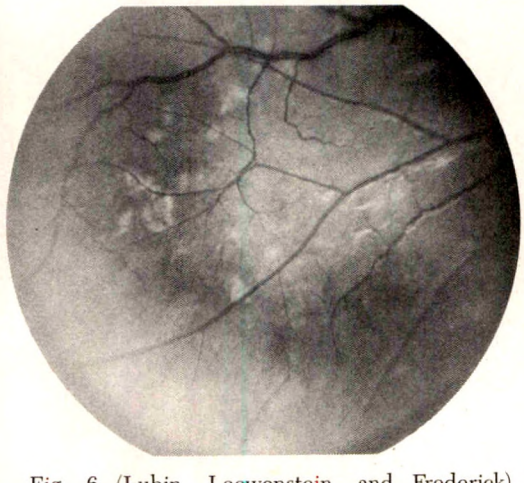


Fig. 6 (Lubin, Loewenstein, and Frederick). Case 2. Right eye. Irregularly shaped, cream-colored flat lesions are scattered throughout the fundus. These lesions are deep, and at the level of the retinal pigment epithelium or choriocapillaris.

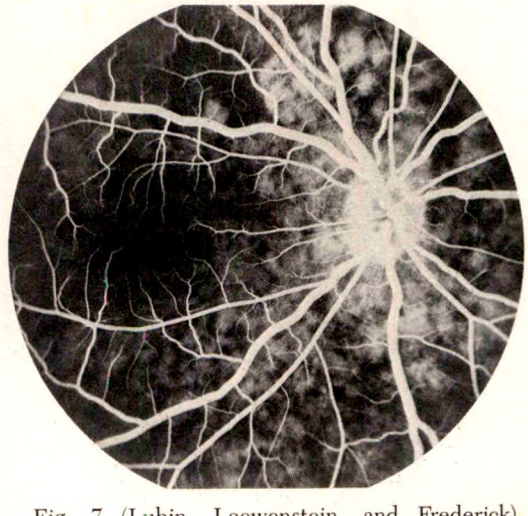


Fig. 7 (Lubin, Loewenstein, and Frederick). Case 2. Right eye. Fluorescein angiogram shows mild optic disk leakage and patchy hyperfluorescence in the peripapillary area, with some spots of hyperfluorescence more peripherally.

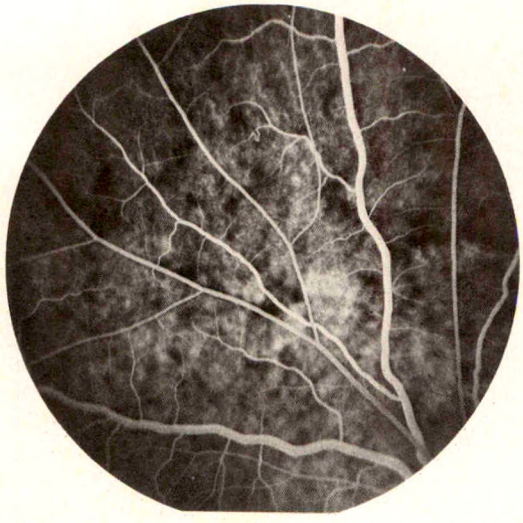


Fig. 8 (Lubin, Loewenstein, and Frederick). Case 2. Right eye. Fluorescein angiogram shows peripheral hyperfluorescent patches at 49 seconds.

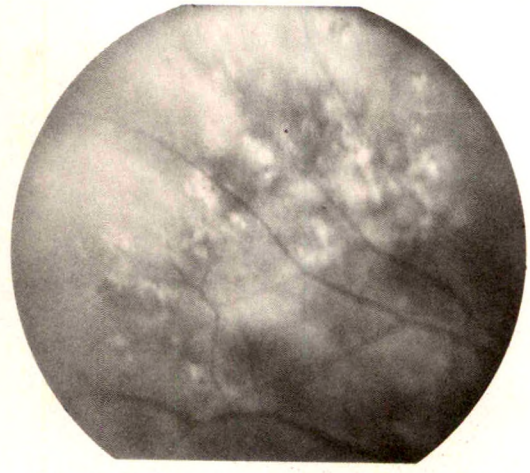


Fig. 9 (Lubin, Loewenstein, and Frederick). Case 2. Right eye. Approximately four months after the initial examination, there are peripheral lesions with darkly pigmented borders.

A second lumbar puncture, performed one week after admission, showed an opening pressure of 190 mm H_2O; there were 17 red blood cells and 392 white blood cells (100% lymphocytes). The patient remained afebrile in the hospital and his symptoms gradually abated. The ataxic gait improved until tandem walk was nearly normal on discharge.

Two months later, visual acuity had improved to 6/6 (20/20) in both eyes, but accommodation was still paralyzed. The pupils were 6 mm in diameter but constricted to 4.5 mm in a bright light. Trace flare and occasional cells in the anterior chambers remained bilaterally. Ophthalmoscopic examination disclosed more white-yellow lesions, but some of the older ones were undergoing pigmentation. At three months, visual acuity remained good; pupils were still 6 mm in diameter but accommodation had returned. The anterior chamber inflammation remained active, with 1+ flare and 2+ cells in both eyes. The vitreous cleared and the fundus lesions were darkly pigmented (Fig. 9). No vitiligo or poliosis appeared.

Discussion

These cases illustrate the protean manifestations of Vogt-Koyanagi-Harada syndrome. There are no firm criteria for the diagnosis of the syndrome with its diverse clinical presentations. Vogt,[6] in 1906, reported a case of uveitis of undetermined origin associated with poliosis and dysacousia. In 1929, Koyanagi[7] reviewed the published reports and found 16 cases of idiopathic uveitis, dysacousia, vitiligo, poliosis, and alopecia, associated with a prodromal phase of fever, headaches, and confusion. Harada,[8] described idiopathic posterior uveitis with bullous retinal detachment in 1926. Yanes and Farrer[9] and Cowper[3] later stressed the marked clinical overlapping in these two syndromes.

Eyes from patients with Vogt-Koyanagi syndrome and those from patients with Harada's disease have similar histopathologic features. There is diffuse bilateral granulomatous uveitis without sparing of the choriocapillaris. There are focal areas of chorioretinal scarring with marked retinal pigment epithelial reaction.[10] However, Perry and Font,[11] in their histopathologic review of nine cases of severe Vogt-Koyanagi-Harada syndrome, found that uveitis in Harada's disease tended to be mild, diffuse, and nongranulomatous. Most of these patients, however, had received corticosteroid therapy at the time of enucleation, which may have reduced the number of epithelioid cells seen on histopathologic sections.[12]

Shimizu[13] has described the characteris-

tic fluorescein findings of Vogt-Koyanagi-Harada syndrome. Usually, central nervous system symptoms precede the ocular manifestations (meningeal phase). The opthalmic phase includes the following: (1) Bilateral retinal detachments which begin in the macular area. The abnormalities spread to involve the peripapillary area and then the periphery. There may be many bullous elevations, usually involving the peripapillary region. (2) Hyperemia of the optic disk with subretinal fluid in the peripapillary area. (3) Inflammatory signs in the vitreous which are minimal during the first few days but which tend to progress. (4) Depigmentation of the retinal pigment epithelium and choroid, giving the characteristic "sunset glow fundus." Ocular and systemic signs of depigmentation are late; the active disease must persist at least three weeks. Pigment changes of the skin, hair, and eyelashes occur in a minority of patients, and may be prevented by early high-dose corticosteroid therapy.[14] Both our patients had most, but not all, of the clinical criteria.

Fluorescein angiographic characteristics, as described by Shimizu,[13] include numerous discrete dots of fluorescence at the level of the retinal pigment epithelium, showing a tendency to enlarge in later sequences and increased permeability of the optic disk capillaries, but normal retinal vessels. These fluorescein angiographic findings suggest that the retinal pigment epithelium-Bruch's membrane complex plays an important role in the Vogt-Koyanagi-Harada syndrome, with leakage of the choroidal fluid into the subretinal pigment epithelial space. There, the retinal pigment epithelial cells are no longer intact; histopathologic sections show a marked pigment epitheliopathy, with disorganization and destruction of cells. The cause of these changes remains obscure, but they may be initiated by a viral infection.

In addition to the sketchy evidence for a viral origin of Vogt-Koyanagi-Harada syndrome,[15-17] there have been several cases reported in which the clinical or fluorescein characteristics of Vogt-Koyanagi-Harada syndrome and acute posterior multifocal placoid pigment epitheliopathy have both been seen.[18-20] Acute posterior multifocal placoid pigment epitheliopathy is thought to have a viral prodromal phase, and may be viral in nature.[21-24]

In their series of 26 patients with acute posterior multifocal placoid pigment epitheliopathy, Wright and Bird[19] found that the patients could be divided into two groups on the basis of clinical findings. The first group had severe acute bilateral disease affecting the retinal pigment epithelium, extensive serous retinal detachments, and other symptoms of Harada's disease. These patients may have had choroiditis, but early fluorescein angiograms were identical to the fluorescein findings in acute posterior multifocal placoid pigment epitheliopathy. The second group had bilateral visual loss, with cream-colored lesions at the level of the retinal pigment epithelium. The fluorescein angiographic pattern was consistent with Harada's disease. These patients, however, had smaller serous retinal detachments, and the other features of Vogt-Koyanagi-Harada syndrome (alopecia, poliosis, vitiligo, tinnitus) were not present. Spontaneous reattachment of the retina usually occurred within one week, and there were no recurrences. This group is similar to our Case 2. One patient described by Wright and Bird had a serous retinal detachment in only one eye but lesions consistent with acute posterior multifocal placoid pigment epitheliopathy in both eyes.

On the basis of clinical and fluorescein angiographic studies and its association with vasculitides, such as erythema nodosum and episcleritis, several authors

believe that acute posterior multifocal placoid pigment epitheliopathy is primarily a vasculitis of the choriocapillaris, with the pigment epithelium secondarily involved.[25,26] Diffuse choroiditis may also be present.

Because of these diverse clinical presentations, Reed[27] proposed that the Vogt-Koyanagi-Harada syndrome be renamed uveomeningoencephalitic syndrome. Others[1,2,11] have repeated this recommendation. There is additional evidence for reclassification of this syndrome.

Many patients with Vogt-Koyanagi-Harada syndrome have had neurologic symptoms and signs.[1-3,5] These usually include cranial nerve palsies (particularly of the III, IV, V, and VIII nerves). Hemiparesis, hemiplegia, dysarthria, and aphasia have been described. Mental changes ranging from mild confusion to psychosis may occur. Many of these findings can be attributed to adhesions of the meninges in certain areas of the brain and spinal cord. Our two unusual cases suggest another origin, based on previous experimental evidence from neurologic and ophthalmic reports.

Acute transverse myelitis is a recognized sequel of some exanthematous diseases and other viral infections.[28-30] Previous viral illness occurs in 21% to 44% of cases of acute transverse myelitis.[28] Transverse lesions have also been described in patients with multiple sclerosis, Devic's disease, acute disseminated encephalomyelitis, idiopathic polyradiculitis, Landry-Gullain-Barré syndrome,[30] systemic lupus erythematosus, polyarteritis, and mixed connective tissue disease.[31] Acute transverse myelitis may be a consequence of a postinfectious, cell-mediated autoimmune (demyelinating) disorder. Acute transverse myelitis may be a response to a component of neural tissue released during the initial infection. Vogt-Koyanagi-Harada syndrome may have a similar origin.[32] Several investigators have experimentally determined the response to specific neural antigens in patients affected with Vogt-Koyanagi-Harada syndrome or acute transverse myelitis. Lymphocyte transformation tests were performed and migration inhibition factor measured by separate groups in cells of patients afflicted with either Vogt-Koyanagi-Harada syndrome or acute transverse myelitis.[30-35]

Abramsky and Teitelbaum[30] found that lymphocytes from 70% of patients with acute transverse myelitis showed lymphoblastic transformation when incubated with myelin basic protein as antigen. The 30% who did not respond were all receiving high doses of corticosteroid at the time of the study. They also obtained a positive response in patients with multiple sclerosis. Cells from patients with postinfectious encephalomyelitis show lymphocyte transformation to human myelin basic protein.[36]

Manor, Livini, and Cohen[32] found migration inhibition in four patients with Vogt-Koyanagi-Harada syndrome. After corticosteroid therapy, two patients had negative responses. Patients who had had the active syndrome four years earlier, in whom the disease was quiescent, and patients with uveitis from other causes also had negative responses.

Our Case 2 illustrated another possible manifestation of this postinfectious cell-mediated autoimmune disorder. The lack of pupillary response to light or near and decreased accommodation and corneal sensation suggested inflammation in or about the ciliary ganglion. These changes may have resulted from diffuse ocular inflammation, but we would not have expected denervation supersensitivity (as shown by constriction with 0.16% pilocarpine) in such severe inflammation. Isolated internal ophthalmoplegia has been reported in acute ciliary ganglionitis in herpes zoster, chicken pox, measles,

diphtheria, syphilis, influenza, scarlet fever, smallpox, pertussis, and sinusitis. Trigeminal ganglion involvement is shown by retro-orbital pain.

The long ciliary nerves contain sensory fibers leading to the cornea, iris, and ciliary body. Some sensory and sympathetic fibers pass through the ganglion without interruption. The preganglionic fibers are myelinated, but the postganglionic fibers here are only lightly myelinated.[37] The long sensory root of the nasociliary nerve contains sensory fibers leading to the cornea, iris, and ciliary body while the short (motor) root contains parasympathetic fibers leading to the iris sphincter and ciliary muscles. A lesion in this area would produce the constellation of signs seen in our Case 2.

A lesion in the midbrain could have produced bilateral internal ophthalmoplegia, but other signs of midbrain dysfunction would have been expected. The presence of denervation supersensitivity also made any intracranial lesion an unlikely cause of our patient's iridoplegia and cycloplegia.

Levy, Kramer, and de Barros[38] reported another case of internal ophthalmoplegia with decreased corneal sensation in a 24-year-old woman with Vogt-Koyanagi-Harada syndrome. The pupils constricted with 2.5% methacholine and 0.002% physostigmine salicylate. They concluded, from their pharmacologic testing and pupillographic tracings, that there was denervation supersensitivity of the pupillary sphincter.

Both of our patients had uveitis associated with inflammation, demyelination, or both. In experimental animal studies, uveitis is an indication of demyelinating diseases in which inflammatory cells (sensitized lymphocytes) appear to initiate the breakdown of myelin. These include acute disseminated encephalomyelitis, experimental allergic encephalomyelitis, and experimental allergic neuritis. Landry-Guillain-Barré syndrome is a disease of humans in this category. Human basic myelin protein, when mixed with complete Freund's adjuvant and injected into the cerebrospinal fluid of experimental animals, induces experimental allergic encephalomyelitis[39] associated with uveitis.

However, Howe and associates[40] produced uveitis and demyelination with both brain-specific protein (myelin basic protein) and "indifferent" protein. (They used purified protein derivative.) These authors concluded that any protein which gains access to the central nervous system may be capable of producing an antibody response resulting in demyelination. In our Case 2, the postganglionic parasympathetic fibers from the ciliary ganglion were unusual in being myelinated.

Cerebrospinal fluid from individuals with Vogt-Koyanagi-Harada syndrome produces changes in the brain and optic nerve when it is injected into the cerebrospinal fluid of rabbits. Crushed brain extracts injected into the eye produce bilateral ocular inflammation and optic neuritis.[1]

These studies suggest that the Vogt-Koyanagi-Harada syndrome is a cell-mediated autoimmune disorder in which a component of myelin acts as an inciting antigen. The clinical courses of Vogt-Koyanagi-Harada syndrome and transverse myelitis suggest a viral or post-infectious origin usually following an influenza-like episode with fever, malaise, headaches, and stiff neck. Morris and Schlaegel[15] found virus-like inclusion bodies in the subretinal fluid of a patient with Vogt-Koyanagi-Harada syndrome, and an untyped virus was found in another case of Harada's disease.[16] No virus has ever been cultured from the cerebrospinal fluid, but viral particles have been identified in hypothalmic sections in cases of encephalitic involvement.[1] Similar lesions of the supraoptic nuclei are

said to affect pigmentation of the hair and skin and cause sensory deafness.[41]

The Vogt-Koyanagi-Harada syndrome may be one of the inflammatory diseases of the uveal tract, autoimmune in nature, ranging from sympathetic ophthalmia to acute posterior multifocal placoid pigment epitheliopathy. Reclassification and redefinition of these conditions may require further immunologic testing of patients and pathologic study of specimens.

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SYMPATHETIC OPHTHALMIA AFTER PARS PLANA VITRECTOMY-LENSECTOMY FOR ENDOGENOUS BACTERIAL ENDOPHTHALMITIS

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A 71-year-old woman developed an endogenous bacterial endophthalmitis in her right eye. Because of the rapid progression she was treated by pars plana vitrectomy-lensectomy and with antibiotics and corticosteroids. The patient improved after the antibiotic therapy. One month later she had a recurrence of the inflammatory signs in the same eye. Examination disclosed lens remnants in the vitreous. Because of the possibility of phacoallergic uveitis, she underwent a second vitrectomy. The corticosteroid dosage was increased. She did well until three months later when, while still taking corticosteroids, she noted loss of visual acuity in the left eye, accompanied by bilateral inflammatory signs. Sympathetic ophthalmia was suspected and the blind right eye was enucleated. The choroid was grossly unremarkable; the routine sections disclosed many inconspicuous foci of mononuclear infiltration. Tangential sections of flat-embedded choroid, however, showed ill-defined nests of epithelioid cells containing phagocytosed melanin, supporting the diagnosis of sympathetic ophthalmia. To our knowledge, this is the first published report of this complication after pars plana vitrectomy-lensectomy in the absence of nonsurgical trauma or other surgical procedures.

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Although several complications are known to occur after vitrectomy,[1] the development of sympathetic ophthalmia appears to be extremely rare; to our knowledge, only six cases have been reported.[2,3] In each of these cases, however, accidental trauma or surgical procedures, or both, might have been responsible for the development of this complication.

We describe herein a case involving a

patient with endogenous endophthalmitis who underwent a pars plana vitrectomy-lensectomy procedure and subsequently developed sympathetic ophthalmia despite concurrent treatment with systemic corticosteroids.

CASE REPORT

A 71-year-old woman was examined on Feb. 13, 1978, because of decreased visual acuity and low-grade fever of one week's duration. Her medical history was noncontributory and there was no history of trauma. The right eye was normal by external and slit-lamp examinations; however, ophthalmoscopic examination disclosed arterial sheathing. The left eye was normal except for a minor nuclear sclerosis. Visual acuity was R.E.: 6/18 (20/60) and L.E.: 6/12 (20/40).

During the next 24 hours, the patient's visual acuity in the right eye decreased to light perception. Examination of the right eye disclosed folds (3+) in Descemet's membrane, 4+ cells and flare in the anterior chamber, and a small hypopyon. The lens had become opaque and the retina and vitreous could not be seen. Intraocular pressure by applanation tonometry was R.E.: 6 mm Hg and L.E.: 16 mm Hg. Because of the strong clinical impression of endogenous bacterial endophthalmitis and in view of its rapid progression, a vitrectomy-lensectomy via the pars plana was performed on Feb. 15, 1978, with the Douvas Rotor-vitrector. The patient received parenteral and retrobulbar therapy with oxacillin and gentamicin and systemic corticosteroid therapy (60 mg of prednisone a day). Culture of the vitreous produced α-hemolytic *Streptococcus* D while the culture from the blood was negative. We were unable to identify the primary focus of infection despite consultation with other services, including infectious disease and cardiology. The patient showed gradual improvement and, after completion of the parenteral therapy, she was discharged. She continued to take oral antibiotics. The media were not clear enough to permit visualization of the posterior pole.

The patient did well until March 27, 1978, when she returned complaining of swelling and pain in the right eye. Examination of the right eye disclosed moderate conjunctival injection and chemosis. Corneal edema, 3+ cells and flare in the anterior chamber, and a small hypopyon were present. The posterior pole was not visible. Visual acuity in the right eye was hand movements at 2 feet. The asymptomatic left eye remained unchanged.

A physical examination disclosed a grade II/IV high-pitched, blowing diastolic murmur at the left sternal border, which had not been present during the initial hospitalization. The clinical diagnosis was subacute bacterial endocarditis with secondary (recurrent) endophthalmitis. Cultures of the blood were negative. The patient was treated with parenteral, topical, and subconjunctival antibiotics (gentamicin and methicillin) and systemic corticosteroids. As posterior segment details became visible, we found that lens fragments were obscuring the optic disk.

Two weeks later, an attempt to taper the dosage of systemic corticosteroids led to a dramatic recurrence of cells and flare, hypopyon, and loss of red reflex. The possibility of phacoallergic uveitis was considered and corticosteroid therapy was reinstituted. After completing the course of antibiotic treatment, the patient underwent a second vitrectomy for removal of the lens fragments, performed through the previous pars plana wound. Histologic examination of the extracted material disclosed irregular fragments of lens without an associated inflammatory reaction. Postoperatively, the patient did well and the intraocular inflammation decreased. Blood cultures after antibiotic therapy was completed were negative. The patient was discharged on May 3, 1978.

Three months later the patient, still taking 60 mg of prednisone every other day, complained of irritation and decreased visual acuity in the left eye. Examination disclosed slight conjunctival injection and chemosis in both eyes. Slit-lamp examination of the right eye demonstrated 2+ flare and cells in the anterior chamber. The left eye showed a few cells and slight flare in the anterior chamber. The lens capsule was intact, but there were cataractous changes that obscured the fundus. The right eye had no light perception, and visual acuity was reduced to 6/21 (20/70) in the left eye. Sympathetic ophthalmia was suspected and enucleation of the blind right eye was performed for diagnostic purposes. The patient's dosage of systemic corticosteroid was increased to 60 mg of prednisone daily; this led to resolution of the intraocular inflammatory signs in the left eye.

During a seven-month follow-up period, attempts to discontinue the corticosteroid treatment were accompanied by recurrence of cells, flare, and keratic precipitates in the anterior chamber. Almost two years after the onset of symptoms, the patient required 20 mg of prednisone every other day to control the ocular inflammation.

Histologic study of the aphakic eye disclosed a total, funnel-shaped retinal detachment. Although there was no gross thickening of the choroid (Fig. 1), microscopic examination disclosed many foci of lymphocytic infiltration and an associated mild eosinophilia (Fig. 2). Tangential sections of flat-embedded choroid showed ill-defined nests of epithelioid cells within some of the lymphocytic infiltrates (Fig. 3). These epithelioid cells contained phagocytosed melanin. Special stains for bacteria, fungi, and acid-fast organisms were negative.

DISCUSSION

Sympathetic ophthalmia is classically characterized as a bilateral granulomatous panuveitis occurring after a penetrating injury to one eye. In patients

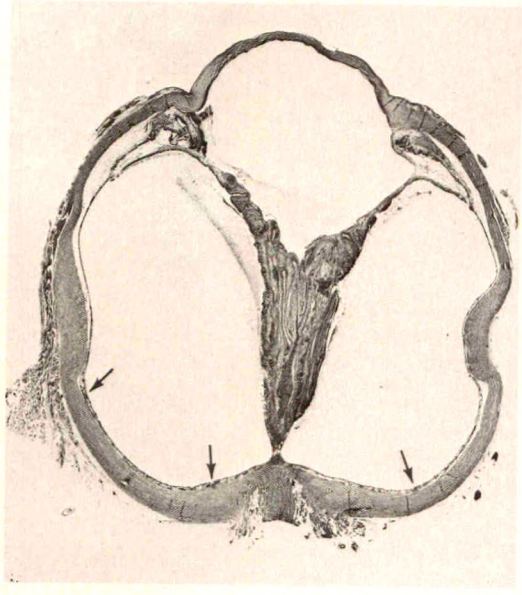


Fig. 1 (Croxatto and associates). Low-power view of the enucleated aphakic eye showing normal thickness of the choroid except for inconspicuous foci of infiltration by inflammatory cells (arrows) (hematoxylin and eosin, ×4).

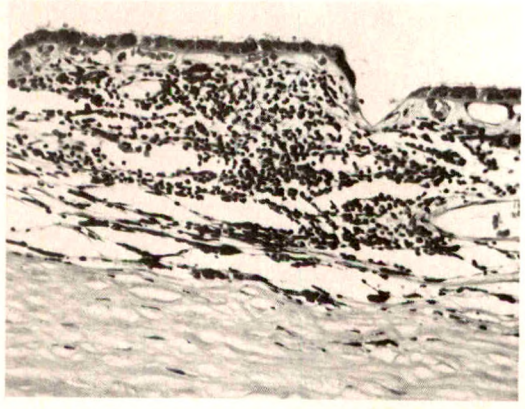


Fig. 2 (Croxatto and associates). Mild thickening of the choroid by a lymphocytic infiltrate that spares the choriocapillaris (hematoxylin and eosin, ×252).

receiving corticosteroid treatment for unrelated conditions, this disease may appear as a very mild inflammation that is difficult to detect or interpret.[4] In such cases, sympathetic ophthalmia may be unsuspected, and diagnosed only after enucleation of the injured eye. There is evidence that corticosteroid therapy modifies the inflammatory reaction and, consequently, the histopathologic pattern.[5] In the present case, gross examination disclosed no evidence of sympathetic ophthalmia, and routine sections of the enucleated eye demonstrated inconspicuous but numerous foci of mononuclear infiltration with mild eosinophilia in the choroid and very early involvement of the scleral canals, but no epithelioid cells. Thus, the histologic findings, although

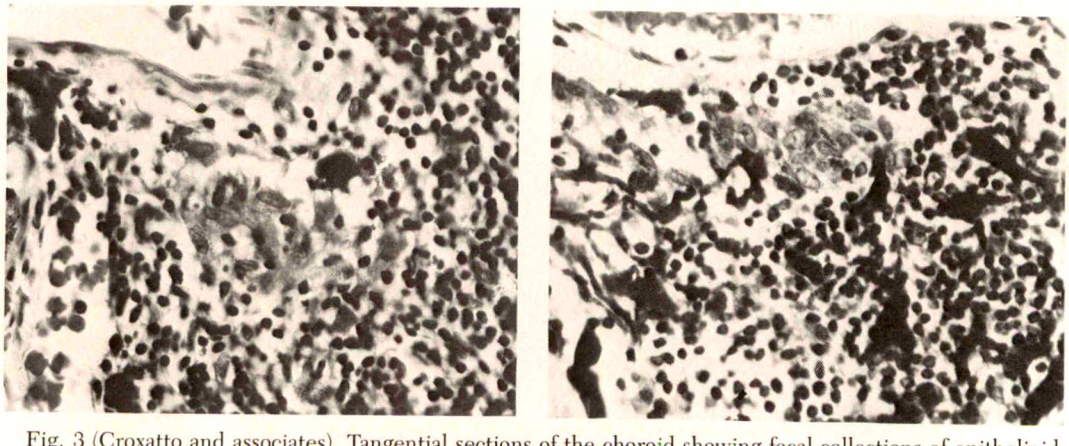


Fig. 3 (Croxatto and associates). Tangential sections of the choroid showing focal collections of epithelioid cells containing phagocytosed pigment granules (hematoxylin and eosin, ×630).

suggestive of sympathetic ophthalmia, were not totally convincing. Additional sections, however, prepared from flat-embedded choroid clearly demonstrated ill-defined nests of epithelioid cells, and provided support for the diagnosis of sympathetic ophthalmia. Although a nongranulomatous form of sympathetic ophthalmia may occur,[6] especially in very early cases or in patients treated with large doses of corticosteroids, the classical criteria for the histopathologic diagnosis include the presence of epithelioid cells.[7]

Lewis, Gass, and Spencer[2] first described five cases of sympathetic ophthalmia after vitrectomy. In these cases, as well as in two other cases on file in the Registry of Ophthalmic Pathology, vitrectomy was performed in patients who had previously sustained accidental or surgical trauma. Recently, Puliafito and associates[3] described another patient who developed sympathetic ophthalmia after vitrectomy but who also underwent a scleral buckling procedure and an intracapsular cataract extraction. Therefore, sympathetic ophthalmia could not be attributed unequivocally to vitrectomy in those cases.

In our case, the patient had undergone two vitrectomy-lensectomy procedures and she had also had recurrent episodes of unilateral endogenous bacterial endophthalmitis. One may question the pathogenic role of the previous bacterial infection. Although several cases of sympathetic ophthalmia and suppurative endophthalmitis in an injured eye have been reported,[8] this appears to be an unrelated, coincidental association; it is unlikely that the endophthalmitis plays a facilitating role in these cases. Indeed, the opposite view was held in the days before the introduction of antibiotic therapy.[9] Thus the present case shows that vitrectomy alone may induce or initiate the development of sympathetic ophthalmia. At a recent meeting of the Verhoeff Society (Washington, D.C., April 24-25, 1980) W. H. Spencer described two other cases in which sympathetic ophthalmia followed pars plana vitrectomy-lensectomy; in both cases there was no history of accidental or surgical trauma. In none of these three cases was incarcerated uveal tissue found in the surgical wound.

The risk of sympathetic ophthalmia after pars plana vitrectomy may not differ greatly from that associated with other surgical procedures, which seems to be about 0.007%.[10] However, with the rapid increase in the use of pars plana vitrectomy we may expect to see this complication more frequently. A recent survey of 23 departments of ophthalmic pathology showed that ten of the 48 histologically confirmed cases of sympathetic ophthalmia that developed in the last five years occurred after vitrectomy (J. D. M. Gass, Verhoeff Society, Washington, D.C., April 24-25, 1980). This emphasizes the possibility of increased risk of sympathetic ophthalmia in patients who undergo pars plana vitrectomy, particularly when this technique is used to restore vision or to save the injured eye after trauma or complicated surgical procedures.

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Ophthalmic miniature

None of us think enough of these things on which the eye rests. But don't let us let the eye rest. Why should the eye be so lazy? Let us exercise the eye until it learns to see the startling facts that run across the landscape as plain as a painted fence. Let us be ocular athletes.

G. K. Chesterton, *Tremendous Trifles*,
New York, Sheed and Ward, 1955

OCULOCUTANEOUS ALBINISM ASSOCIATED WITH CORNEAL MESODERMAL DYSGENESIS

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A 48-year-old woman had tyrosine-negative oculocutaneous albinism and corneal mesodermal dysgenesis of the Axenfeld type. Results of chromosome analysis were normal, and no other family members showed the association of these two conditions. The rarity with which the association has been reported suggests that this combination of findings is probably coincidental.

Generalized oculocutaneous albinism is an autosomal recessive hereditary condition that may be associated with numerous ocular anomalies. François[1] listed atypical coloboma, incomplete aniridia, persistent pupillary membrane, corectopia, polycoria, microphthalmia, optic atrophy, Leber's disease, capsular cataracts, and external ophthalmoplegia. The absence of an ophthalmoscopically or histologically identifiable fovea is a well-recognized abnormality of the albino visual system.[2-5]

Whereas the iris in generalized albinism may show various degrees of hypoplasia, ranging from pseudopolycoria to aniridia, cleavage anomalies of the anterior chamber have not been found to be associated with albinism. The anterior chamber cleavage syndrome[6] may be associated with numerous anomalies of the anterior eye (including atrophy of the iris, aniridia, heterochromia, corectopia, polycoria, ectropion uveae, flat cornea, microcornea, coloboma of the iris and choroid, and corneal opacities[1]), but it is generally not associated with systemic abnormalities.

I examined a woman with oculocutaneous albinism and mesodermal dysgenesis of the Axenfeld type.

CASE REPORT

The patient was a 48-year-old white woman with oculocutaneous albinism. Her family pedigree is shown in Figure 1. Her hair, eyebrows, and eyelashes were all white. Her skin was pale and easily burned by exposure to the sun. Her skin contained no pigmented nevi or freckles. She had had nystagmus and photophobia since birth. Uncorrected visual acuity had been 6/60 (20/200) in both eyes for many years. With telescopic high-plus lenses, she was able to meet the 6/15 (20/50) visual standard necessary to obtain a driver's license. She read J5. Color vision was normal by the Farnsworth-Munsell 100-hue test.

Slit-lamp biomicroscopy disclosed a light, diaphanous blue iris with hypoplastic stroma and absent

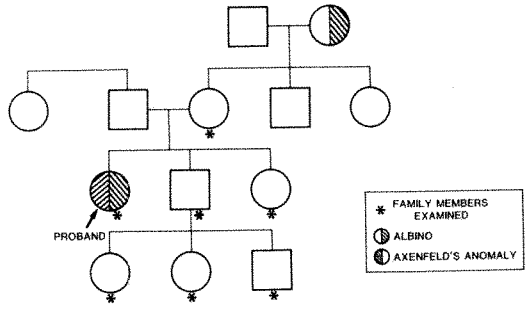


Fig. 1 (Lubin). Family pedigree of the patient described. No other member of the family showed the combination of findings. Circles, females; squares, males.

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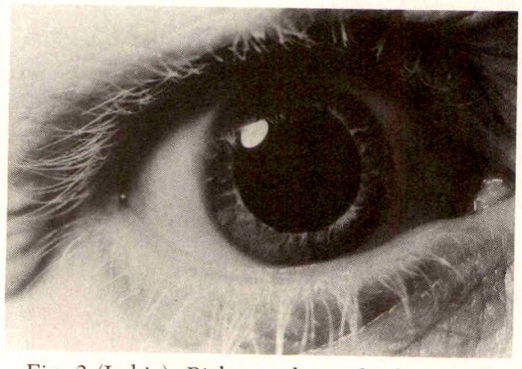


Fig. 2 (Lubin). Right eye has a diaphanous blue iris. Iris strands insert onto the posterior surface of the cornea. Note the pure white eyelashes and pale skin.

pigment in the epithelial layers. The iris sphincter was easily seen. The iris transilluminated bright orange with retroillumination from a slit-lamp beam. The corneas were clear, with prominent Schwalbe's lines peripherally and many fine iris strands traversing the chamber angle to insert on Schwalbe's ring (Fig. 2). Gonioscopy showed that these strands were present for 360 degrees around in the chamber angle. Applanation tension was 18 mm Hg in both eyes. There was no central corneal or lenticular opacity or defect.

Extraocular movements were full, with a high-amplitude, low-frequency pendular nystagmus. A small latent component was present when one eye was occluded. She was grossly orthophoric. The pupils were equal in size and reactive to light; there was no afferent pupillary defect.

Ophthalmoscopic examination with the pupils dilated showed a striking lack of pigmentation and prominent choroidal vessels. The background color was yellow-white (Fig. 3, left), except in the macular area where it was a flat yellow-orange without underlying vessels (Fig. 3, right). The foveal reflex was absent bilaterally.

Hairbulbs from the patient were plucked and incubated at 37 C with 80 mg of tyrosine in 100 ml 0.1N phosphate buffer (pH 6.8) by the method of Kugelman and Van Scott.[7] The bulbs did not darken, and, therefore, the patient was tyrosine-negative.

Chromosome analysis disclosed the full complement of chromosomes without deletion or translocation of any segments.

Discussion

There have been only two reports of oculocutaneous albinism associated with mesodermal dysgenesis.[8,9] Valvo, Romei, and Morin[8] described a 5-year-old boy with albinism who had an anterior segment dysplasia of unclear type in the right eye only. Results of a chromosome analysis were normal. Hales[9] described a 20-year-old man with the clinical features of generalized albinism who had had poor vision and nystagmus since childhood. There were anterior chamber anomalies consistent with Axenfeld's anomaly. Hales concluded that the two findings were probably coincidental.

There has been one other report of partial (ocular) albinism, neurofibromatosis, congenital deafness, and peroneal

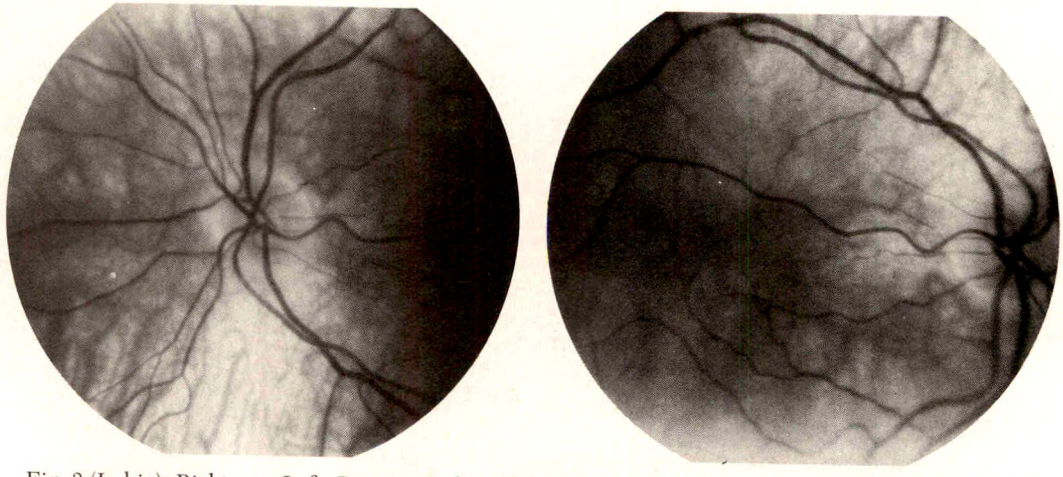


Fig. 3 (Lubin). Right eye. Left, Prominent choroidal pattern and lack of pigmentation. Right, Macular area lacks a foveal pit and yellow foveal reflex.

muscular atrophy.[10] Axenfeld's anomaly was found in two siblings in this family. The authors drew no conclusions as to the association between ocular albinism and the anterior chamber anomaly.

In my case, labeling the anterior chamber anomaly was difficult. In the stepladder classification of Waring, Rodrigues, and Laibson,[11] Axenfeld's anomaly consists of a prominent Schwalbe's line (posterior embryotoxon) and iris strands spanning the chamber angle to insert on Schwalbe's ring. The next step up the ladder is Reiger's anomaly, which adds hypoplasia of the anterior iris stroma. These authors[11] stated that Reiger's anomaly is autosomal dominant in 70% of cases, with 95% penetrance, and that glaucoma occurs in 60% of these patients. Because no other family members were affected, because there was no evidence of glaucoma, and because the iris of albinos may appear to be hypoplastic, the features in this case probably represented Axenfeld's defect rather than Reiger's.

This case raised several questions about the association of albinism and corneal mesodermal dysgenesis. Chromosomal analysis of this patient and others[8] did not support a chromosomal abnormality as the basis of such a combination. We know that oculocutaneous (tyrosine-negative) albinism results from an enzyme deficiency in which there is no functioning tyrosinase available for the hydroxylation of tyrosine to dihydroxyphenylalanine (dopa), and the oxidation of dopa to dopaquinone, with both steps catalyzed by the deficient enzyme.[12,13] The genetic loci for albinism (tyrosine deficiency) and corneal mesodermal dysgenesis are probably not close because of the rarity with which these conditions are associated.

Does the lack of pigment cause retardation in the movement of cells during normal cleavage in the chamber angle? Again, these two conditions are rarely associated. Margolis and associates[14] described nine patients with Apert's syndrome, five of whom showed an associated hypopigmentation of the hair, skin, and eyes. Spotty distribution of the hypopigmentation, the relatively normal visual acuity, and the lack of nystagmus led these authors to conclude that an insult in early embryonic life may cause associated ocular and skeletal abnormalities. During the 42nd to the 49th day of gestational life (the 12- to 17-mm stage) disturbance of migration of melanoblasts in the sensory crest to peripheral sites occurs at the same time as limb differentiation. The same would not be true in this case because the defects noted in albinism occur at the 7-mm stage (the end of the fourth week of gestation), whereas the delay in development which causes Axenfeld's anomaly begins at the 18-mm stage (eighth week); cleavage begins during the seventh month.[6,15] Normal cleavage of the anterior chamber is probably not affected by pigment.

Lack of pigment in the eye can, however, cause secondary abnormalities in development. Creel, O'Donnell, and Witcop[16] discussed the potential importance of normal pigmentation of the optic cup in the embryologic development of retinogeniculate projections. They used visual-evoked potentials to compare responses in humans with albinism and controls. They found misrouted optic fibers, an excessive number of which were erroneously crossed at the optic chiasm, terminating in a portion of the dorsal lateral geniculate nucleus normally reserved for uncrossed fibers. There was a dramatic difference in monocularly stimulated humans with albinism compared with the normally pigmented counterparts. Because these anomalous projections occur in various forms of oculocutaneous albinism, the authors concluded that the lack of pigment in early development was the cause.

The rarity with which the association of corneal mesodermal dysgenesis and oculocutaneous albinism has been reported suggests that this association is coincidental. However, the defect in development of the macula has not been explained, nor has the mechanism of production of the various associated anomalies mentioned earlier.[1] Perhaps there is a generalized retardation of ocular development caused by the lack of pigment or an associated metabolic deficiency.

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EXTERNAL CAROTID-CAVERNOUS FISTULA IN AN INFANT

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A 7-week-old infant developed a nontraumatic fistula of the external carotid artery and the cavernous sinus. Therapeutic embolization produced an uncomplicated clinical and angiographic cure. Treatment was prompted by the availability of new methods of treatment and our hope of preventing irreversible anatomic and visual sequelae.

Spontaneous fistula of the external carotid artery and the cavernous sinus was first described by Castaigne, Laplane, and Djindijian in 1966.[1] Since then, 30 cases of dural-cavernous shunts have been recorded,[2-13] all of which were in patients over 20 years of age. Fistulas in infants are usually posttraumatic and involve the internal carotid artery. Specific indications for the treatment of infants for this condition have not been established. We examined a 7-week-old infant with proptosis of the globe and found a nontraumatic fistulous communication between a branch of the internal maxillary artery and the cavernous sinus.

CASE REPORT

A 7-week-old boy was admitted to the hospital because of proptosis. He was the product of a full-term uncomplicated pregnancy and an uneventful vaginal delivery. Low forceps were used, but there was no excessive cephaledema or molding. Birthweight was 2,834 g.

When he was 1 week old, his left eye became prominent when he cried or strained. Six weeks later, excessive lacrimation and increased vascular markings of the conjunctiva were observed. The proptosis increased and involved the fellow eye.

Physical examination disclosed bilateral intermittent proptosis, which was 4 mm greater on the left side. The eyelids were swollen and the left eye had engorged and tortuous episcleral vessels, conjunctival injection, and mild chemosis (Fig. 1). Mild, nonpulsatile resistance was encountered with gentle retrodisplacement of the globe, but rotation of the globe was not restricted. Ophthalmoscopic examination disclosed engorged retinal veins (Fig. 2). Although intraocular pressure was R.E.: 16 mm Hg and L.E.: 24 mm Hg, there was no cupping of the optic nerve. The cornea was clear and had a horizontal diameter that measured 10.5 mm in each eye. A loud, intermittent systolic bruit was heard over the left globe. Results of the remainder of the physical examination were normal.

Skull roentgenograms were normal. Computed axial tomography showed thickened extraocular muscles and proptosis of the globe. Intravenous enhancement showed a prominence of the left cavernous sinus.

The diagnosis of a left-sided external carotid-cavernous fistula was confirmed by transfemoral cerebral angiography. Selective injection of the left external carotid artery identified the fistulous com-

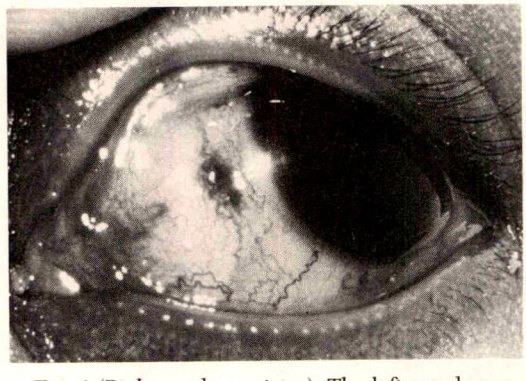


Fig. 1 (Biglan and associates). The left eye demonstrates proptosis, conjunctival injection, and mild chemosis.

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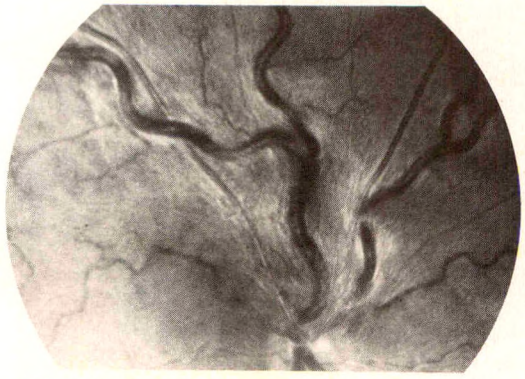


Fig. 2 (Biglan and associates). Pretreatment fundus photograph shows the engorged retinal veins observed in the left eye.

munication between a hypertrophied branch of the internal maxillary artery and the posterolateral compartment of the cavernous sinus (Fig. 3). Angiography of the left internal and right common carotid arteries gave normal results. Because of the persistence of the intraocular hypertension and potential for irreversible globe and orbital changes, therapeutic embolization was performed. We guided two pieces of absorbable gelatin sponge 1 × 1 mm into the distal external carotid artery by the flow control technique. The sponge occluded the main feeding vessel near its origin, obliterating the communication with the cavernous sinus. Proptosis regressed within hours and conjunctival injection improved. The next day, intraocular pressure was R.E.: 14 mm Hg and L.E.: 18 mm Hg. Cutaneous and cerebral circulation was well preserved.

Five weeks after embolization, we re-examined the patient with angiography. The fistula remained closed and the blood flow to the cerebrum was normal. The patient's appearance and retinal vasculature were normal. Intraocular pressure was R.E.: 14 mm Hg. and L.E.: 17 mm Hg. The bruit we had noted previously was absent.

Discussion

The patient described had a dural-cavernous fistula originating from a branch of the external carotid artery. Fistulas involving the cavernous sinus are frequently associated with trauma. The arterial blood supply usually originates from the internal carotid artery, although feeders from the external carotid artery have been reported.[14] The occurrence of spontaneous anomalous vascular fistulas involving the external carotid artery in infants or young children is uncommon, and the natural progression and sequelae of and the guidelines for treatment are poorly defined in this age group. Although the clinical features are similar, differences in the hemodynamic characteristics of the fistulas of the external carotid artery and internal carotid artery warrant separating these two fistulas into two distinct conditions.

Logical treatment plans evolve from an understanding of the vascular anatomy and its variations. The intracranial dura receives its blood supply from the internal carotid, the external carotid, and the vertebral arteries. Abnormal communication with the cavernous sinus may be supplied by any combination of these sources. The relationship of the afferent vessels has been clarified by a study of 35 patients with dural arteriovenous malformations.[15] The external carotid artery was involved in 89% of the patients, the internal carotid artery in 30%, and the vertebral artery in 6%. In malformations involving the cavernous sinus alone, however, only three of 11 patients had feeding vessels arising from the external carotid artery.[10] Supply from the vertebral artery is uncommon.[16] When the main blood supply originates from the external carotid system, the middle meningeal artery is the most frequently involved branch communicating with the venous sinus.[4,14,17-19] In our patient, the anomalous vessel was a grossly hypertrophied accessory meningeal vessel from the internal maxillary artery.

Venous egress from the cavernous sinus depends on the site of the fistulous connection, the position of the head in relation to the heart, and rhythmic changes in the central venous pressure associated with respiration.[2] Drainage into the superior ophthalmic vein is the most common pattern of venous outflow, followed by drainage into the inferior

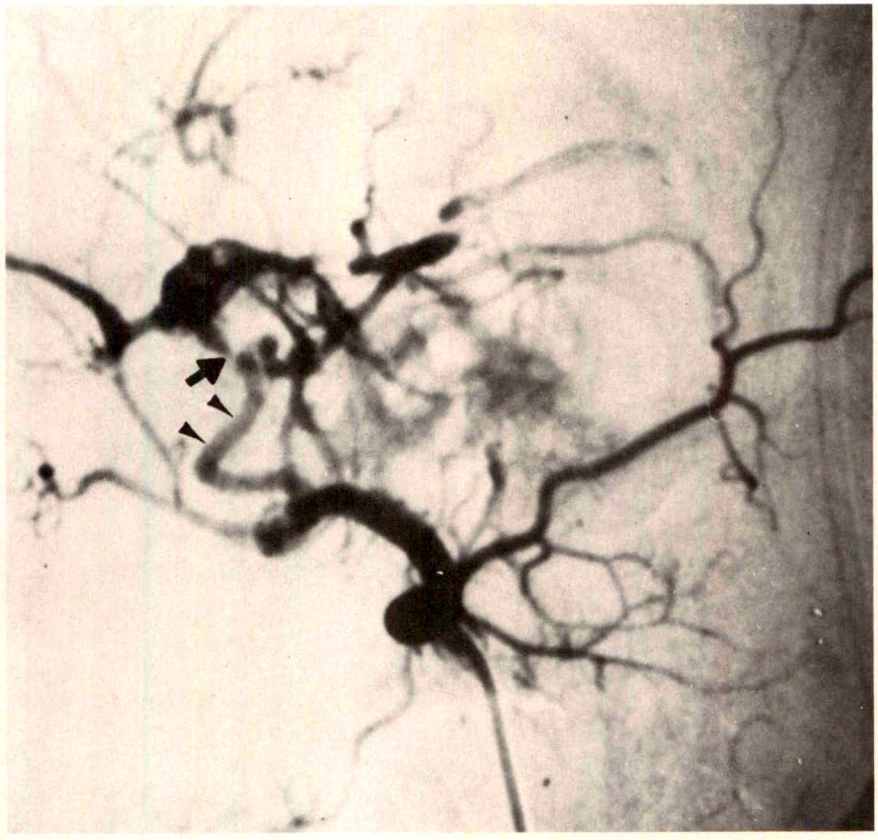


Fig. 3 (Biglan and associates). Selective injection of the left external carotid artery shows the hypertrophied branch of the internal maxillary artery communicating with the posterolateral compartment of the cavernous sinus.

ophthalmic vein, the petrosal sinuses, and the clival plexus.[10]

Although the dural-cavernous fistula and the posttraumatic internal carotid-cavernous fistula have common symptoms, it is important to separate these two types of fistulas because they greatly differ in severity and prognosis. Fistulas involving the internal carotid artery are high-flow, high-pressure shunts and cause markedly increased venous pressure. The dural-cavernous fistulas, conversely, are low-flow, low-pressure shunts because their arteries are thin-walled, small-caliber, and subject to a dampened pressure head. Such a low-flow, low-pressure state can be deduced from angiographic data, even though direct pressure measurements in these dural fistulas are unobtainable.[10] Symptoms caused by dural-cavernous fistulas are less severe and may be transient.[10]

The lower venous pressure in the dural-cavernous fistula is the reason bilateral venous engorgement caused by intracavernous communication is less likely to occur in these shunts than in those originating in the internal carotid artery. Proptosis, therefore, is not severe and is usually unilateral. Because of this flow, bruits occur in only 50% of the patients and are frequently transient.[4,5,10]

Features common to both fistulas include unilateral headache, ipsilateral retro-orbital pain,[4,10] eyelid swelling, and dilated episcleral and conjunctival vessels with chemosis and increased tearing that may be misinterpreted as conjunctivitis, episcleritis, iritis, or glaucoma.

Abducens nerve (VI) palsy is sometimes observed, especially if venous drainage is primarily into the petrosal sinus. In this area, a distended petrosal sinus may compress the abducens nerve as it dips into Dorello's canal.[20] Distended retinal veins, retinal hemorrhages, exudates, and papilledema occasionally occur. Intracranial complications are rare.

Diagnostic studies should include computed axial tomography to exclude defects in the orbital wall. Complete bilateral angiographic studies are needed because an expanding shunt may recruit arterial supply from the contralateral source.[21] Angiography discloses the extent of the fistula and provides helpful information for planning treatment.[3,17]

Angiographically, dural-cavernous fistulas can be divided into two types—those that lie within the walls of the cavernous sinus itself and drain directly into the sinusoids, and those that involve a distant dural sinus that communicates with the cavernous sinus.[10] Communication may be via the petrosal[17,18] or the sphenoparietal[22] sinuses. Newton and Hoyt[10] studied 11 cases; only two patients had shunts belonging to the latter category. Most fistulas empty into the posterior portion of the cavernous sinus, as was the case in our patient.

The indication for treatment of cavernous fistulas is loss of vision. Most authors will agree that cerebral ischemia is rarely a complication unless obliterative surgery on the carotid artery has been performed.[23,24] The pathophysiologic findings are similar to those of internal carotid-cavernous fistulas. Loss of visual acuity may be caused by decreased retinal perfusion resulting from narrowing of the arteriovenous pressure gradient secondary to increased venous pressure.[25,26] In patients with internal carotid-cavernous fistulas, the retinal arterial pressure is also decreased because shunting occurs proximal to the ophthalmic artery.[26] In a purely dural fistula originating from the external carotid artery, however, the decrease in retinal arterial pressure is probably negligible, as is the risk of symptomatic retinal ischemia.

Intractable glaucoma accounts for blindness in some patients with cavernous fistulas.[27] An increase in the back venous pressure from the ciliary veins increases intraocular pressure. Additionally, venous congestion in the filtration angle may mechanically retard aqueous egress.[27] Anterior segment hypoxia can occur and lead to neovascular changes on the peripheral iris, cataract formation, folds in Descemet's membrane, and corneal opacification. Choroidal detachment has been associated with bilateral dural-cavernous fistulas.[28] Exophthalmos and secondary exposure keratitis may also reduce visual acuity. Because of the hemodynamic differences between these two forms of fistulas, the above findings, although present, are diminished in patients with dural-cavernous fistulas.

We have little experience with the visually immature patient with a fistula. Robb[29] and Cuttone and associates[30] have recently explored the relationship between the soft-tissue swelling around the orbit and the production of astigmatic refractive errors. Additionally, the long-term effects of increased intraocular pressure on the globe of an infant are well known. Poor visual outcome is associated with buphthalmos, opacification of the cornea, and large refractive errors associated with glaucoma. These patients may develop refractive as well as strabismic amblyopia.[31]

In adults and visually mature children, periodic monitoring of visual acuity, visual fields, and the degree of proptosis and control of intraocular pressure are considered sufficient management. However, in the infant, the risks of surgical intervention should be weighed against these additional factors, which may irreversibly alter the developing globe, orbit, and visual system. We took these factors into account in treating our patient and recommended surgery.

In the past, a number of therapeutic techniques have been advocated. Extracranial ligation of the external carotid artery has had some success. With this method, Markham[18] and Parkarinen[22] were able to close two dural fistulas involving the petrosal and sphenoparietal sinuses. Mingrino and Moro[8] successfully obliterated a cavernous sinus shunt fed by a single branch of the internal maxillary artery. All three of these fistulas had single feeding vessels. When several feeding vessels are involved, simple external carotid ligation is inadequate.[3] In such cases, a combination of external carotid artery ligation and direct intracranial occlusion of the dural feeders has been tried successfully by Fincher,[17] Ramamurthi and Balasubramanian,[32] and Iraci and Carteri.[33] However, intracranial ligation of deeply situated feeders may be technically difficult.

More recently, embolization has been used as an adjunct or as an alternative to direct vascular obliteration.[34-36] Mahaley and associates[7] successfully treated an external carotid-cavernous fistula by injecting Silastic-coated steel balls up into the external carotid artery. Manaka, Izawa, and Nawata[37] have used silicone to treat a dural fistula. In 1978, Costin and associates[4] reported closing of a dural-cavernous fistula with absorbable gelatin sponge. Absorbable gelatin sponge and polyvinyl alcohol both have a low mass and low inertia and, therefore, are carried easily by the increased flow into the fistula itself. These materials offer control unattainable by older methods.

We treated our patient with absorbable gelatin sponge embolization. Embolization of the aberrant artery produced normal intraocular pressure and caused the proptosis to regress and the changes in the peripapillary and peripheral retina to subside. The low pressure associated with external carotid-cavernous fistulas permits permanent thrombosis with fragile emboli such as this material.

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ORBITAL MELANOCYTIC HAMARTOMA

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A male Chinese neonate had a melanocytic hamartoma that involved the entire orbit and the eye, but did not involve the orbital bones or eyelid skin. Although usually benign, these hamartomas may be locally invasive, and rare cases of malignant transformation have occurred.

Ocular congenital pigmentary disorders with hamartomatous stromal melanocytic involvement include nevus of Ota, nevus of Ito, melanocytoma, and congenital ocular melanosis.[1,2] Usually these lesions occur in hyperpigmented individuals. Only rarely do these hamartomatous lesions become locally invasive or have areas of malignant transformation that can metastasize.

We treated a Chinese neonate with a congenital black orbital tumor that had destroyed the eye but which had not invaded the orbital bones or other intracranial structures.

CASE REPORT

This boy was the product of a normal pregnancy and delivery. At birth the patient was found to have a protruding blue-black orbital mass that had destroyed the right eye. The surface of the mass measured approximately 21 × 16 mm. The lesion involved the entire orbit (including the palpebral and bulbar conjunctiva), but not the eyelid epithelium. It did not pulsate or transilluminate (Fig. 1). There were many darkly pigmented cutaneous papules on other areas of the body (Fig. 2). A large mongolian spot was present over the sacral region. A biopsy of one of these cutaneous papules disclosed that it was histologically characteristic of a blue nevus.

Computed axial tomographic studies of the brain and orbits demonstrated expansion of the right orbit; a mass occupied the entire orbit but there was no bone invasion (Fig. 3). The mass appeared to involve the globe; the right optic canal was slightly enlarged. Results of other laboratory and radiologic examinations, including a computed axial tomographic body scan and cerebrospinal fluid analysis, were within normal limits.

When he was 4 days old, the patient underwent an uncomplicated exenteration of the right orbit. The exenteration healed by primary granulation. At the age of 6 months, the patient showed no evidence of local recurrence. The blue nevi were unchanged in size and appearance.



Fig. 1 (Char and associates). Melanocytic hamartoma of the right eye.

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Fig. 2 (Char and associates). Systemic blue nevi (pigmented papules).

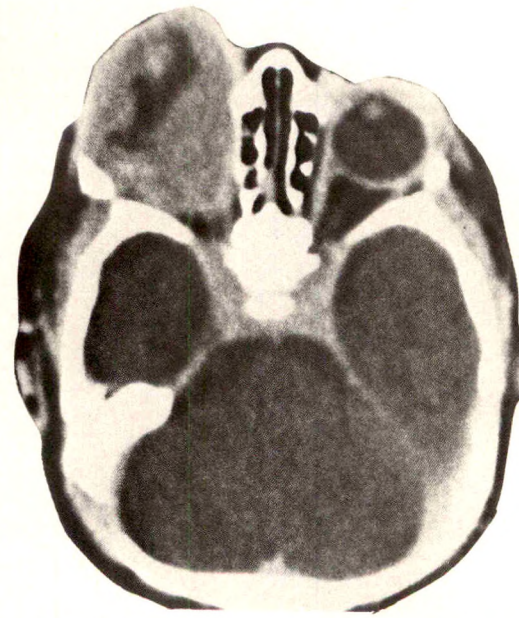


Fig. 3 (Char and associates). Computed axial tomographic section through ocular melanocytic hamartoma.

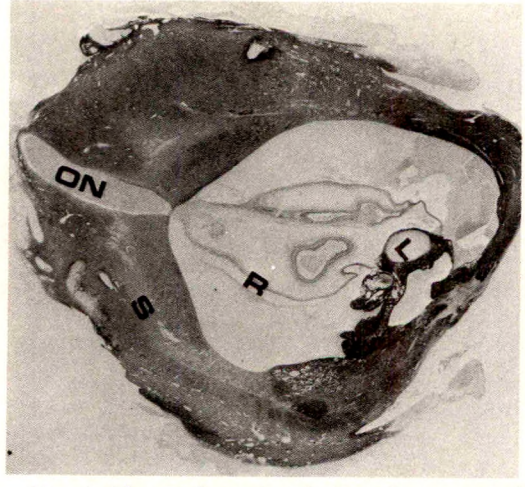


Fig. 4 (Char and associates). Heavily pigmented tumor filling the orbit, surrounding the optic nerve (ON), and involving the cornea and most of the sclera (S) and uveal tract. The lens (L) is almost entirely surrounded by tumor; the retina (R) is detached.

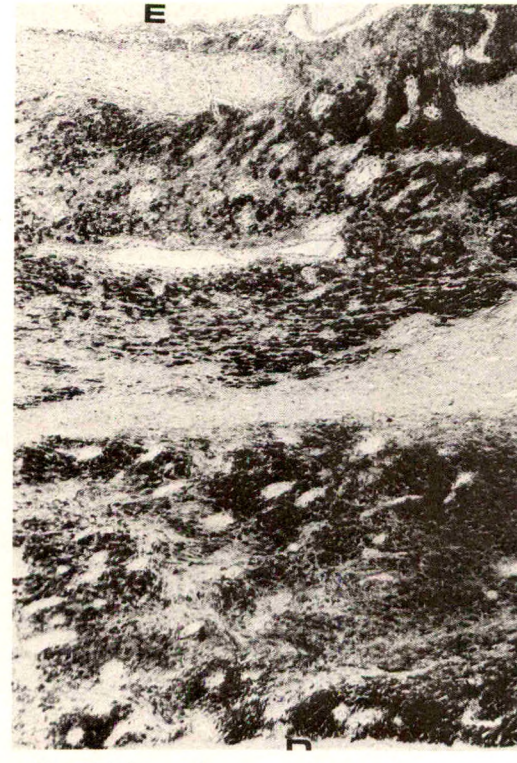


Fig. 5 (Char and associates). Heavily pigmented tumor involving all the layers of the cornea from epithelium (E) to Descemet's membrane (D) (hematoxylin and eosin, ×17).

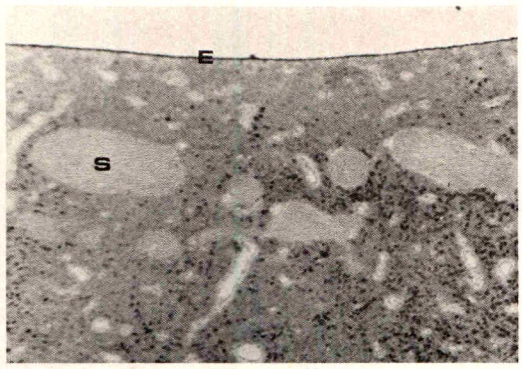


Fig. 6 (Char and associates). Tumor involving uvea and sclera with only remnants of the sclera (S) remaining. Retinal pigment epithelium is visible (E) (hematoxylin and eosin, ×17).

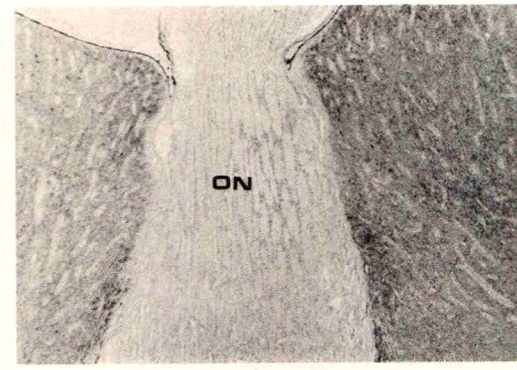


Fig. 7 (Char and associates). Tumor surrounding the optic nerve (ON) (hematoxylin and eosin, ×10).

Histologic findings—The lesion appeared to be a congenital melanocytic hamartoma of the orbit. A heavily pigmented tumor filled the orbit (Fig. 4). The superior and inferior fornices, cornea (Fig. 5), and most of the sclera (Figs. 6 and 7) and uveal tract were involved; however, the tumor did not involve the orbital bones or eyelid epithelium.

The tumor was composed of ovoid melanocytes (Fig. 8), spindle-shaped melanocytes (Fig. 9), and dendritic melanocytes (Fig. 10). Some nuclear pleomorphism was present; there was less than one mitotic figure per 200 40-power microscopic fields.

A pigmented skin lesion not involving the ocular structures was biopsied; histologically it appeared to be a blue nevus (Fig. 11.)

Discussion

Ocular hamartomatous melanocytic proliferations include nevus of Ota (oculodermal melanocytosis or oculocutaneous melanosis), nevus of Ito, melanocytoma, and ocular melanosis. These conditions are closely related to other congenital pigmentary disorders, including blue nevus, cellular blue nevus, mongolian spot, neurocutaneous melanosis syndrome, and melanotic neuroectodermal tumor of infancy (retinal anlage tumor).[3-10] Most ocular melanocytic hamartomas occur in hyperpigmented individuals; our patient was Chinese.[1]

Usually these hamartomas remain localized; however, some become locally invasive, involve regional lymph nodes, or turn into melanomas and metastasize.[10-15]

Orbital involvement is rare in associa-

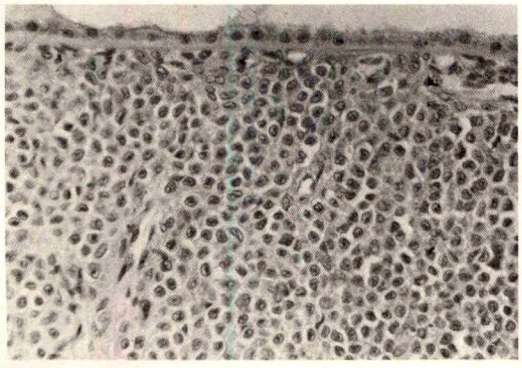


Fig. 8 (Char and associates). Ovoid and cuboidal melanocytes filled the uveal tract (the retinal pigment epithelium is a single layer of cells at top (hematoxylin and eosin, ×100).

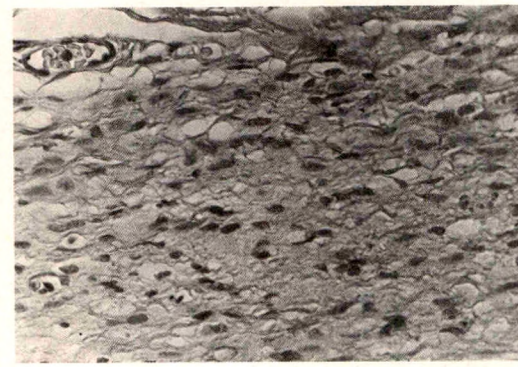


Fig. 9 (Char and associates). Spindle-shaped melanocytes in the orbital portion of the tumor (hematoxylin and eosin, ×150).

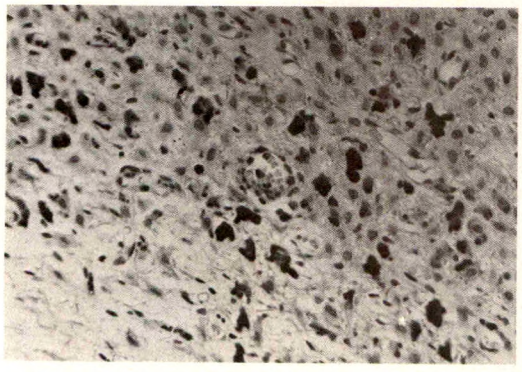


Fig. 10 (Char and associates) Dendritic melanocytes in the orbital portion of the tumor (hematoxylin and eosin, ×100).

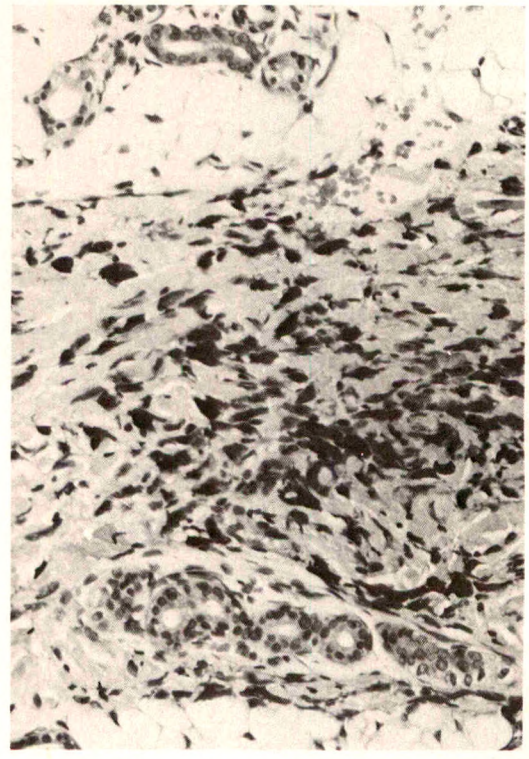


Fig. 11 (Char and associates). Blue nevus of the skin. Elongated melanocytes are present in the middle and lower third of the dermis adjacent to sweat glands and subcutaneous fat (hematoxylin and eosin, ×17).

tion with hamartomatous pigmentary disorders of the ocular structures.[3,4,16-19] A few orbital tumefactions in association with nevus of Ota have been reported. Speakman and Phillips[16] observed two patients with this syndrome who developed orbital tumors in adulthood; one patient had a cellular blue nevus and the second a cellular blue nevus with malignant change and developed metastases. Montgomery[3] reported two cases of malignant blue nevi originating from nevus of Ota, and others have also observed orbital melanomas in patients with this condition.[18,19] Blue nevi have also been reported to occur in the orbit.[4,17,20] Blue nevi may be locally aggressive, but usually do not metastasize.

To our knowledge, there has been only one case similar to ours.[21] A 3-month-old African boy had what was thought to be a large pigmented orbital mass; however, histologic examination showed the mass to be a melanocytic hamartoma confined to the eye. In our case, however, both the orbit and the eye were involved but we could not determine the site of origin.

The histologic classification of our patient's pigmented hamartoma was difficult. The patient had a sacral mongolian spot and many blue nevi on other parts of his body.

The intraocular and orbital portion of this melanocytic hamartoma was characterized by the presence of large, benign melanocytes. These diffusely involved the uveal tract and sclera and proliferated around the lens. All melanocytic hamartomas have abnormal stromal melanocytes as a common denominator. Blue nevi are usually small (2 to 15 mm), solitary papules that occur primarily in whites. Histologically they are made up of elongated, slender, sometimes wavy melanocytes with long dendritic processes in the dermis; their long axis is parallel to the epidermis. Cellular blue nevi also have islands of spindle-shaped melanocytes with abundant cytoplasm in sparse pigment; these sometimes form a nevoid pattern.[22] Some cellular blue nevi

have a superficial resemblance to malignant melanoma, a problem that initially led to errors in classification.[23]

The prognosis is excellent for most patients with melanocytic hamartomatous proliferations. Even in those proliferations with significant pleomorphism, such as cellular blue nevi, the incidence of metastasis is rare. In nevus of Ota, nevus of Ito, melanosis oculi, and melanocytoma, reports of locally aggressive behavior or the development of frank neoplasia are rare. All patients with ocular melanocytic hamartomas need to be re-examined periodically throughout their lives.

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DEMODICOSIS OF OPHTHALMIC CONCERN

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Hair-follicle mites are the only metazoan organism commonly found in the pilosebaceous components of the eyelid of man. Our study showed that *Demodex folliculorum* in all stages is found in the small hair follicles and the eyelash hair follicles. This species, in adult and immature forms, consumes epithelial cells, produces follicular distension and hyperplasia, and increases keratinization leading, in eyelashes, to cuffing consisting of keratin and lipid moieties. *Demodex brevis* (in all stages) is present in the eyelash sebaceous glands, small hair sebaceous glands, and in the lobules of the meibomian glands. Adults and immature forms consume the gland cells in all of these loci and, when infestations are heavy, can affect the formation of the superficial lipid layer of the tear film coacervate.

Comparative studies of demodicids from man and other mammals suggest that keratinization, hyperplasia, distension, and melanocyte aggregation may be even more extensive if large populations of *D. folliculorum* build up in the follicles of the eyelid. Large populations of *D. brevis* may destroy the glandular cells, produce granuloma in the eyelid, and plug the ducts of the meibomian or sebaceous glands.

Further studies may incriminate either or both species, in conjunction with microorganisms, as transfer agents or synergists, or both, in producing ocular disease in man. Prevention and control of these mites must await experimental studies with infested laboratory animals (such as the squirrel monkey, *Saimiri sciureus*). These mites are probably most vulnerable during transfer stages, when they leave their glandular or follicular habitats.

The hair-follicle mites of man, first described by Simon[1] as *Acarus folliculorum*, are of special interest to ophthalmologists because they are found in 97% of all cadavers,[2] infest all races of man, are ophthalmically pathogenic,[3] and are potential vectors of other disease organisms.[4] Using an eyelash epilation technique, Norn[5] recovered *Demodex* (that is, *Acarus*) *folliculorum* from 82% of 100 patients. However, the difficulties he had in retrieving mites by this method led him to believe that all eyelids are infested.

Since then, Desch and Nutting[6] reported and Grosshans, Kremer, and Maleville[7] confirmed that two different spe-

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cies, *Demodex folliculorum* (Simon)—the true hair-follicle mite—and *D. brevis* Akbulatova—the sebaceous-gland mite—have been mislabeled *D. folliculorum*. We have confirmed that both species are found in the pilosebaceous components of the eyelid.

Material and Methods

We preserved biopsy and autopsy specimens from human and other mammalian eyelids and lesions in Bouin's solution, 4% formaldehyde, or 60% isopropyl alcohol. The specimens were embedded in paraffin and sections 8 to 10 μ thick were stained.[8]

A series of 50 human eyelashes with cuffing were selectively epilated and fresh-stained with either oil red O[8] or 1% aqueous acid fuchsin to determine the lipid-keratin ratio of the cuffs and to determine the effect of these stains on hair-follicle mites. We placed plucked eyelashes on depression slides and added one of the solutions. After five minutes, we rinsed the eyelashes with distilled water and examined them by light microscopy.

We obtained mite specimens from the meibomian glands of 50 patients whose glands were distended or oversecreting. We performed conjunctival lavage with balanced saline solution on these patients, hoping to find free-moving mites in the conjunctival cul-de-sac.

All samples and sections were studied with phase-contrast, interference, or conventional light microscopy.

Results

Although hair-follicle mites of the ocular area and lesions associated with their presence have been found often since 1903,[9] the precise location of each of the two mite species on the eyelids, the specific derangements they cause, and their correct differentiation has not been detailed. Norn's[5] illustrations clearly showed that *D. folliculorum* organisms can be found on epilated eyelashes and English certainly found *D. brevis* organisms in his samples.[10] We know that in eyelids, as in the rest of the body, *D. folliculorum* mites inhabit the hair follicles and *D. brevis* mites the sebaceous glands (Figs. 1 and 2). Both species puncture and consume cell cytoplasm and, possibly, ingest ambient secretions.

Demodex folliculorum—All stages of *D. folliculorum*, from egg to adult, are present in the upper part of the eyelash follicle (Fig. 1) and deeper in the small hair follicles, near the eyelid margin (Fig. 3). The opisthosomata of adult mites often project from the follicular orifice of the eyelash hairs, as Coston[11] observed with a slit lamp after stimulating the mites with ether. Although sections show that immature parasites and ova are positioned a bit deeper than adults, none have been found below the sebaceous gland duct. This also applies to the smaller hair loci. All the mites, whatever stage they are in, have their anterior surface toward the hair bases and legs and ventral gnathosomata against the follicular epithelium. The gnathosomata produces evacuation and destruction of the cells of the follicular epithelium.

Cuffing, noted by several investigators,[11] always seemed to be associated with *D. folliculorum*. Our sections (Fig. 4) stained with Masson's stain indicated that the bulk of the cuff was keratin although small gaps in the cuff were probably lipid-filled. Others were primarily lipid with a little keratin. Prominent staining with acid fuchsin indicated keratin; weak and incomplete staining with oil red O indicated small quantities of lipid. This excess keratin may be the result of the abrasive action of the mites' sharp claws and trapping the lipid in successive layers of keratin during cuff formation.



Fig. 1 (English and Nutting). Eyelid section shows *D. folliculorum* (M) in the hair follicle. Note mite mouthparts (arrow) embedded in epithelium and strap-like layers of keratin (hematoxylin and eosin, ×400).

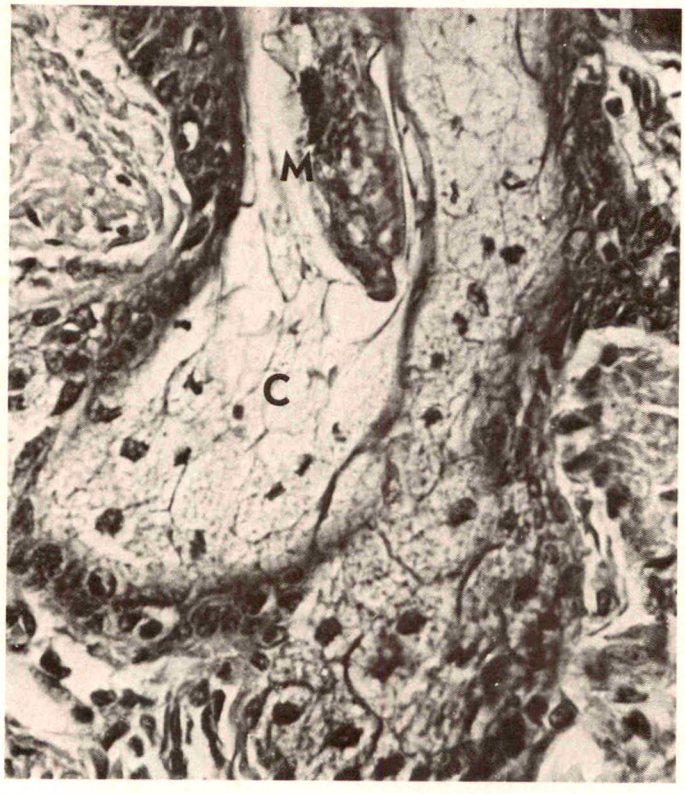


Fig. 2 (English and Nutting). Section of sebaceous gland of an eyelash shows *D. brevis* (M). Note gland cell (C) destruction (McManus, ×375).



Fig. 3 (English and Nutting). Cross-section through small hair follicle of the eyelid. Note the distension, hyperplasia, and moderate epithelial keratinization caused by the activities of *D. folliculorum* (arrow) (hematoxylin and eosin, ×375).

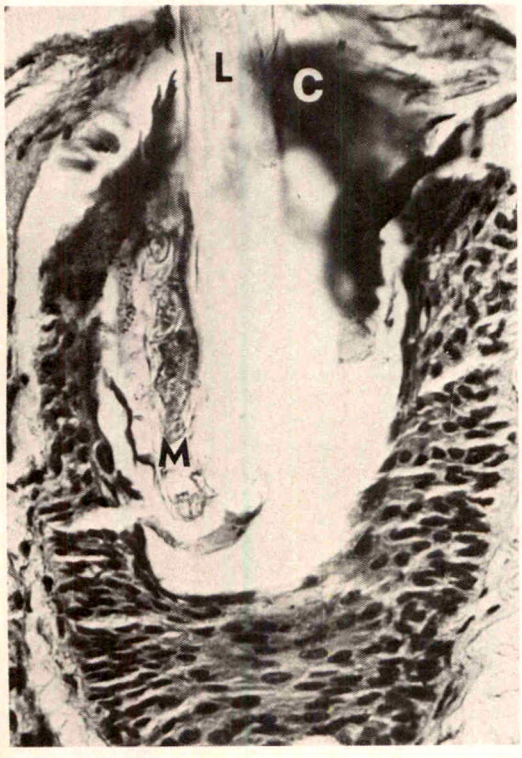


Fig. 4 (English and Nutting). Section of eyelid shows eyelash (L), cuffing (C), and small segment of *D. folliculorum* (M). Note layering of cuff (Masson, ×275).

Acid fuchsin stained the exoskeletons of *D. folliculorum* adults faintly, whereas oil red O was rapidly absorbed by internal structures,[12] deeply staining the posterior gut cells and, later, the salivary glands.

Populations of *D. folliculorum* were generally small (one to six mites) in our sectioned eyelash follicles, but slightly larger (three to eight mites) in the small hair follicles of the eyelid. In several heavily populated sections (Fig. 3), the infestations showed follicular distension and epithelial hyperplasia with an increase of keratinization adjacent to the claws of the mite.

English and associates[4] reported that *D. folliculorum* is associated with bacteria. Their electron micrographs clearly showed that bacteria adhere to the exoskeletons of the mites. To our knowledge, no bacteria have been found within the mites themselves, although Borrel[13] found *Mycobacterium leprae* adjacent to both mite species in facial areas.

Demodex brevis—Although unidenti-

fied until 1972, *D. brevis* is probably the species noted by Majocchi[14] in 1891 and by English[10] in 1971. We found this species adhering to epilated eyelashes and also in some of the lavage samples.

In our sections, *D. brevis* organisms were located in the sebaceous glands of the eyelashes (Fig. 2) and the small hairs of the eyelid. We found the first definite instance of infestation of the meibomian glands by this species (Fig. 5). The parasite was located deep inside the gland, explaining the difficulty of demonstrating demodicids by tarsal massage techniques. Populations of this species seem limited to one to four per sebaceous gland lobule. These are usually a female with an egg or an immature organism; occasionally a male is found.

Cell destruction (Fig. 2) by *D. brevis* is evident and the mites often produce channels reaching to the germinal epithelium. We surmise that this species was responsible for the granulomatous condition noted by Stcherbatchoff.[9] We found no hyperkeratinization or hyperplasia.

Pathogenesis—The only gross symptoms of ocular disease we have observed are cuffing related to *D. folliculorum* and itching, which may be caused by either or both species. At the cellular level, both species puncture and suck out cell cytoplasm and, probably, ambient secretions. In *D. folliculorum* infestation, mechanical abrasion produces keratin and eyelash cuffing. The amount of lipid in the cuffs varies. Lipid is probably removed by washing unless it is trapped by keratin. Both species are, therefore, minor pathogens of the ocular area.

Discussion

Studies of hair-follicle mites situated elsewhere on the human body and of

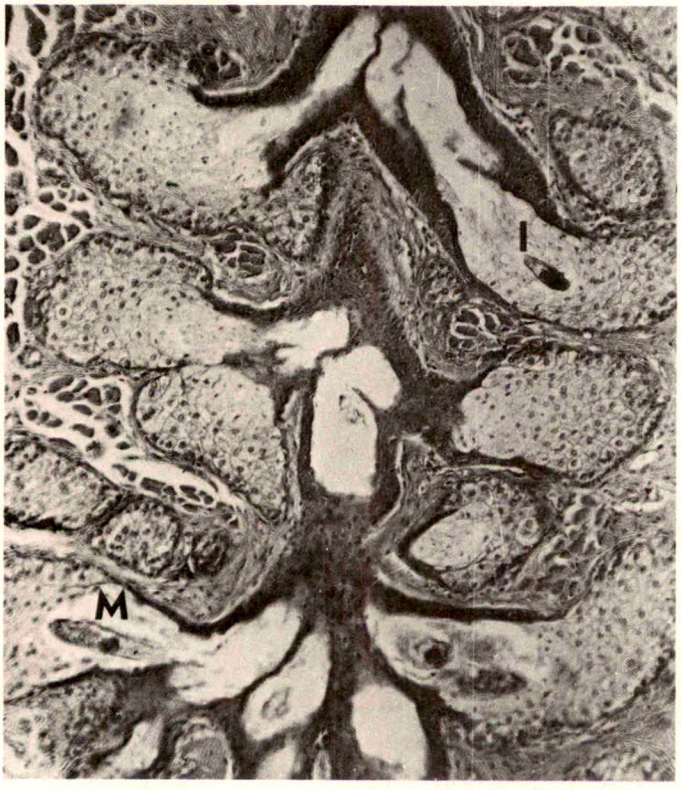


Fig. 5 (English and Nutting). Section through a meibomian (tarsal) gland shows an immature (I) and an adult (M) *D. brevis*. Infested lobules show marked cellular destruction (hematoxylin and eosin, ×150).

demodicid species in other mammals have indicated a greater potential for pathogenesis in the ocular area than has hitherto been suspected. Nutting's[15] report and our recent findings suggest the following conclusions.

1. *Demodex folliculorum*, especially in facial areas,[16] produces large lesions and itching.[17] Large populations produce marked follicular distension, increased keratinization that plugs the follicular orifice, epithelial hyperplasia, and an increase in the number of capillaries (Fig. 6).

Demodex brevis has now been incriminated in granuloma formation.[18] Studies have shown that giant cells ingest the mites (Fig. 7). One report[19] described an early inflammatory response, related to *D. brevis* organisms which may have penetrated the dermis and capillary network.

2. Nutting and Dailey[20] found markedly increased keratinization of the follicular epithelium in sections of *Demodex*-infested skin (Fig. 8) from a California sea lion. As the population of mites increased, the mites moved deeper into the hair follicle, dislodging hairs, and increasing hyperplasia and distension below the advancing sheet of keratin. This strongly indicated abrasive action by the mites' claws, producing keratin as in *D. folliculorum* infestation of the eyelids.

3. Plugging of the meibomian ducts by *D. gapperi*[21] (Fig. 9) in the red-backed vole seems responsible for duct distension, increased melanocyte aggregation, and decreased glandular lubrication of the eyelids leading to sealing of the eyelids. Gross signs, other than sealing, include moderate swelling and erythema above each ductal orifice.

An unidentified *Demodex* organism



Fig. 6 (English and Nutting). Section of skin from the nasolabial area shows marked distension, epithelial hyperplasia, and massive keratinization leading to orifice plugging. M, *D. folliculorum* (hematoxylin and eosin, ×425).

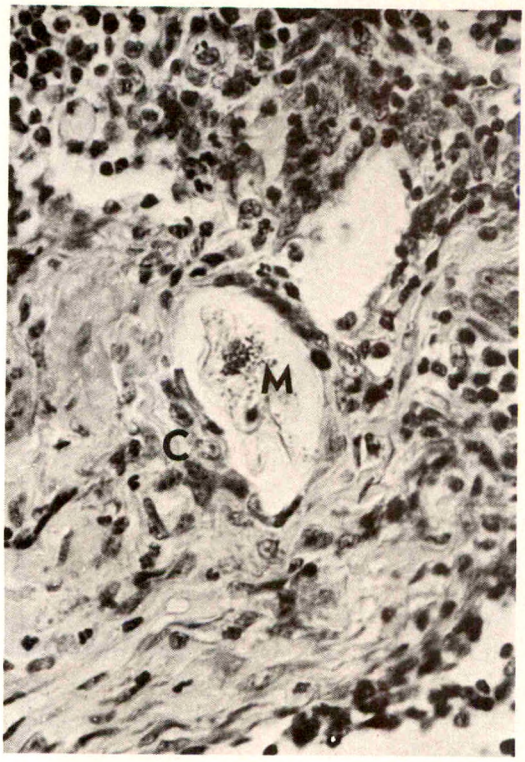


Fig. 7 (English and Nutting). Section of skin shows reaction to *D. brevis* (M) by forming a giant cell (C) and other host cells typical of a granulomatous condition (hematoxylin and eosin, ×450).

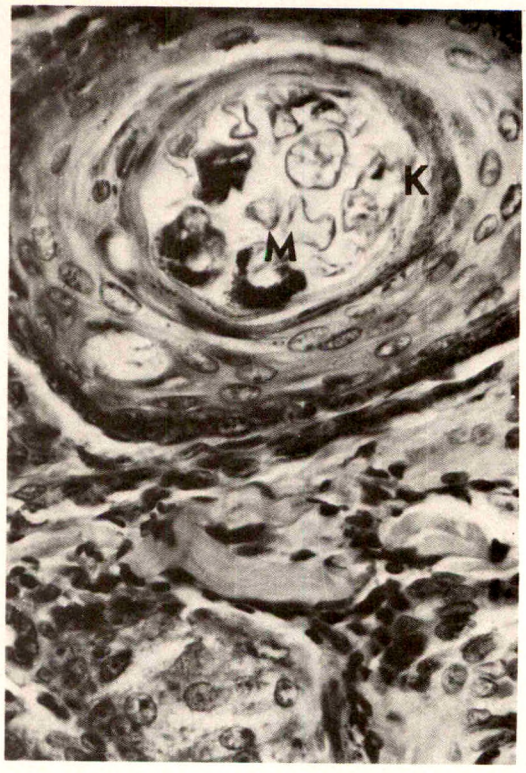


Fig. 8 (English and Nutting). Section of skin of the California sea lion, *Zalophus californicus*, shows follicular distension, hyperplasia, and keratinization (K) caused by *D. zalophi* (M) (hematoxylin and eosin, ×425).



Fig. 9 (English and Nutting). Section of eyelid of the red-backed vole, *Clethrionomys gapperi*, shows marked plugging of the meibomian duct by demodicids (M) and some duct distension. Note melanocyte aggregation (hematoxylin and eosin, ×425).

found in a nine-banded armadillo (*Dasypus novemcinctus*) has been studied by Marvin Baker. Sections of the eyelid showed that, like *D. brevis*, this organism invades the sebaceous gland. Unlike *D. brevis*, large populations build up in each lobule, decimate the cells (Fig. 10), move into the dermis, and produce granuloma (Fig. 11) (unpublished data).

Hakugawa[17] demonstrated large populations of *D. brevis* in facial lesions.

Cell destruction, granuloma formation, and duct obstruction may occur during *D. brevis* invasions of several of the pilosebaceous components of the ocular area, including the meibomian gland complex. This may be a factor in chalazion formation.

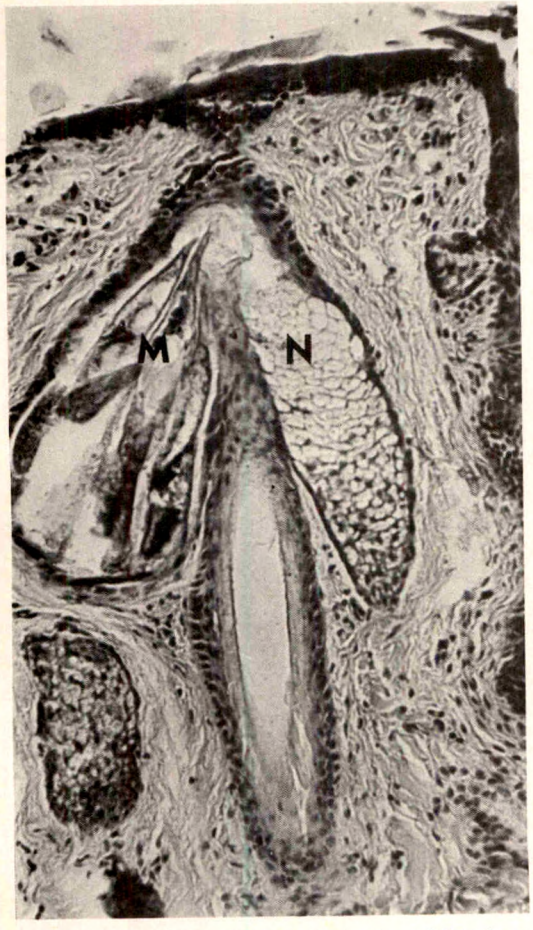


Fig. 10 (English and Nutting). Section of skin from a nine-banded armadillo (*Dasypus novemcinctus*) shows decimation of glandular cells by an unidentified *Demodex* species. M, mites; N, uninfested sebaceous gland (hematoxylin and eosin, ×225).

4. Systemic invasions by *D. canis*, including infestation of lymph nodes, often occurs in cases of generalized demodectic mange in dogs (Fig. 12).[22] Other demodicid mites have been located in venules of the marsupial mouse[15] and the California sea lion.[20]

5. Massive invasions of *D. canis* seem to be associated with *Staphylococcus aureus*. Scott, Farrow, and Schultz[23] reported the only immune response in demodicosis. Thierry[24] has recently reported a weak in vitro Ouchterlony reaction using *D. folliculorum*.

We believe that large populations of *D. brevis* in the ocular pilosebaceous areas can penetrate the lymphatics or possibly venules, lodge in the cervical lymph nodes, and cause either an immune reaction or mild toxemia. Either species in man may also produce the synergistic reactions evident in demodectic mange of the dog which often begins with localized distortion of the ocular area.[25]

6. A new genus of demodicid, *Ophthalmodex artibei*, has been found in a Surinam bat (*Artibeus lituratus*).[26] They are minute, tortoise-shaped, free-living organisms (Figs. 13 and 14) which wander across the conjunctiva and apparently feed on conjunctival cells. These mites also abrade the cornea and plug the lacrimal ducts. In primitive conditions, such a free-living demodicid could transfer readily to man as it is not species-specific. From this, we conclude that *D. brevis* and *D. folliculorum* may occasionally invade the lacrimal complex. However, trying to manipulate a free-floating parasite into the lacrimal punctum is a formidable and time-consuming project.

7. The observation of English and associates[4] that bacteria adhere to the exoskeletons of *D. folliculorum* and the

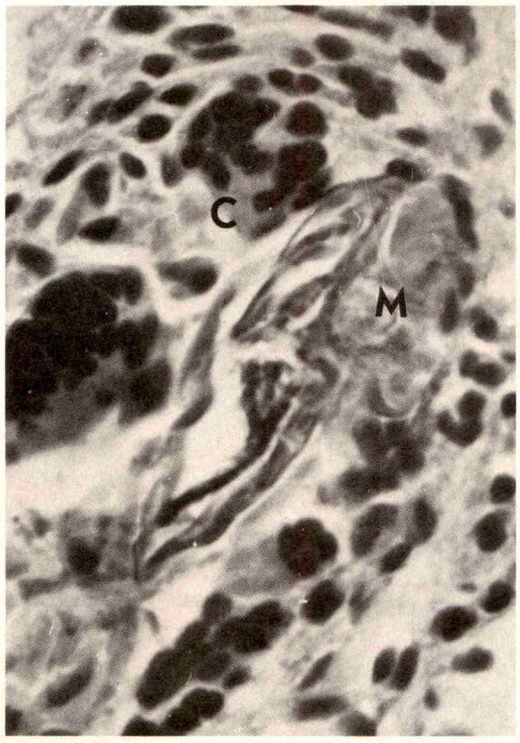


Fig. 11 (English and Nutting). Section of skin from a nine-banded armadillo infested by an unidentified *Demodex* species shows granulomatous condition around mites in the dermis; M, mite; C, giant cell (hematoxylin and eosin, ×425).

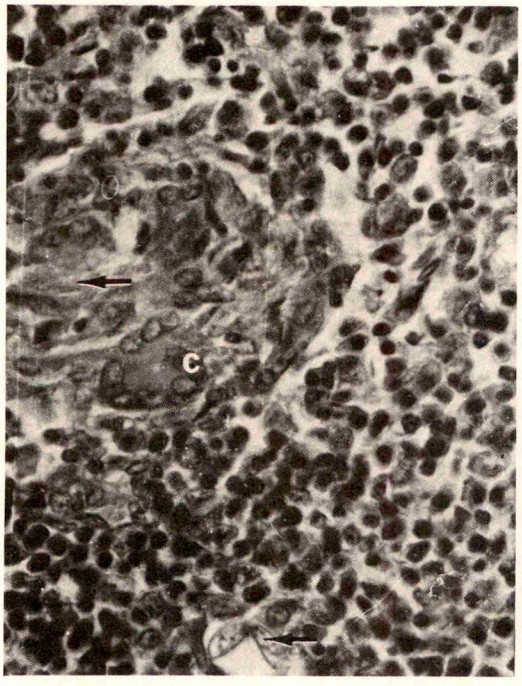


Fig. 12 (English and Nutting). Section of canine lymph node shows *D. canis* (arrows). C, giant cell (hematoxylin and eosin, ×440).

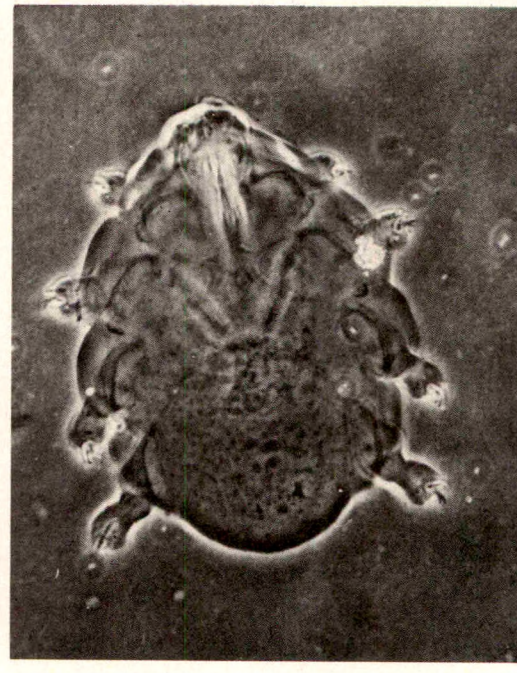


Fig. 13 (English and Nutting). Male mite, *Ophthalmodex artibei*, from a Surinam bat (*Artibeus lituratus*). This mite species is a free-living demodicid that wanders across the cornea (phase contrast, ×325).

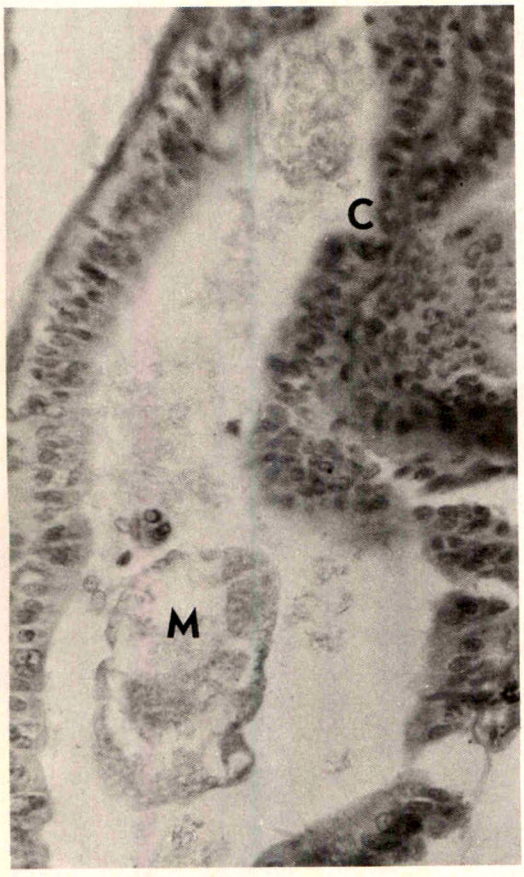


Fig. 14 (English and Nutting). Section through the ocular fornix of a Surinam bat (*Artibeus lituratus*) shows *Ophthalmodex* species (M) and derangement of the conjunctiva (C) (hematoxylin and eosin, ×325).

suggestion by Post and Juhlin[3] that both suppurative and ulcerative blepharitis are *Demodex*-related show that further experimental studies are needed. We must learn how to culture, seed, and provide an optimal environment for these mites and any transported organisms.

Similar conditions are present in canine demodicosis in which large mite populations are associated with blepharitis.[25] The major difference between canine and human demodicosis is that only one demodicid, *D. canis*, has been found in dogs to date. This species, however, does invade sebaceous glands, as well as hair and eyelash follicles, thus producing initial lesions comparable to those caused by both demodicids in man.

8. The squirrel monkey, *Saimiri sciureus*, is probably the best animal for experimental work on ocular demodicosis.[27] Lebel and Nutting[28] described an elongated demodicid, *D. saimiri*, very similar to *D. folliculorum*, found in the eyelid of this monkey. They also discovered two other congeners which, like the demodicids of man, seem to have distinctive habitat differences.

Topical applied medications (even in combination) have little if any effect on these mites while they are deep inside the pilosebaceous complex. When, however, the adults move from the hair follicle or glandular ducts and surface on the skin to relocate, as Norn[29] has suggested, they are vulnerable. Trials with a variety of topically applied drugs in Golden hamsters,[30] Coston's[11] use of ronnel on himself, and the reputed cures of canine demodicosis with ronnel have not tested this transfer stage theory or proven mite extirpation. These treatments and Ayres'[31] reported reduction of clinical signs of human demodicosis with Danish ointment should be repeated with adequate controls and sequential biopsy examinations in a series of laboratory experiments with special attention to the ocular and nasolabial fold areas. Our early results suggest that cryotherapy is a promising method of destroying individual parasites.

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MIOSIS FROM ECHOTHIOPHATE IN PATIENTS WITH TOTAL OCULOMOTOR NERVE PARALYSIS FROM INTRACRANIAL ABNORMALITIES

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Seven subjects with intracranial pathologic abnormalities received bilateral instillations of echothiophate. Six had unilateral third (oculomotor) nerve paralysis; one had unilateral third nerve paresis. All 14 eyes became miotic. Miosis was greater in the defective eye in three subjects, greater in the normal eye in two subjects, and equal in two subjects.

The neurons originating in the ciliary ganglion and terminating on the sphincter pupillae muscle may exhibit spontaneous activity. Echothiophate, by preventing acetylcholine hydrolysis, may have allowed this transmitter to accumulate and to produce miosis.

The amount of maximal miosis in the abnormal eye after bilateral instillation of echothiophate drops may vary from that in the normal eye. The causes of this variation include denervation supersensitivity of the sphincter pupillae muscle, partial paresis of the dilator pupillae muscle, and increased drug penetration in the abnormal eye.

The preganglionic parasympathetic fibers to the sphincter pupillae muscle begin in the gray matter dorsal to the nucleus of the third nerve.[1] These axons travel with the intracranial portion of the oculomotor nerve. In the orbit, they leave the inferior division of the third (oculomotor) nerve in the branch to the inferior oblique muscle and terminate in the ciliary ganglion. The postganglionic fibers travel in the short ciliary nerves to the sphincter pupillae muscle where they release acetylcholine.[2]

Cholinesterase-inhibitors exert their effects indirectly by preventing the hydrolysis of endogenously released acetylcholine. For example, miosis will not occur after the application of diisopropylfluorophosphate to the eye if the postganglionic fibers of the ciliary ganglion have been destroyed, because these fibers are the source of endogenous acetylcholine.[3,4] If, however, preganglionic denervation occurs, the cell bodies in the ciliary ganglion survive. Thus, the peripheral autonomic fibers, unlike central nervous system neurons, do not undergo sequential (postsynaptic) degeneration.[2,5,6] If a cholinesterase inhibitor is placed on the eye of a patient with a proximal denervation of the ciliary ganglion, one of two pupillary responses occurs. The pupil may remain dilated because the postganglionic fibers do not spontaneously release acetylcholine, or the pupil may constrict because the postganglionic fibers do spontaneously release small amounts of acetylcholine. I studied the

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response of the sphincter pupillae muscle to echothiophate in seven patients with intracranial pathologic abnormalities. Six had complete absence of third nerve function unilaterally and one had minimal third nerve function unilaterally.

Subjects and methods

I used commercial preparations of echothiophate, cyclopentolate, atropine, and phenylephrine. Photographs were taken with a 35-mm camera with fixed-focus lenses. Room lighting was constant for a given patient on a given day but varied from patient to patient and from day to day. When photographs were taken to demonstrate ocular motility, no attempt was made to control accommodation. When photographs were taken to demonstrate pupillary diameter, the patient was instructed to fixate on a mark on the camera.

All patients except Patient 4 had no evidence of third nerve function by the following criteria: no pupil response to light by slit-lamp examination, no adduction or elevation of the globe, and no depression of the eye except that associated with intorsion, that is, superior oblique muscle activity. Patient 4 had minimal pupillary constriction observable with the slit lamp.

Patients 1, 2, 5, and 7 kept both eyelids closed for periods of ten to 35 minutes immediately after receiving echothiophate drops. Patients 3, 4, and 6 were permitted to open the eyelids of their unaffected eyes.

Case reports

Case 1—This 28-year-old man had chronic third nerve paralysis and acute third nerve transection resulting from recurrent meningioma of the left middle cranial fossa. Initial removal of the meningioma had taken place two years previously. Two months before his second operation, he developed a left third nerve paresis with diplopia followed by blepharoptosis. The tumor was removed along with cranial nerves three, four (trochlear), and six (abducens); the left internal carotid artery was ligated and embolized. Postoperatively, his corrected visual acuity in the left eye was 6/6 (20/20); the cornea had no sensation. One drop of 0.25% echothiophate was instilled in each eye on the sixth postoperative day. Both eyelids were kept closed for ten minutes after the drops were instilled. The following day, 1% atropine was instilled in the left eye only (Fig. 1).

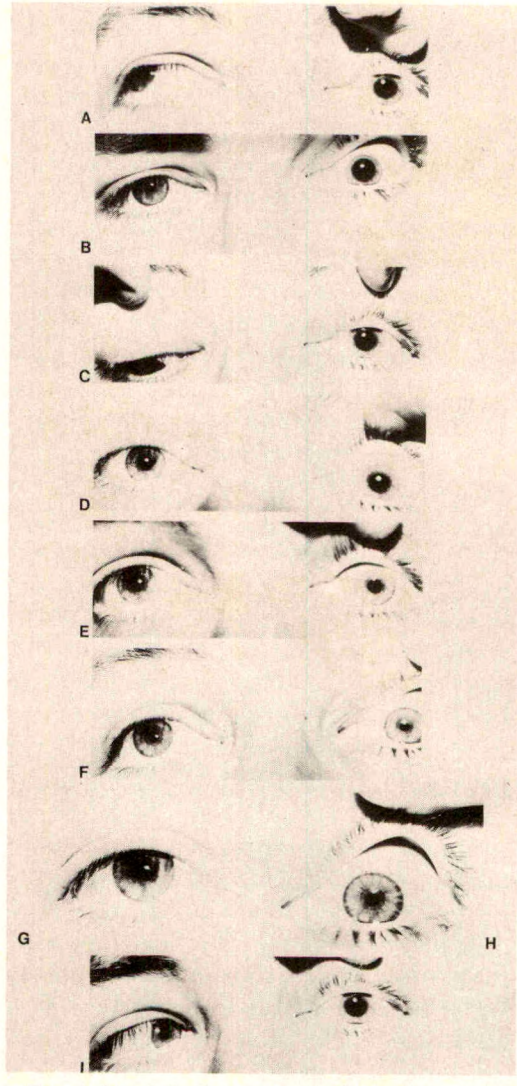


Fig. 1 (Mindel). Patient 1. Paralysis of the third, fourth, and sixth cranial nerves (A, B, C, and I) of the left eye. Miosis begins 45 minutes after echothiophate instillation (D), progresses at 75 minutes (E) and 150 minutes (F) after instillation, and reaches its maximum point at 150 minutes in the right eye (G) and the left eye (H). Maximal miosis is less in the right eye than in the left. Mydriasis is present in the left eye (I) 24 hours after echothiophate instillation and two hours after instillation of 1% atropine. Note the transient paradoxical increase in the pupillary diameter of the right eye as the pupil of the left eye constricts (B, D, and E).

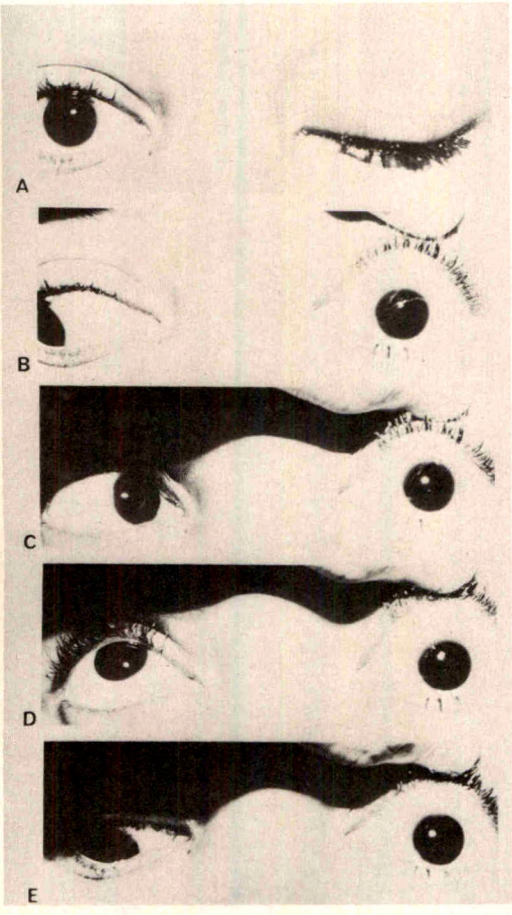


Fig. 2 (Mindel). Patient 2. Total paralysis of the left third, fourth, and sixth cranial nerves.

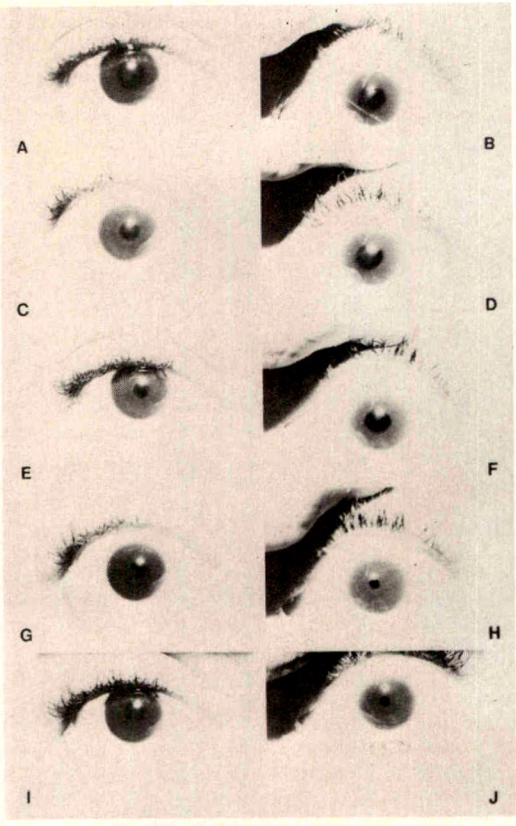


Fig. 3 (Mindel). Patient 2. The cornea of the left eye is congenitally smaller than that of the right eye. Before echothiophate instillation, the horizontal, but not the vertical, pupillary diameter is smaller in the normal eye (A) than in the paretic eye (B). Miosis progresses 30 (C and D), 90 (E and F), and 135 (G and H) minutes after echothiophate instillation. There is a paradoxical dilation of the normal pupil as the miosis of the abnormal pupil exceeds that of the normal pupil. Maximal miosis is still greater in the paretic eye 18 hours after echothiophate instillation (I and J).

Case 2—This 31-year-old woman, who had undergone partial resection of a meningioma of the left middle cranial fossa one week previously, had third nerve paralysis (Fig. 2). The tumor involved the left clivus, sella, and parasellar area. The anterior segment of the left eye was congenitally smaller than the right. Postoperatively, the corrected visual acuity in her left eye was 6/6 (20/20); the cornea had no sensation and the globe was completely immobile. One drop of 0.25% echothiophate was instilled in each eye and the eyes were kept closed for ten minutes (Fig. 3).

Case 3—This 53-year-old woman had third nerve paralysis after a traumatic carotid-cavernous fistula caused by an accidental penetrating injury of the right intracavernous carotid artery during partial removal of a suprasellar pituitary tumor. When she awoke after the surgery, she had complete paralysis of the right third nerve and partial paralysis of the right sixth nerve. The right cornea had no sensation. A large carotid-cavernous fistula was demonstrated angiographically. One week postoperatively, one drop of 0.125% echothiophate was instilled twice in each eye; the instillations were five minutes apart.

Case 4—A 39-year-old man with a previous small subarachnoid hemorrhage from an aneurysm of the left posterior communicating artery had surgical trauma to the third nerve. At surgery the aneurysm was clipped. Postoperatively, there was a nearly total paralysis of the left third nerve. A slit-lamp examination disclosed a minimal pupillary response to bright light. Eight days postoperatively, one drop of 0.25% echothiophate was instilled in each eye (Fig. 4).

Case 5—A 70-year-old man had a left suprasellar tumor, demonstrated by computed axial tomography, that caused compression of the third nerve. He had a two-month history of left blepharoptosis. I found paralysis of the left third nerve and paresis of

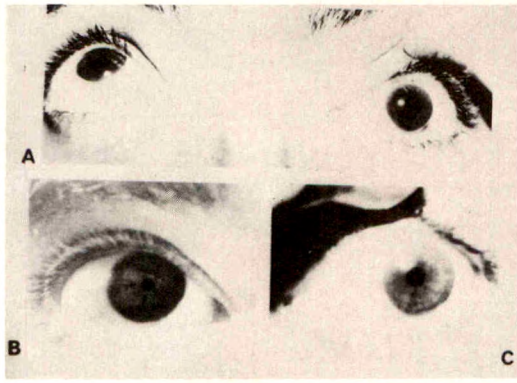


Fig. 4 (Mindel). Patient 4. Almost total third nerve paralysis in the left eye (A). Maximal miosis is greater in the normal eye (B) than in the paretic eye (C) three hours after echothiophate instillation.

the left fourth and sixth nerves. Corneal sensation was normal. One drop of 0.25% echothiophate was instilled in each eye and both eyelids were kept closed for 20 minutes (Fig. 5).

Case 6—A 73-year-old woman suffered left third nerve paralysis after subtotal removal of a sphenoid meningioma. Three weeks postoperatively, one drop of 0.125% echothiophate was instilled twice in each eye; the instillations were five minutes apart. The following day, both eyes received one drop each of 1% cyclopentolate and 10% phenylephrine (Fig. 6).

Case 7—A 67-year-old man suffered a right third nerve paralysis after a frontal craniotomy for removal of a parasellar tumor. Nine days postoperatively both eyes received three applications of 0.125% echothiophate; the instillations were five minutes apart; both eyes were kept closed for 35 minutes (Fig. 7).

Results

Echothiophate produced miosis in both eyes of all seven patients even though six had unilateral complete third nerve paralysis and the seventh had a marked unilateral third nerve paresis. The latencies and intervals until maximal miosis varied in both normal and abnormal eyes. In all seven normal eyes, noticeable miosis occurred within 60 minutes of echothiophate instillation and reached its maximum within two hours of instillation. The onset of miosis in the abnormal eye usually, but not always (Fig. 1), appeared to lag behind that of the normal eye. In eyes with defective third nerve innervation, miosis reached its maximum

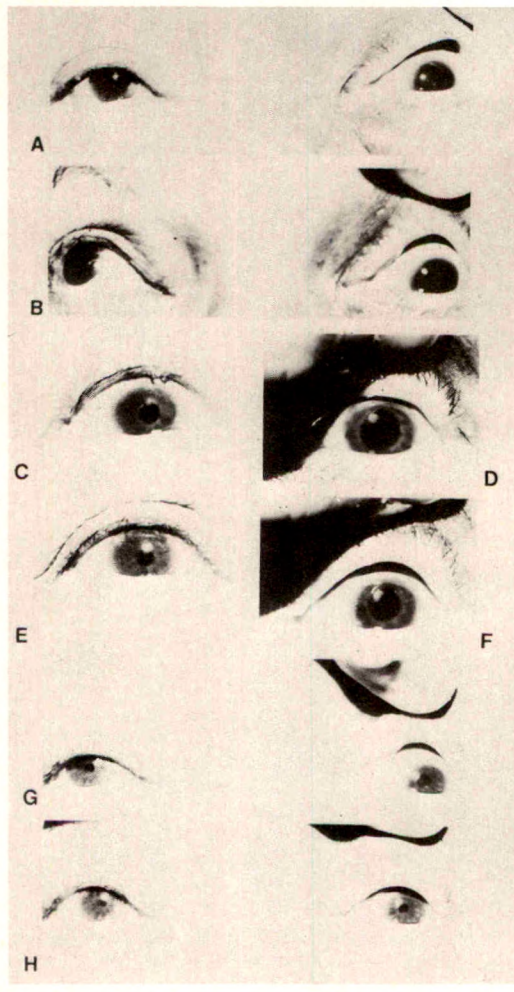


Fig. 5 (Mindel). Patient 5. Left third nerve paralysis and left sixth nerve paresis (A and B). There is almost no pupillary response in the right eye (C) or the left eye (D) 20 minutes after echothiophate instillation. There is early miosis in the right (E) and the left (F) eyes one hour after echothiophate instillation and significant miosis after 90 minutes (G). Maximal miosis is slightly greater in the normal eye two hours after echothiophate instillation (H).

within 150 minutes after instillation. A transient paradoxical increase in the pupillary diameter of the normal eye was occasionally demonstrated after echothiophate (Fig. 1, B and D to F, and Fig. 3).

In all three subjects, Patients 1 to 3, the maximal miosis produced by echothiophate was greater in the eye with defective third nerve innervation (Figs. 1 and

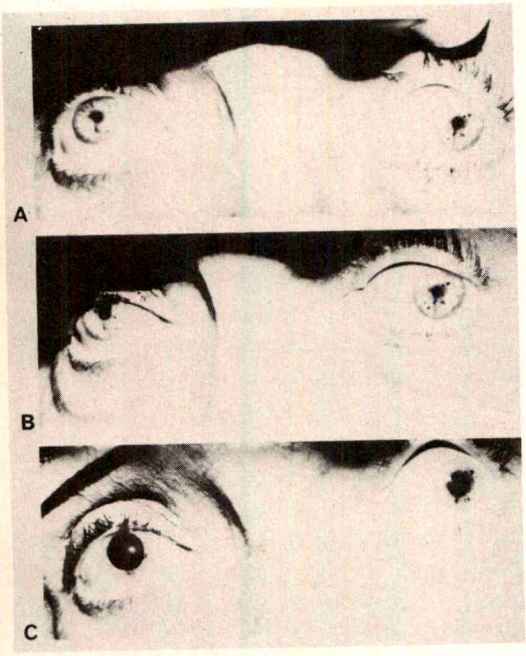


Fig. 6 (Mindel). Patient 6. A, Maximal miosis is approximately equal in the normal right eye and the paretic left eye two hours after echothiophate instillation. B, At 18 hours, the miosis of the normal pupil has partially regressed but not that of the left eye. C, Mydriasis is greater in the normal eye 19 hours after echothiophate instillation and one hour after instillation of 1% cyclopentolate and 10% phenylephrine.

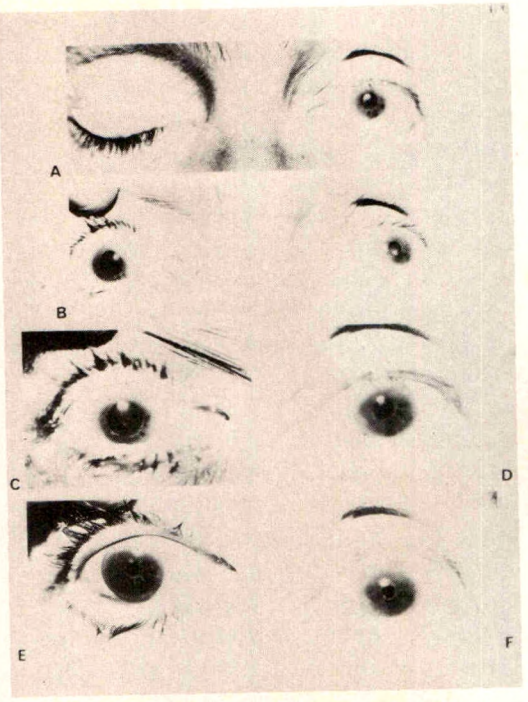


Fig. 7 (Mindel). Patient 7. A and B, Right third nerve paralysis. C, Baseline size of right pupil. D, Baseline size of left pupil. Maximal miosis is approximately equal in the right (E) and left (F) eyes 90 minutes after echothiophate instillation.

3). The maximal miosis was almost equal in two subjects, Patients 6 and 7 (Figs. 6 and 7). Patient 4 had minimal residual function of the sphincter pupillae muscle in the paretic eye; maximal miosis was greater in the normal eye in this subject and also in Patient 5 (Figs. 4 and 5). Recovery occurred more rapidly in the normal eye in Patient 6 (Fig. 6), that is, the duration of maximal miosis was longer in the defective eye.

Discussion

To my knowledge, there have been no previous reports of the pupillary responses elicited by cholinesterase inhibitors in eyes with total third nerve paralysis caused by intracranial pathologic abnormalities. This may be the result of the residual function found in most patients with a third nerve defect. This means that the intracranial lesion is incomplete and that some transmission occurs along the oculomotor nerve. At other times, especially in older lesions, aberrant regeneration is present; sector contractions of the sphincter pupillae muscle occur during ocular movements and in response to light stimulation; the eyelids retract on downgaze; and the superior and inferior recti muscles cocontract on upgaze and downgaze.[7]

In this study, only six patients, out of a much larger number that were examined, met the following criteria for complete intracranial third nerve paralysis: no pupillary response to light by slit-lamp examination; no adduction or elevation of the globe; and no depressions of the eye

except those associated with intorsion, that is, superior oblique muscle activity. Even these criteria were suspect because residual function of the inferior rectus muscle might have been mistakenly attributed to superior oblique muscle activity. For this reason, Patients 1 and 2 are the most important in this study (Figs. 1 to 3). They had no ocular motility in any direction and no sphincter pupillae muscle activity. At surgery, Patient 1 had the intracranial third, fourth, and sixth nerves to the left eye transected and partially removed. Patient 2 had no pupillary movement after partial removal of a meningioma of the middle fossa. However, the immobile pupils of both these patients did constrict when echothiophate was applied. This suggested that some spontaneous activity was occurring in the neurons originating in the ciliary ganglion. The amount of acetylcholine released by the postganglionic fibers before the instillation of echothiophate was too small to stimulate the fibers of the sphincter pupillae muscle and was therefore unable to overcome the mydriatic force of the sympathetic dilator pupillae muscle. Echothiophate allowed the acetylcholine to accumulate. The resulting miosis can be reversed, as was demonstrated in Patient 1, by atropine, a drug that competes with acetylcholine for the muscarinic receptor. In subjects with other causes of third nerve paralysis, such as carotid-cavernous fistula, aneurysm surgery, and tumor compression, echothiophate produced miosis consistent with this explanation.

There are other sources of intraocular acetylcholine in addition to the parasympathetic nerve terminals in the iris and ciliary body. Acetylcholine has been detected in the corneal epithelium[8,9] but not in the stroma or endothelium.[10] Corneal acetylcholine probably did not produce miosis in these patients because the acetylcholine was located within the epithelial cells[11] and not in nerve endings from which it could be easily released. Furthermore, prolonged eyelid closure, like that which occurred in these patients with blepharoptosis, markedly reduces the level of corneal acetylcholine in experimental animals.[9] Acetylcholine-like activity has also been detected in the retina and posterior uvea,[12,13] but the few applications of echothiophate used in this study probably did not significantly affect the acetylcholinesterase activities in these retrolental tissues.

An interesting feature in Patients 1 and 2 was a transient paradoxical increase in the pupillary diameter of the normal eye after bilateral instillation of echothiophate (Fig. 1, B and D to F, and Fig. 3). I assumed that this was caused by the initial marked dilation of the abnormal eye. As the abnormal pupil constricted, less light entered, decreasing the stimulus for bilateral reflex miosis. After 150 minutes, the pharmacologic action of echothiophate, rather than the amount of light reaching the retina, became the major factor determining the diameter of the normal pupil. The miosis produced in the normal eye by a single drop of 0.25% echothiophate did not appear to be maximal; additional drops would probably have produced more constriction.

Another interesting feature in Cases 1 to 3 was that the maximal miosis produced by echothiophate was more marked in the defective eye than in the normal eye. There are three possible explanations:

1. Denervation supersensitivity of the sphincter pupillae muscle—If the single application of echothiophate produced a submaximal contraction of the sphincter pupillae muscle in the normal eye, then supersensitivity could have produced a greater miosis in the defective eye.

2. Paresis of the dilator pupillae muscle—Patients 1 and 3 had trauma to the carotid artery and cavernous sinus. If

there were damage to the postganglionic sympathetic fibers contained in these structures, Horner's syndrome would result. However, the miosis and blepharoptosis produced would have been masked by the effects of the third nerve paralysis. The weakness of the dilator pupillae muscle would only have become apparent when the sphincter pupillae muscle contracted in response to echothiophate. The result would be an exaggerated miosis.

3. Increased drug penetration—Although the affected eyes of Patients 1 to 3 lacked corneal sensation, they were protected by their blepharoptotic eyelids. However, increased drug penetration could have occurred by two mechanisms. First, there was decreased reflex tearing in the eyes without corneal sensation; the echothiophate would not have been washed out as rapidly as in the normal eye. Second, the blepharoptosis itself would have made the drug-cornea interaction more prolonged than that of the normal eye, which could open and blink. Of these variables, the only one that could be controlled was the opening and blinking of the eyelids of the normal eyes. Patients 5 and 7 were instructed to keep both eyes closed after receiving their echothiophate drops. The resulting maximal miosis was bilaterally equal (Figs. 5 and 7). Patients 1 and 2 also kept both eyelids closed, yet their maximal miosis was unequal. One or both of the other factors may have been at work.

Other variations in the pupillary response to echothiophate demonstrate the number of factors involved. Patient 4 (Fig. 4) developed greater miosis in the normal eye. Because the normal eye was permitted to blink, echothiophate penetration should have been greater in the affected eye. Additionally, this was the only subject in the study with a residual light response in the affected eye, indicating some intracranial oculomotor nerve function. More acetylcholine should have accumulated in the paretic iris of this patient than in the paralyzed irides of the other patients. Yet, unlike Patients 1 to 3, Patient 4's miosis was greater in the normal eye. Perhaps the paretic iris lacked supersensitivity. Patient 6 (Fig. 6) had bilaterally equal maximal miosis, but, 18 hours after echothiophate instillation, partial recovery was evident only in the normal eye. What combination of unequal drug penetration, sphincter pupillae muscle supersensitivity, and dilator pupillae muscle paresis produced this effect? The subsequent greater mydriasis of the normal eye to cyclopentolate plus phenylephrine did not clarify the situation.

Two subjects (Patients 1 and 2) were young enough for a detectable accommodation of the lens to be expected if acetylcholine were accumulating in the ciliary muscle. Only Patient 2 was tested in this regard. Her uncorrected visual acuity for near before echothiophate instillation was 6/4.5 (20/15) in the normal eye and 6/24 (20/80) in the affected eye. A $+2.00$ $+1.00 \times 90$ lens improved the visual acuity for near of the affected eye to 6/6 (20/20). This presbyopia disappeared after echothiophate instillation and uncorrected visual acuity improved to 6/4.5 (20/15).

Miosis consistently occurred after echothiophate instillation despite total oculomotor nerve paralysis from intracranial pathologic abnormalities. Perhaps the neurons originating in the intraorbital ciliary ganglion exhibit spontaneous activity with the release of small quantities of acetylcholine. The variation in the relative miosis in the abnormal and normal eyes of a bilaterally treated subject indicated that several additional factors play a role in determining the amount and duration of miosis. The level of spontaneous ciliary ganglion activity may vary from patient to patient, resulting in

different degrees of sphincter pupillae muscle supersensitivity. The strength of the antagonistic pupillae dilator muscle may be impaired either by the intracranial abnormality or by surgery. Corneal penetration of echothiophate may be increased by blepharoptosis and by a loss of reflex tearing, the former resulting from oculomotor nerve paralysis and the latter resulting from fifth (trigeminal) nerve involvement.

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SENILE LENS CHANGES AND DIABETES IN TWO POPULATION STUDIES

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Clinical and epidemiologic studies of the association between senile cataract and diabetes have produced what initially appears to be conflicting evidence. Three studies of extracted cataract have found a positive association in persons less than 70 years old. Conversely, two studies of senile lens changes (including aphakia) reported no association with diabetes. These apparently disparate findings led to the hypothesis that the diseases are in fact not associated, but that diabetics are more likely to be referred to ophthalmologists and therefore to have cataracts removed.

We tested this hypothesis with data from two population surveys, the Framingham Eye Study and the Health and Nutrition Examination Survey (HANES). Both studies showed a marked excess prevalence of senile cataract (including aphakia) in diabetics less than 65 years old (relative risks of 4.02 and 2.97, respectively). Beyond the age of 64 years, the HANES study but not the Framingham study showed an excess prevalence, although it was less marked (relative risks of 1.63 and 1.02, respectively). For senile lens changes other than cataract, both studies showed little, if any, association with diabetes.

Our results for cataract (whether removed or not) were generally consistent with those of previous studies of extracted cataract for persons under the age of 70 years and for persons aged 70 years or more. The two studies that reported no association between lens changes and diabetes provided information inadequate to support or refute the foregoing findings. The various studies failed to support increased referral of diabetics to ophthalmologists as an explanation for the observed association. All these studies found the association in persons less than 70 years old, whether the cataract had been removed or not.

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Clinical and epidemiologic investigations of the question of whether senile cataract and diabetes are associated have resulted in what appears to be conflicting evidence. Studies of extracted cataracts have uniformly found a positive association, at least in people under the age of 70 years.[1-3] Thus, Caird and associates[1,2] re-

ported that senile cataract extraction was more common in diabetics than in nondiabetics living near Oxford, England. Similarly, Hiller and Kahn[3] found excess cataract extractions among diabetics under the age of 70 years in a national survey of United States hospitals and in a Washington, D.C., hospital.

Conversely, Waite and Beetham,[4] in a study of clinic patients in Boston, reported, "There is an equal incidence in diabetics and non-diabetics of cataracts of all sorts except flocculi in juvenile diabetics." Similarly, McGuinness[5] reported, "The prevalence of diabetes in a sample of 340 persons of the general population of Rhondda Fach [Wales] was not found to differ in persons with and without lens opacities."

Reviewing these reports, Sommer[6] concluded that:

> The best available evidence indicates that diabetics are at no greater risk of developing cataracts, only of having them removed. The traditional explanation for this paradox, repeated frequently enough to have become an accepted fact, is that cataracts mature more quickly in diabetics than in the general population. But another plausible explanation exists: diabetics are more likely to be seen by internists and referred to ophthalmologists, and, therefore, more likely to have their cataracts identified and removed.

In order to shed light on the hypothesis proposed by Sommer, it is necessary to study the association between cataract and diabetes in the general population, not in a clinical setting. To avoid selection bias, the studies must not be limited to persons with aphakia but should also include those whose cataracts have not yet been removed.

We examined the association between senile lens changes and diabetes in data from two separate general population studies: those who participated in the Framingham Eye Study and those included in the Health and Nutrition Examination Survey.

Material and Methods

Framingham Eye Study—The first of our two data sources was the Framingham Eye Study,[7-9] a survey conducted from 1973 to 1975 of the surviving members of the Framingham Heart Study. This population had been examined periodically by physicians from the National Heart, Lung, and Blood Institute since 1948.

The lens examinations, carried out by 15 residents in ophthalmology (97.5% of the examinations were done by eight examiners) and performed as part of the screening examination protocol,[9] included dilation of the pupil, slit-lamp biomicroscopy, and direct ophthalmoscopy. Subjects with aphakia or with best corrected visual acuities of 6/9 (20/30) or worse in eyes with definite or possible opacities were referred for a cataract examination, performed at a later date by a more experienced physician who designated as senile all cases of cataract that could not be ascribed to congenital, secondary, or other specific causes. This examiner was also asked to record whether the opacity could entirely account for the visual acuity deficit. Of the 431 persons referred for cataract examinations, 349 (81%) actually completed one. Of the 2,675 Framingham subjects, 2,670 were white; 2,477 were still living in the Framingham area. The examiner found either senile aphakia or, after adequate lens examination, definite senile lens changes in at least one eye in 1,487 subjects. We designated these 1,487 cases as senile lens change. Another 830 individuals (about one half as many) had no senile lens changes and constituted a control group. We excluded 358 persons because we could not be certain that both eyes were free of either senile lens change or senile aphakia; in these individuals we could not definitely establish the absence of senile lens change because (1) a lens was not examined; (2) adequacy

of the lens examination was not specified; or (3) one of the following was present: congenital, traumatic, or secondary lens change; aphakia of nonsenile origin; or questionable or unknown lens change.

CLASSIFICATION OF SENILE LENS CHANGES—In our definition of senile lens changes, we included aphakia, early or late cortical changes, early or late nuclear sclerotic changes, early or late subcapsular changes, and other senile lens changes such as the coronary type of opacity.

We classified senile lens changes as aphakic, cataractous, or precataractous.

1. Aphakia: Aphakia that could not be ascribed to congenital, secondary, or other causes. The term aphakia referred to any lens that was operated upon, even if lens remnants were present, and included nontraumatically subluxated or dislocated lenses. Unless otherwise specified, we combined cases of senile aphakia with cases of senile cataract.

2. Cataract: A senile lens change which "could entirely account for a visual acuity deficit of 6/9 (20/30) or more." (This differs from the more common Framingham definition: lens changes in the presence of visual acuities of 6/9 (20/30) or worse.[7-9])

3. Precataract: This term comprised senile lens changes other than those included under aphakia or cataract. This category included the 82 persons who were referred for cataract examinations but did not complete one.

We classified persons with senile lens change according to the eye with the more advanced change; for this purpose we considered cataract as intermediate between aphakia and precataract.

DIABETES—Persons were classified as having diabetes if a diagnosis of diabetes mellitus had been established during Framingham Heart Study examinations 1 to 10 or 12. Data were not available from the 11th examination; the 12th examination took place at about the time of the Framingham Eye Study (1972-1973). Diagnosis of diabetes at examinations 1 to 10 was based on any of the following: a history of treatment for diabetes; abnormal results for a glucose tolerance test (a challenge of 100 g of glucose after 12 hours of fasting, resulting in a serum glucose concentration of 160 mg/100 ml or more at one hour, of 140 or more at two hours, and of higher than baseline at three hours); or a serum glucose concentration of 150 mg/100 ml or more for two consecutive Framingham Heart Study examinations.[10] Diagnosis at examination 12 was based on the examining physician's clinical impression of the presence of diabetes mellitus.[9,10]

Health and Nutrition Examination Survey—The second data source was the National Health and Nutrition Examination Survey (HANES) of the National Center for Health Statistics, which included ocular examinations for 10,127 persons aged 4 to 74 years, performed by 91 ophthalmologists (mostly ophthalmology residents) at mobile clinics in 35 geographic areas in the United States between April 1971 and October 1972. The data were from a national probability sample of the civilian, noninstitutionalized population and were weighted more heavily toward low-income groups, older groups, preschool children, and women of child-bearing age.[11]

After dilation of the pupil, the lens was examined with both the slit lamp and the direct ophthalmoscope.[12,13] Lens changes (except decreased lucency of the nucleus) were recorded only if they were observed with both instruments.

Our study population, limited to persons aged 50 to 74 years, consisted of 2,647 subjects of whom 2,028 (77%) were white. There were 1,234 with senile lens changes and 1,413 without such changes. We excluded 17 persons with congenital, traumatic, or secondary lens changes, one person who had pseudoexfoliation of

the lens without other senile changes, and four persons for whom no diabetic history was available. The presence of diabetes was defined by an affirmative response to the question as to whether a doctor had ever told the subject that he or she had diabetes.

We classified senile lens changes as aphakic, cataractous, or precataractous.

1. Aphakia: Aphakia that was not characterized as congenital, traumatic, or secondary. Aphakia is the optical condition in which the lens, after attempted surgical removal, is not in the visual axis and rays of light are refracted solely by the cornea. We regarded eyes with lens remnants as aphakic.

2. Cataract: Senile lens changes (cortical, nuclear, or posterior-subcapsular) consistent with best corrected visual acuities of 6/9 (20/30) or worse. (This differs from the definition used by the HANES survey: lens change consistent or associated with visual acuities of 6/7.5 [20/25] or worse.)

3. Precataract. This term comprises senile lens changes other than those included under aphakia or cataract.

We classified persons with senile lens change according to the eye with the more advanced change. We considered cataract as intermediate between aphakia and precataract.

RELATIVE RISK AND ODDS RATIO—We used two closely related measures of strength of association between lens change and diabetes: the relative risk and the odds ratio.[14-16] We obtained these by dividing the subjects into four groups: A, individuals with senile lens change and diabetes; B, individuals with senile lens change without diabetes; C, individuals with diabetes without senile lens change; and D, individuals with neither diabetes nor senile lens change.

The relative risk is the risk of having senile lens change for persons with diabetes relative to the risk for persons without diabetes:

$$\text{Relative risk} = \frac{A/(A + C)}{B/(B + D)}$$

The odds ratio is the odds of senile lens change to no senile lens change among persons with diabetes relative to those without diabetes:

$$\text{Odds ratio} = \frac{A/C}{B/D} = \frac{AD}{BC}$$

A relative risk greater than 1.00 is a direct measure of excess prevalence of senile lens change in diabetics. Thus, a relative risk of 1.40 indicates an excess prevalence of senile lens change in diabetics of 40%. A relative risk of 1.00 indicates an equal prevalence of senile lens change in diabetics and nondiabetics. A relative risk of less than 1.00 indicates a lower prevalence of lens change in diabetics. The relative risk is greater than, equal to, or less than 1.00 as the odds ratio is greater than, equal to, or less than 1.00.

We used the methods described by Gart[17] and Thomas[18] to construct confidence limits and perform significance tests, including tests for interaction, on odds ratios. A test on the odds ratio is equivalent to a test on the relative risk. When a 95% confidence interval excludes unity, then the odds ratio is statistically significantly different from unity at the 5% level. (Because approximations were used, this relationship did not always hold exactly.)

RESULTS

We compared affected individuals and controls from the Framingham and the HANES studies for a number of factors (Tables 1 and 2). The Framingham population (aged 52 to 85 years) was older than the HANES population (aged 50 to 74 years).

At comparable ages in the two populations, the percentage with any lens

TABLE 1

AGE, LENS CHANGES, AND DIABETIC STATUS OF THE TWO STUDY POPULATIONS

Lens change	Framingham				HANES			
	With Diabetes		Without Diabetes		With Diabetes		Without Diabetes	
	No.	%	No.	%	No.	%	No.	%
	Subjects 50 to 64 Years Old*							
All persons	97	100.0	1,091	100.0	79	100.0	930	100.0
No senile lens change	36	37.1	593	54.4	52	65.8	678	72.9
Any senile lens change	61	62.9	498	45.6	27	34.2	252	27.1
Aphakia	3	3.1	14	1.3	2	2.5	8	0.9
Cataract†	4	4.1	11	1.0	8	10.1	31	3.3
Precataract	54	55.7	473	43.4	17	21.5	213	22.9
	Subjects 65 to 74 Years Old							
All persons	87	100.0	666	100.0	162	100.0	1,476	100.0
No senile lens change	19	21.8	151	22.7	48	29.6	635	43.0
Any senile lens change	68	78.2	515	77.3	114	70.4	841	57.0
Aphakia	2	2.3	13	2.0	13	8.0	47	3.2
Cataract†	6	6.9	49	7.4	52	32.1	299	20.3
Precataract	60	69.0	453	68.0	49	30.2	495	33.5
	Subjects 75 to 85 Years Old							
All persons	49	100.0	327	100.0	—	—	—	—
No senile lens change	4	8.2	27	8.3	—	—	—	—
Any senile lens change	45	91.8	300	91.7	—	—	—	—
Aphakia	4	8.2	30	9.2	—	—	—	—
Cataract†	13	26.5	72	22.0	—	—	—	—
Precataract	28	57.1	198	60.5	—	—	—	—

*The youngest Framingham subject was 52 years old.
†Excluding aphakia.

change was considerably higher for the Framingham group (Table 2). For example, in the group aged 50 to 64 years, 47.1% of the Framingham group and only 27.7% of the HANES group had lens changes. In the group aged 65 to 74 years, the corresponding percentages were 77.4 and 58.3. The percentage with cataracts (including aphakia), conversely, was less in the Framingham group than in

TABLE 2

SELECTED SUBGROUPS OF THE TWO STUDY POPULATIONS

Subgroup	Age 50 to 64 Years				Age 65 to 74 Years				Age 75 to 85 Years (Framingham)	
	Framingham		HANES		Framingham		HANES			
	No.	%	No.	%	No.	%	No.	%	No.	%
All subjects	1,188	100.0	1,009	100.0	753	100.0	1,638	100.0	376	100.0
Men	522	43.9	482	47.8	300	39.8	805	49.1	150	39.9
With diabetes	97	8.2	79	7.8	87	11.6	162	9.9	49	13.0
With any senile lens change	559	47.1	279	27.7	583	77.4	955	58.3	345	91.8
Aphakia	17	1.4	10	1.0	15	2.0	60	3.7	34	9.0
Cataract*	15	1.3	39	3.9	55	7.3	351	21.4	85	22.6
Precataract	527	44.4	230	22.8	513	68.1	544	33.2	226	60.1

*Excluding aphakia.

the HANES group: 2.7% vs 4.9% in the group aged 50 to 64 years and 9.3% vs 25.1% in the group aged 65 to 74 years. The percentages with diabetes were similar in the two groups (8.2% of those aged 50 to 64 years and 11.6% of those aged 65 to 74 years in the Framingham group and 7.8% and 9.9% in the HANES group).

The percentages with diabetes, lens change, or cataract in Table 2 should not be taken as unbiased estimates of population prevalence because (1) we excluded 358 persons from the Framingham data because we were uncertain of their lens change status, and (2) to get appropriate prevalence estimates for the HANES group (a probability sample of the United States population in which several different sampling ratios were used), we would have to weight the data according to the relevant demographic variables.

In the following analyses we combined cases of aphakia (because the number was too small for separate study) with cases of cataract.

Principal findings—In the Framingham Eye Study senile lens change was more common in diabetics than in nondiabetics for each five-year age group up to age 69, and thereafter lens changes appeared with equal frequency in the two groups (Figure). In the HANES group, lens change was more common in diabetics than nondiabetics for all ages except for the group aged 60 to 64 years, for whom lens changes were slightly more common in nondiabetics. The number of cases in the HANES study, particularly among

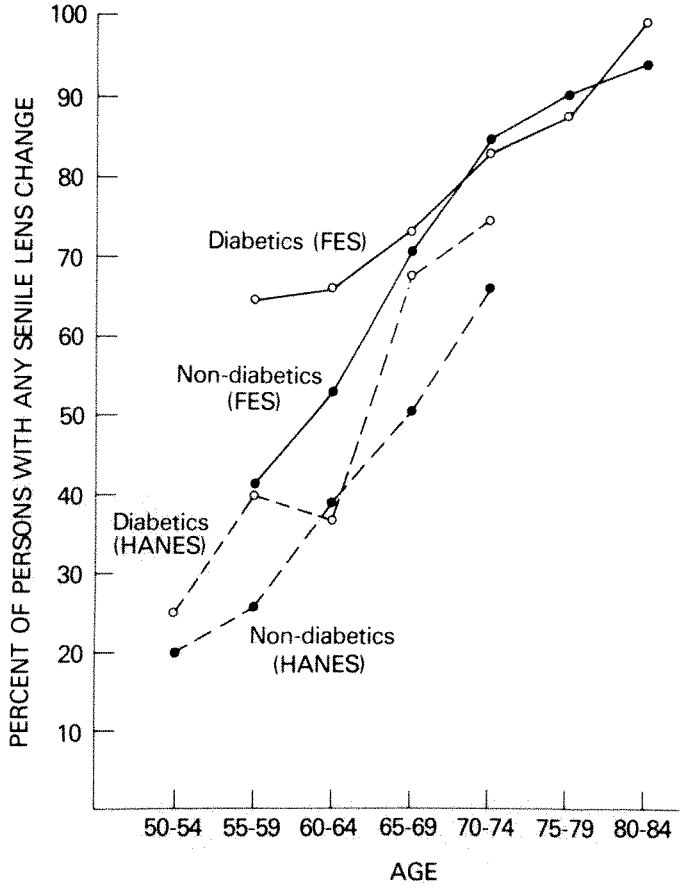


Figure (Ederer, Hiller, and Taylor). Percentage of individuals with senile lens changes in one or both eyes. FES, Framingham Eye Study; HANES, Health and Nutrition Examination Survey.

diabetics, was small within five-year age groups, and, therefore, the sampling error was large. Thus, for the group aged 60 to 64 years, 95% confidence limits on the percentage of diabetics with lens changes were 20% and 56%.[19]

The results presented in terms of relative risks and odds ratios are more reliable statistically because we used broader age groups (Table 3). Because we found no evidence of a statistically significant difference in the association of lens change and diabetes between whites and non-whites in the HANES group (Table 4), the Figure and Table 3 show combined results for the racial groups.

For the group aged 50 to 64 years, the Framingham study disclosed a highly statistically significant association between diabetes and the following senile lens change categories: any lens change, cataract (including aphakia), and precataract. The relative risks were 1.38, 4.02, and 1.35, indicating excess prevalences in diabetics of 38%, 302%, and 35%, respectively. The corresponding odds ratios are 2.0, 4.6, and 1.9. For the HANES data, only senile cataract showed a highly significant association with diabetes. The relative risks were 1.26, 2.97, and 1.03, respectively.

For the group aged 65 to 74 years, HANES data showed significant associations for any lens change and for cataract (relative risks of 1.24 and 1.63, respectively) but not for precataractous lens changes (relative risk of 1.15), whereas Framingham data showed no association for any of the categories of lens change (relative risks of 1.01, 1.02, and 1.01, respectively).

For the oldest age group, those 75 to 85 years old, Framingham data again showed no association (1.00, 1.02, and 0.99, respectively).

There were generally similar results for men and women (Table 3); interaction tests (not shown) disclosed no evidence of a statistically significant sex difference in any age group.

Discussion

Before discussing in detail the findings from the two studies, we should compare them and assess the quality of the information gathered.

Scope and quality—The Framingham study was limited mainly to four eye diseases, whereas the HANES study encompassed the whole spectrum of ocular abnormalities. Also, more time was available for planning and protocol development in the Framingham study than in the HANES study. For these reasons the Framingham lens and cataract examinations were more detailed, as shown by examination protocols covering a total of 12 typewritten pages, compared with only two pages for the HANES study.

The Framingham study was carried out in one location compared with 35 for the HANES study. The Framingham study used only 15 persons to examine lenses in the screening examination and eight for the definitive cataract examination, whereas the HANES study used 91 examiners for the lens-cataract examinations. The Framingham screening examiners and the HANES examiners had about the same level of experience (they were first-, second-, and third-year ophthalmology residents), whereas the definitive cataract examiners in the Framingham study were more experienced (third-year residents and staff members). Both studies included replicate ocular examinations by more experienced examiners, but the percentage of such examinations was higher in the Framingham study (7.4% of screening examinations)[20] than in the HANES study (2.5%).[13] These differences indicate that the diagnosis of cataract was probably made with more accuracy and consistency at Framingham.

Examination procedures and diagnosis of early lens change—In both studies the

TABLE 3

RELATIVE RISKS AND ODDS RATIOS FOR LENS CHANGES AND DIABETES BY AGE AND SEX*

	Men and Women				Men				Women			
	Framingham		HANES		Framingham		HANES		Framingham		HANES	
Lens Changes	Relative Risk	Odds Ratio†	Relative Risk	Odds Ratio†	Relative Risk	Odds Ratio†	Relative Risk	Odds Ratio†	Relative Risk	Odds Ratio†	Relative Risk	Odds Ratio†
					Subjects 50 to 64 Years Old							
Any senile lens change	1.38‡	2.0‡(1.3-3.2)	1.26	1.4 (0.8-2.3)	1.44§	2.0§(1.1-3.9)	1.59§	2.0§(0.9-4.4)	1.37§	2.2§(1.1-4.3)	1.03	1.0 (0.5-2.1)
Cataract or aphakia	4.02‡	4.6‡(1.7-12.2)	2.97‡	3.3‡(1.5-7.4)	5.17‡	6.0‡(1.4-23.8)	2.75§	3.2§(1.0-10.1)	3.41	3.9 (0.8-16.5)	3.96‡	4.4‡(1.3-14.6)
Precataract	1.35‡	1.9‡(1.2-3.0)	1.03	1.0 (0.6-1.9)	1.39§	1.9§(1.0-3.6)	1.46	1.7 (0.7-4.1)	1.36§	2.1§(1.0-4.1)	0.76	0.7 (0.3-1.6)
					Subjects 65 to 74 Years Old							
Any senile lens change	1.01	1.0 (0.6-1.9)	1.24‡	1.8‡(1.2-2.6)	1.09	1.5 (0.6-3.4)	1.18	1.5 (0.9-2.5)	0.95	0.8 (0.4-1.8)	1.27§	2.2§(1.3-3.8)
Cataract or aphakia	1.02	1.0 (0.4-2.6)	1.63‡	2.5‡(1.6-3.8)	1.22	1.3 (0.3-5.3)	1.58‡	2.1‡(1.2-3.9)	0.89	0.9 (0.2-3.2)	1.65‡	2.9‡(1.6-5.3)
Precataract	1.01	1.0 (0.6-1.9)	1.15	1.3 (0.8-2.0)	1.10	1.5 (0.6-3.5)	1.03	1.0 (0.6-2.0)	0.95	0.8 (0.4-1.9)	1.26	1.6 (0.9-3.1)
					Subjects 75 to 85 Years Old							
Any senile lens change	1.00	1.0 (0.3-3.6)	—	—	1.02	1.2 (0.2-8.1)	—	—	0.99	0.9 (0.2-6.0)	—	—
Cataract or aphakia	1.02	1.1 (0.3-4.3)	—	—	0.89	0.7 (0.1-7.1)	—	—	1.02	1.2 (0.2-8.6)	—	—
Precataract	0.99	1.0 (0.3-3.5)	—	—	1.04	1.3 (0.3-9.4)	—	—	0.96	0.7 (0.1-5.0)	—	—

*Individuals without senile lens changes served as controls.
†95% confidence interval.
‡$P < .01$.
§$P < .05$.

TABLE 4

ODDS RATIOS FOR LENS CHANGES AND DIABETES BY RACE IN THE HANES STUDY*

Lens Changes	White (Men and Women) Diabetes Yes	White (Men and Women) Diabetes No	White (Men and Women) Odds Ratio†	Non-White (Men and Women) Diabetes Yes	Non-White (Men and Women) Diabetes No	Non-White (Men and Women) Odds Ratio†	Test for Race Interaction
			Subjects 50 to 64 Years Old				
Any senile lens change	14	175	1.2 (0.6-2.4)	13	77	1.4 (0.6-3.2)	P = .87
Cataract or aphakia	5	31	2.5 (0.8-7.2)	5	8	5.1‡ (1.3-20.2)	P = .41
No senile lens change	36	548	—	16	130	—	—
			Subjects 65 to 74 Years Old				
Any senile lens change	74	604	1.5§ (1.0-2.2)	40	237	4.3‡ (1.4-14.6)	P = .24
Cataract or aphakia	44	243	2.2‡ (1.4-3.5)	21	103	5.2‡ (1.6-18.6)	P = .55
No senile lens change	44	533	—	4	102	—	—

*Individuals without senile lens changes served as controls.
†95% confidence interval.
‡$P < .01$.
§$P < .05$.

lens was examined with a slit lamp and a direct ophthalmoscope, but only the HANES protocol required that the presence of lens opacities be confirmed by both instruments (except for nuclear sclerosis, which could be diagnosed by slit-lamp examination alone). The more stringent HANES diagnostic criteria tended to exclude early lens changes. The diagnosis and documentation of early lens change was emphasized more in the Framingham study than in the HANES study. The Framingham protocol required the recording of both early and late lens changes, and specifically mentioned vacuoles, waterclefts, spokes, and lamellar separations of the anterior cortex. The HANES protocol did not use the terms "early" and "late," and the coding procedures specifically stated that vacuoles were not to be coded. The data in Table 5 are consistent with the differential emphasis on the recording of early lens changes in the two studies: the percentage of subjects with any senile lens change is greater by some 20 percentage points in the Framingham study for each age group. When early cortical changes (vacuoles, waterclefts, spokes, and lamellar separations of the anterior cortex) are excluded from the Framingham results, the percentage with senile lens change was about the same in the two studies. These differences suggest that early lens changes were more likely to have been recorded in the Framingham study than in the HANES study.

Definitions of cataract—In our analysis of Framingham data, we limited cataract to lens changes which "could entirely account for a visual acuity deficit of 6/9 (20/30) or more." This means, for example, that we did not classify a 6/21 (20/70) cataract accompanied by macular disease that decreased visual acuity to 6/60 (20/200) as cataract in our Framingham analysis, but as precataract. In the HANES study, conversely, we would have classified this case as cataract, more conventionally defined as the presence of lens opacity "consistent with visual acuities of 6/9 (20/30) or worse." The lower percentage of cataracts in the Framingham study for the groups aged 50 to 64 years and 65 to 74 years was consistent with the difference in definitions, a differ-

TABLE 5

SPECIFIED LENS CHANGES AND ODDS RATIOS FOR SENILE LENS CHANGE AND DIABETES BY AGE*

Group	Framingham			HANES		
	No.	%	Odds Ratio†	No.	%	Odds Ratio†
			Subjects 50 to 64 Years Old			
All subjects	1,188	100.0	—	1,009	100.0	—
Any senile lens change	559	47.1	2.0‡ (1.3-3.2)	279	27.7	1.4 (0.8-2.3)
Early cortical change	278	23.4	1.5 (0.8-2.6)	—	—	—
Other changes	281	23.7	2.6‡ (1.6-4.3)	—	—	—
			Subjects 65 to 74 Years Old			
All subjects	753	100.0	—	1,638	100.0	—
Any senile lens change	583	77.4	1.0 (0.6-1.9)	955	58.3	1.7‡(1.2-2.6)
Early cortical change	161	21.4	1.0 (0.5-2.1)	—	—	—
Other changes	422	56.0	1.1 (0.6-1.9)	—	—	—

*Individuals without senile lens changes served as controls.
†95% confidence interval.
‡$P < .01$.

ence which may have led to the classification of certain Framingham cases as precataract that would conventionally be considered cataract and would have been classified as cataract by the HANES study. This possible definitional difference may account for a discrepancy between the two studies in relative risks of precataractous lens changes.

Ascertainment bias—Both cataract and diabetes are insidious diseases that often remain asymptomatic and undiagnosed for a long time, leading to possible biases of ascertainment if (1) diabetics are more likely to have their cataracts diagnosed or if (2) persons with cataract are more likely to have their diabetes diagnosed.

Sommer[6] pointed out how the first type of bias could operate: diabetics are more likely to be referred to ophthalmologists and therefore more likely to have their cataracts removed. In both studies this bias was avoided because the ascertainment of cataract was made by routine lens examination of all subjects and separately from the ascertainment of diabetes.

The second type of bias is possible in studies of cataract and diabetes because serum or urine glucose measurements are a routine part of the examination before cataract (and other) operations. Because the ascertainment of diabetes in the present studies was based not on laboratory tests alone but either entirely (HANES) or partly (Framingham) on history of the disease, both studies were liable to this bias, although we do not know to what extent, if any, the bias was operative. However, when we excluded cases of aphakia (the most likely sources of bias), the relative risk for cataract increased for the group aged 50 to 64 years and decreased only slightly for the group aged 65 to 74 years, suggesting that in these studies bias did not play a prominent role. Excluding cases of aphakia, the relative risk for cataract in the group aged 50 to 64 years changed from 4.02 to 5.49 in the Framingham study and from 2.97 to 3.05 in the HANES study; the corresponding changes for the group aged 65 to 74 years were from 1.02 to 0.98 and from 1.63 to 1.62 (derived from Table 1).

We also considered the possibility that the observed association between cataract and diabetes might be caused in part by an association between diabetic retinopathy and visual acuity. We therefore performed a separate analysis that ex-

cluded cases of diabetic retinopathy that, in the opinion of the examiner, affected visual acuity. There was one such case in the group aged 52 to 64 years in the Framingham study, and two and nine such cases, respectively, in the groups aged 50 to 64 years and 65 to 74 years in the HANES study. When these cases are excluded, the three relative risks were reduced but not substantially: 4.02 to 3.53, 2.97 to 2.45, and 1.63 to 1.53.

In our judgment, bias of ascertainment did not account for the observed association between senile cataract and diabetes.

The association between senile lens changes and diabetes—The number of cases of senile cataract or aphakia among diabetics in specific age groups was often small (Table 1) and, consequently, the confidence intervals for the odds ratios were usually large, although several of them clearly excluded unity. When the 95% confidence interval excludes unity, then the odds ratio and relative risk are significantly different from unity at the 5% level.

In persons under the age of 65, both studies showed a marked excess prevalence of senile cataract (including senile aphakia) in diabetics, as shown by relative risks substantially greater than one (4.02 in the Framingham study and 2.97 in the HANES study). For precataract there appeared to be less agreement, although the difference was not large: the Framingham study showed a statistically significant excess in diabetics (relative risk of 1.35), while the HANES study showed the same prevalence in diabetics and nondiabetics (relative risk of 1.03).

In persons aged 65 to 74 years, the results appeared to differ somewhat for cataract but not for precataract. For cataract, the HANES study found a small excess prevalence (relative risk of 1.63), but the Framingham study did not (relative risk of 1.02). For precataract the relative risks were 1.01 and 1.15 for the Framingham and HANES studies, respectively.

According to an interaction test, the difference between the Framingham (1.35) and HANES (1.03) relative risks for precataract in the group aged 50 to 64 years was not statistically significant at conventional significance levels ($P = .17$). However, if this difference was not caused by chance alone, then it might be explained by the different definitions used for cataract or diabetes. We believe that chance or definitional variations accounted for the apparent discord in precataract. Both studies, however, agreed that there is a marked excess prevalence of senile cataract among diabetics in this age group.

The second apparent difference in results between the two studies was for senile cataract in the group aged 65 to 74 years. Again, an interaction test found that the difference in relative risks (1.02 for the Framingham study and 1.63 for the HANES study) was not statistically significant ($P = .27$). If the difference was not caused by chance alone, the more accurate and consistent diagnostic methods at Framingham and the definitional differences might have been contributing factors. The two studies agreed that, in this age group, the association with diabetes is at most small for cataract, and does not exist for precataract. The Framingham results for the group aged 75 to 85 years were essentially the same as for the group aged 65 to 74 years.

In summary, the two studies agreed on a marked excess prevalence of senile cataract in diabetics up to the age of 64 years. The Framingham study ($P = .03$) but not the HANES study ($P = .64$) showed a significant reduction in this excess at older ages (age 65 to 85 years for the Framingham study and age 65 to 74 years for the HANES study). They also agreed that there is little, if any, excess of

precataractous lens change for diabetics up to the age of 64 years and that there is none thereafter.

Previous studies—We compared the findings of previous studies, done at Oxford[1,2] and in the United States,[3] that looked for associations between cataract extraction and diabetes with our studies of cataract (whether removed or not) (Table 6). For comparability with the available data from the other studies, we classified our data by age groups 50 to 69 years and 70 years and older. We included our results for aphakia alone and for cataract (which includes aphakia). Waite and Beetham[4] and McGuinness[5] studied lens changes but did not report their results in a form suitable for inclusion in Table 6.

The study at Oxford was of 454 patients who had had cataracts extracted at the Oxford Eye Hospital while living in a defined surrounding area.[2] The number of diabetics without cataracts was estimated from the percent population prevalences of diabetes determined in a previous survey. The odds ratios of 7.9 and 4.3 for the two age groups are the highest in

TABLE 6

RESULTS OF HOSPITAL AND POPULATION SURVEYS

Studies	Men and Woman 50 to 69 Years Old			Men and Women 70 or More Years Old		
	Diabetes			Diabetes		
	Yes	No	Odds Ratio*	Yes	No	Odds Ratio*
Hospital Studies						
Oxford, England[2]						
Cataract (senile or unspecified)	25	262	7.9†	35	491	4.3†
Control	642	53,331	—	279	16,775	—
U.S. Hospital Discharge Survey[3]						
Cataract (senile or unspecified)	55	552	—	52	813	—
Fractures, sprains, and strains	84	1,927	2.3§ (1.6-3.3)	102	1,712	1.1 (0.7-1.5)
Hemorrhoids	12	251	2.1‖ (1.1-4.2)	8	67	0.5 (0.2-1.3)
Varicose veins	11	396	3.6§ (1.8-7.4)	5	74	0.9 (0.3-2.8)
Washington Hospital Center, U.S.[3‡]						
Cataract (senile or unspecified)	84	617	1.8‖ (1.1-3.0)	66	739	1.0 (0.6-1.7)
Fractures, sprains, and strains	22	291	—	21	235	—
Population Surveys						
Framingham Eye Study						
Senile cataract or aphakia	11	51	3.1§ (1.4-6.7)	22	138	1.0 (0.5-2.4)
Aphakia	5	18	4.0§ (1.2-12.2)	4	39	0.7 (0.2-2.5)
No senile lens change	48	699	—	11	72	—
Health and Nutrition Examination Survey (HANES)						
Senile cataract or aphakia	45	182	3.2§ (2.1-4.9)	30	203	1.9‖(1.0-3.8)
Aphakia	11	23	6.3§ (2.8-14.1)	4	32	1.6 (0.4-5.7)
No senile lens change	84	1,104	—	16	209	—

*95% confidence interval.
†We did not determine the significance level or the 95% confidence interval because the number of diabetics in the control group was not known but only estimated from the percent prevalence obtained in a previous survey.
‡Odds ratios for the Washington Hospital Center are shown opposite controls rather than cases.
§$P < .01$.
‖$P < .05$

Table 6, but are based on estimates of diabetes prevalence in the control group of only 1.2% and 1.8%, percentages that have been criticized as being understated.[21] Hiller and Kahn[3] said that if this criticism is valid, the Oxford estimates overstate the odds ratios, which "may, in fact, not be essentially different from ours."

Hiller and Kahn used hospital discharge data from a national sample survey and a local hospital to study the association between cataract extraction and diabetes.[3] In both data sets they found a positive association for the group aged 50 to 69 years, as we did, although their odds ratios were somewhat lower (P = .04) than ours for aphakia alone. For the group aged 70 years and older, their finding of no association is compatible with that for the Framingham study and not strongly incompatible with that for the HANES study.

In 1935, Waite and Beetham[4] looked at the association between diabetes and lens changes. They examined 2,002 diabetic patients from the New England Deaconess Hospital and 457 nondiabetic patients from Massachusetts General Hospital, classifying lens changes first anatomically and then descriptively. From the published report it was impossible to organize the results according to the format of Table 6, either by severity of lens change or by age and, therefore, we could not determine whether the findings were compatible with an excess prevalence of cataract among diabetics under the age of 70 years.

McGuinness'[5] study was of a sample of 340 persons aged 40 years or more from the general population of Rhondda Fach in Wales. The lenses were examined with a slit lamp and blood was taken for a fasting serum glucose determination. Although 180 persons were found to have lens opacities, only ten had cataract (aphakia or mature lens opacity). From the published data, presented in terms of mean fasting serum glucose concentrations, we were able to determine, by the direct method,[22] age- and sex-adjusted means of 120.7 and 91.3 mg/100 ml, respectively, for the ten persons with cataract and the 95 without lens opacities (P = .05). In these calculations, we used data only from the six age-sex categories including at least one patient with reported serum glucose values among both those with cataract and those without lens opacities. In determining the adjusted means, we used as weights the number of cases in the six age-sex categories for those without lens opacities. Because standard deviations were not available for five of the six cataract age-sex categories, we used those for the "no lens opacity" group as estimates. Although we found a higher age- and sex-adjusted serum glucose concentration in persons with cataract in McGuiness'[5] study, this may not be statistically significant because of the uncertainty of our estimates. It does suggest that McGuiness'[5] results may not be at variance with those shown in Table 6.

To summarize, three studies of cataract extraction and two studies including present and extracted cataracts agreed that diabetics under the age of 70 years have an excess prevalence of senile cataract. All five studies found the excess to be less or nonexistent in diabetics aged 70 years or more. Two additional studies found no association between lens opacity and diabetes, but these studies provided information inadequate to support or refute the foregoing findings on cataract in persons less than 70 years old. Sommer's hypothesis is not supported by these various studies in that they found that cataract, whether removed or not, is associated with diabetes in persons under the age of 70 years.

We offer two possible explanations, not mutually exclusive and both entailing competing risks, for the decrease in relative risk with age. (1) The risk of both cataract and death are positively associated with severity of diabetes, and the proportion of diabetics with severe disease, because of their higher mortality, diminishes with age. Because of the smaller proportion of diabetics with severe disease, the cataract risk is reduced in older persons. (2) Senile cataract has both diabetic and nondiabetic causes, and the risk from nondiabetic (senile) causes increases with age more rapidly than the risk from diabetes. At younger ages, the risk from nondiabetic causes is smaller than the diabetes risk, producing a large relative risk for diabetes, while at older ages the risk from other causes dominates the diabetes risk. Decreasing relative risks with age have also been noted in the relationship of blood pressure, serum cholesterol, and cigarette smoking to coronary heart disease[10] and of diabetes to death from coronary heart disease.[23]

Note—The Framingham Eye Study, a collaborative effort of the National Eye Institute, the Boston University Department of Ophthalmology, and the Harvard University Department of Social and Preventive Medicine, was supported in part by contract NIH-NEI-72-2112. The ophthalmic components of the Health and Nutrition Examination Survey were a collaboration of the Division of Health Examination Statistics, National Center for Health Statistics, Department of Health, Education, and Welfare, and the National Eye Institute.

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OPHTHALMIC MINIATURE

Even her new spectacles didn't seem to do any good. And that, she reflected, was because obviously there came a time when oculists, in spite of their luxurious waiting room, their up-to-date instruments, the bright lights they flashed into your eyes, and the very high fees they charged, couldn't do anything much more for you. Miss Marple reflected with some nostalgia on how good her eyesight had been a few (well, perhaps not a few) years ago.

Agatha Christie, *The Mirror Crack'd*

CICATRICIAL ECTROPION AND LACRIMAL OBSTRUCTION ASSOCIATED WITH THE SCLERODERMOID VARIANT OF PORPHYRIA CUTANEA TARDA

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A patient with porphyria cutanea tarda developed bilateral cicatricial ectropion with stenosis of the puncta and canaliculi. The pathophysiologic findings suggested a photosensitivity reaction in sites of uroporphyrin and 7-carboxyl porphyrin deposition.

Porphyria cutanea tarda is a disorder of porphyrin metabolism in which uroporphyrin and 7-carboxyl porphyrin are found in abnormally increased quantities in the urine.[1,2] Increased blood levels of these porphyrins appear to produce photosensitization, resulting in blistering, mechanical fragility, scarring, hypertrichosis, and scleroderma-like changes in sun-exposed skin.[1,3] Ocular abnormalities in porphyria include changes in the eyelids, conjunctiva, sclera, cornea, and intraocular structures. In the patient described here, bilateral cicatricial ectropion and obstruction of the lacrimal excretory apparatus were associated with porphyria cutanea tarda and were the clinical basis for suggesting the diagnosis of this disorder.

CASE REPORT

A 70-year-old white woman had had bilateral epiphora since 1977; there was no mucous discharge or periocular infection. The skin of the lower eyelids had progressively thickened since 1976, with mild eversion and ectropion of the lower eyelid margins. In 1977, she had undergone a right lateral canthoplasty but there was little improvement in the eyelid position. Bilateral obstruction of the lacrimal excretory pathways was noted in 1978, at which time irrigation of saline into the nose was attempted without success. In 1974, cataracts had been removed from both eyes with no complications. Visual acuity was reduced in the right eye because of a macular scar. She had no history of glaucoma, and had used no ocular medications except for a brief period after the cataract extractions.

She had been unaware of any problems with her skin until 1969 when she had developed a severe blistering eruption on her face, upper chest, upper back, lateral arms, and lower legs (sun-exposed surfaces) after relatively intense sun exposure in Florida. She was treated with soaks and triamcinolone cream. Although her physician did not make a specific diagnosis, it was suggested that this problem might be caused by sun exposure. The eruption healed with increased and decreased pigmentation, scarring, and stiffness of the skin (Figs. 1 and 2). Since that time, she has minimized sun exposure and suffered no recurrence of the blistering, although her skin has become increasingly fragile. She took no systemic medications except phenytoin sodium, 100 mg three times a day, because of "fainting" since childhood. She denied using estrogens or alcohol and recalled no exposure to fungicides. There was no family history of similar skin disorders.

Our examination disclosed a visual acuity of R.E.: 6/60 (20/200) and L.E.: 6/12 (20/40) with aphakic corrections. A macular scar was present in the right eye. No significant abnormalities of the corneas or scleras were apparent. Tear formation (Schirmer strips without anesthesia) was 7 mm of wetting in 5 minutes bilaterally. Both lower eyelids were slightly everted from the eyes, with a somewhat greater ectropion laterally than medially (Fig. 3). The skin of the lower eyelids was thick and tight. The periorbital area was faintly violet. Although the conjunctiva near

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Fig. 1 (Sober, Grove, and Muhlbauer). Sclerotic skin, hyperpigmentation, and hypopigmentation in sun-exposed areas compatible with the sclerodermoid type of porphyria cutanea tarda.



Fig. 2 (Sober, Grove, and Muhlbauer). Changes similar to those in Figure 1 on the lateral aspects of the upper arm (sun-exposed). There are no changes on the medial aspects (non-exposed).

the everted eyelid margins was dry, the conjunctiva covering the eyes and the fornices did not appear abnormal. The right upper and lower puncta and the left upper punctum were occluded and could not be observed. The left lower punctum was stenosed and was enlarged with a dilator. After dilation, a probe passed 3 mm into the canaliculus at which point a total obstruction was met.

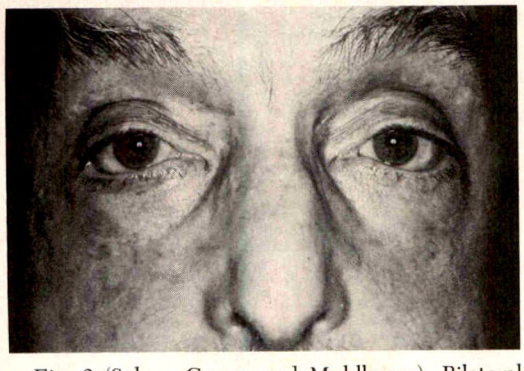


Fig. 3 (Sober, Grove, and Muhlbauer). Bilateral ectropion (right greater than left). Skin of the lower eyelids is sclerotic.

Extraocular examination disclosed firm sclerotic skin on the forehead, sides of the face, lateral arms, upper chest, back, and lower legs (Figs. 1 and 2). Areas of hyperpigmentation and hypopigmentation were present. Increased wrinkling was noted about the mouth (rhagades). Increased vellus hairs were present on the cheeks and increased coarse hairs about the mouth and chin.

Laboratory tests gave normal results for a complete blood cell count and serum iron, iron-binding capacity, and liver function tests. Serologic tests for lupus erythematosus were also negative. Porphyrin levels in urine and fecal specimens were distinctly abnormal (Table) and characteristic of porphyria cutanea tarda. A biopsy specimen of skin from the right temple adjacent to the right eye disclosed dermal sclerosis with hyalinization of the dermal venular walls (Fig. 4). Dermal collagen was quite homogenized, with focal thickening of the epidermal and vascular basement membrane zones. Basal layer hyperpigmentation was also observed. Direct immunofluorescent study of the biopsy specimen showed homogeneous deposition of IgG, IgA, IgM, and albumin of the epidermal basement membrane, the dermal venules, and the dermal connective tissue, consistent with porphyria cutanea tarda.

TABLE

RESULTS OF PORPHYRIN STUDIES*

Porphyrin Fraction	Urine (μg/day)†	Feces (μg/g of dry weight)†
Uroporphyrin	1,350 (0-40)	Not measured
Coproporphyrin	215 (0-280)	155 (0-50)
Protoporphyrin	—	70 (0-50)

*The Watson-Schwartz test on a urine specimen was negative.

†Normal values are given in parentheses.



Fig. 4 (Sober, Grove, and Muhlbauer). Skin biopsy specimen from area on right temple illustrates early bulla formation (arrowhead, separation at the dermo-epidermal junction), sclerosis of collagen, and thickening of capillary basement membranes (double arrowhead) (hematoxylin and eosin, ×224).

DISCUSSION

Porphyria cutanea tarda is a metabolic disorder of hemoglobin synthesis in which uroporphyrin and 7-carboxyl porphyrin accumulate in the liver and are subsequently excreted into the blood and urine in the symptomatic phase of the disease. These porphyrins absorb radiation in the 380- to 420-nm region of the spectrum and may induce photosensitivity when circulating through the capillaries of the skin.[1] The major clinical manifestations of porphyria cutanea tarda include skin fragility and bullae on sun-exposed skin. Healing of bullae often results in scarring and milia formation. The patient may also observe darkening of the skin, increased hair growth lateral to the eyes, and a violaceus hue in the periorbital area. Occasionally, the affected skin becomes firm and sclerotic, producing plaques clinically similar to scleroderma. Porphyria cutanea tarda has been related to the use of either estrogens or alcohol or to an iron overload state. Many of these patients appear to have a genetic predisposition, defined by a partial deficiency of the enzyme uroporphyrinogen decarboxylase as measured in hepatocytes and sometimes in erythrocytes.[4-6] Hexachlorobenzene, a fungicide, caused an epidemic of poryphyria cutanea tarda in Turkey when inhabitants ate grain contaminated with this substance.[7] Phenytoin sodium has not been reported to provoke porphyria cutanea tarda. Phlebotomy and low-dose oral chloroquine therapy have proven effective in the treatment of porphyria cutanea tarda.[8,9]

Barnes and Boshoff[10] described the ocular findings in 84 patients with porphyria. Of the 84, 46 were non-European subjects who presumably had porphyria cutanea tarda although no laboratory data were cited to confirm this. Two of the 46 patients had ectropion; one of these two also had symblepharon. Twelve of 46 patients had lesions of the ocular adnexa; the most common lesions observed were blepharochalasis, hyaloid thickening of the conjunctiva, and external or internal

scarring of the eyelids. Blepharospasm and conjunctival congestion were also observed in several patients. Eight of the 46 patients had either scleromalacia perforans or corneal scarring. Lacrimal duct obstruction was not reported in this series. One European woman, aged 34, had cataracts.

Sevel and Burger[11] studied a 51-year-old man with confirmed porphyria cutanea tarda who had bilateral scarring of the eyelids, conjunctivas, and corneas. One eye was enucleated because of a corneal perforation. Thin-layer chromatographic analysis of necrotic scleral tissue from this eye demonstrated the presence of uroporphyrin and 7-carboxyl porphyrin. This finding supported the hypothesis that these porphyrins act as chromophores for a photosensitivity reaction, causing vesication, scarring, and ulceration of the sclera or cornea. The enucleated eye also had a cataract.

Chumbley[12] described two additional patients with confirmed porphyria cutanea tarda who had scleral ulcerations. One patient had brawny scleritis, scleromalacia, and cataracts. The other had scleral ulcers that showed pink fluorescence when exposed to ultraviolet light, suggesting porphyrin deposition. In both patients, the scleral ulcers were located in the interpalpebral region of the anterior sclera in the horizontal meridian and extended to continguous corneoscleral limbal and corneal tissue, suggesting that sun-exposed sites were most affected.

To our knowledge, this was the first case of cicatricial ectropion associated with porphyria cutanea tarda confirmed by urinary and fecal studies. The mechanism of ectropion formation in our patient may have been analogous to that proposed for cutaneous and scleral lesions, namely, deposition of a uroporphyrin chromophore in the site with subsequent photosensitive reactions requiring 380- to 420-nm solar radiation. There are no experimental data using eyelid tissue to support this suggestion, however. Our patient also had stenosis of the lacrimal puncta and canaliculi which, to our knowledge, has not been previously described in patients with porphyria cutanea tarda. These lacrimal abnormalities may have been caused by the same process that has produced conjunctival changes in other patients with this disorder.

Ectropion has also been reported in erythropoetic porphyria (Gunther's disease), a rare autosomal recessive disorder in which severe photosensitivity begins in early childhood and leads to mutilating scars of the face and hands, combined with erythrodontia, splenomegaly, and hemolytic anemia.[13] Ocular changes may be prominent and include scleromalacia perforans, keratomalacia, cataracts, and corneal scars in addition to ectropion.[14] An early report by Stokes[15] mentioned eyelid scarring and ectropion in patients diagnosed as having hydroa vacciniforme; some of these individuals appear to have had erythropoietic porphyria.

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OPHTHALMIC MINIATURE

Handel was "couched" (from the French coucher, to lay down) for his cataracts on three occasions; that is, he underwent a needling, with knocking of the lens backwards. The first oculist was Mr. Samuel Sharp of Guy's Hospital in London in 1751, and the second Mr. William Bramfield, surgeon to the Princess of Wales, in 1752. The third oculist was commonly known as "Chevalier" John Taylor from Norwich, who operated on Handel in August, 1758, in Tunbridge Wells. This same Taylor had operated for cataract on J. S. Bach, equally unsuccessfully, in 1749. It was Sharp who asked Handel whether he could not continue to play the organ in public for the performance of his oratorios, even though he was blind, to whom Handel replied with a loud laugh: "Mr. Sharp, have you never read the Scriptures? Do you not remember, if the blind lead the blind, they fall into the ditch?"

Milo Keynes: Handel's Illnesses.
Lancet 2:1355, 1980

NOTES, CASES AND INSTRUMENTS

BENDING OF EXCESSIVELY LONG INTRAOCULAR SUTURE MATERIAL BY ARGON LASER

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Detroit, Michigan

Excessively long nylon or polypropylene sutures can cause corneal edema. Kinking the material back on itself with an argon laser allows it to clear the surface of the cornea. Of seven patients who were successfully treated by this method, none suffered complications.

In recent years, the implantation of some types of intraocular lenses has necessitated the placement of sutures either directly through iris tissue or between the loops of an implant through an iridectomy. These sutures have generally been either nylon or polypropylene and, fortunately, are pigmented.

During the last four years, I have had the opportunity to treat seven eyes in which intraocular sutures had inadvertently been cut too long or had become unraveled within the eye. As a result of the excessive suture length, a bristle of suture material rubbed against the endothelial surface of the cornea, causing a localized corneal edema.

All of these cases (four polypropylene sutures and three nylon sutures) were successfully treated by kinking the suture back on itself with an argon-laser beam.

With the patient under topical anesthesia, a three-mirror gonioscopic lens is inserted onto the eye in normal fashion. Because the black nylon suture seems to absorb the beam better than the blue polypropylene suture does, less power is required. I use about 500 mW for nylon and about 800 mW for polypropylene. A 50-μ spot size is used. While viewing the suture under ×10 magnification, the laser operator draws the beam across the suture near its base. The suture then kinks abruptly, allowing it to clear the posterior corneal surface.

This simple procedure was successful in all seven attempts; there were no adverse reactions. In no case did the suture return to its straight configuration, but in one case it did unbend slightly. Severing the suture with a free-floating end has not been a problem.

This procedure is a simple way of avoiding surgery to correct one perplexing complication of intraocular lens implantation surgery.

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A COUNTER FOR FIXED-FRAME ENDOTHELIAL CELL ANALYSIS

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We developed a simple electronic counter for fixed-frame endothelial cell analysis by modifying an ink cartridge pen and an inexpensive pocket calculator.

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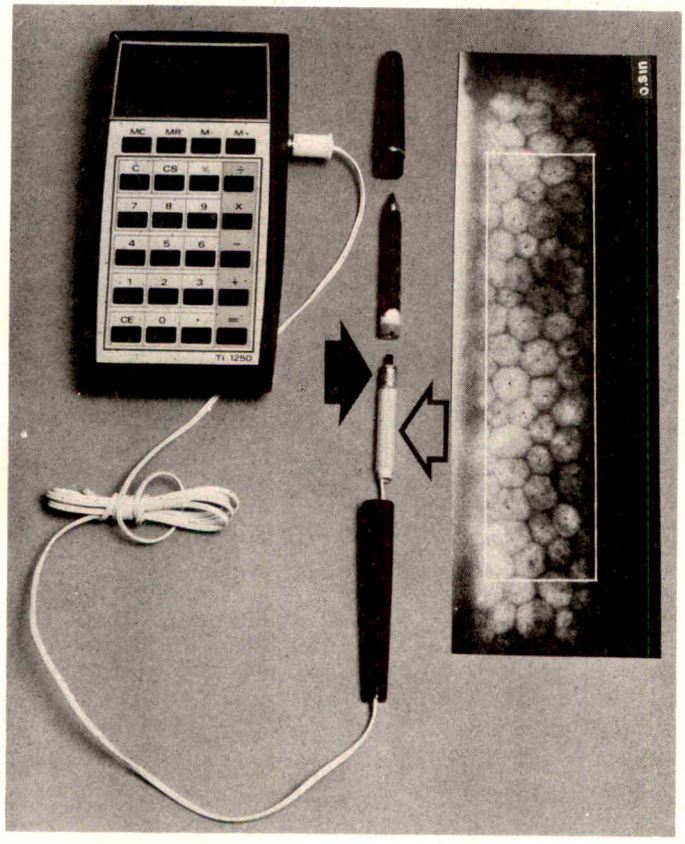


Figure (Alanko and Airaksinen). The pocket calculator and the counting pen. The pen shaft is opened to show the ink cartridge, the switch (solid arrow), and the supporting sleeve (open arrow).

A common method of counting corneal endothelial cells on specular photomicrographs is the fixed-frame cell analysis. With this method, a rectangle of a known area is drawn on the photographs and the cells inside of the frame are counted.[1] To avoid overestimates of the cell density, the cells touching two perpendicular sides of the rectangle are omitted.[2] The number of cells counted is divided by the area of the rectangle and the result is expressed as cell density, that is, cells per square millimeter.[3]

To make the monotonous counting procedure easier, we have devised an electronic cell counter by using an ink cartridge pen, a miniature pushbutton switch, a piece of thin, flexible twin-lead cable, and a pocket calculator with automatic constant and memory capabilities.

We made the following modifications: We opened the shaft of the pen and removed the spare cartridge. We drilled a small hole for the cable on the end of the shaft. The switch soldered to the cable was assembled inside of the shaft with a piece of plastic tube as a supporting sleeve. The length of the tube was trimmed so that the backward motion of the cartridge closes the switch when the tip of the pen is pushed. We connected the cable from the switch to the calculator via a miniature plug and soldered the leads parallel to the equal key on the printed circuit board inside the unit.

The counting procedure—The calculator is turned on and the actual area of the counting rectangle, measured in square millimeters, is stored in the memory. The display is cleared and the sequence

" 1 + = " is entered. The calculator is then ready for counting. Each time the tip of the pen is pressed on the cell to be counted, the switch inside the pen closes and the calculator adds one to the number on the display. The tip of the pen simultaneously marks the cells counted.

When the counting of the cells in one picture is complete, the number on the display is divided by the actual area of the counting rectangle, recalled from the memory. The result is the cell density, that is, the number of cells per square millimeter on the analyzed picture. The counting procedure is repeated for subsequent pictures.

The counting can be done from paper prints or from negatives projected on the screen of a viewer.[3]

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A NEW MOBILE ARMREST

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We have developed a weight-and-pulley armrest for surgeons to use during ocular microsurgery. The armrest is mobile, sterilizable, and allows the weight stress on the forearm to be regulated.

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Using an armrest in ocular microsurgery increases surgical precision and eliminates painful muscle contractions, especially during lengthy operations.

The efficacy of the traditional armrest is unfortunately countered by a loss of mobility for the surgeon. We have overcome this disadvantage with our new mobile armrest which perfectly counterbalances the weight of the surgeon's hands and forearms (Fig. 1). The apparatus includes one fixed component, with two cords and four pulleys and weights, and one component that can be completely gas-sterilized. The latter is made up of two padded sleeves and two cord segments with a fastener at the end of each (Fig. 2).

Before starting surgery, the surgeon

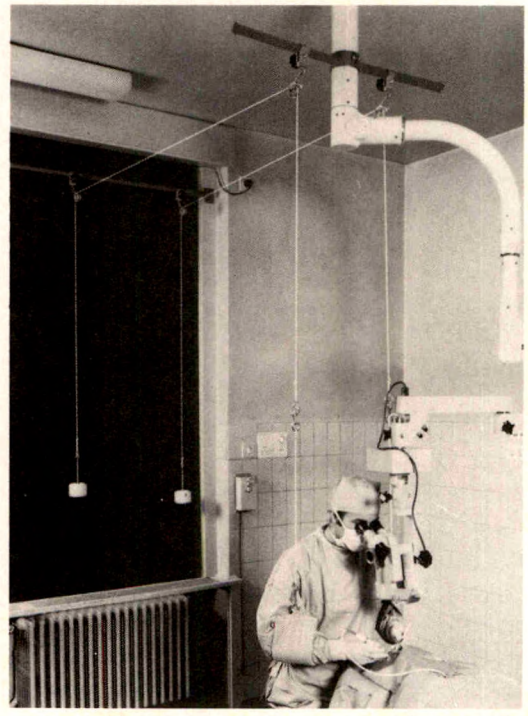


Fig. 1 (Gonvers and Zografos). General view of the operating field. Each of the surgeon's arms passes through a sleeve, suspended by a cord leading to one wall of the operating room by two pulleys. The traction on each sleeve is regulated by changeable weights on the other end of each cord.

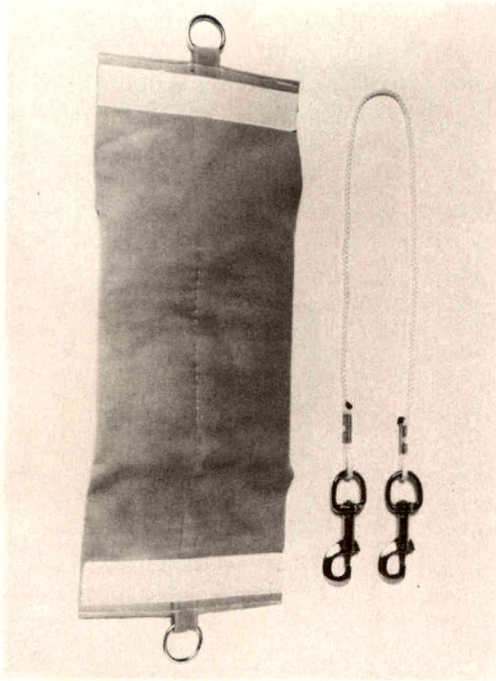


Fig. 2 (Gonvers and Zografos). Details of the gas-sterilized components: padded sleeve with Velcro tape and segment of cord with snap fasteners.

puts his forearms into the sleeves, which he then closes with Velcro tape. He connects the sleeves by a length of sterile cord to a cord suspended from the ceiling. This latter passes through two pulleys and its end is weighted until the surgeon's arm is perfectly counterbalanced (about 2 to 3 kg).

The surgeon feels as though he is working in a weightless environment; this permits great precision in all movements while requiring minimal muscular effort. Because the sterile cord is so long, the surgeon can easily disengage himself from the system at any time if he wants to leave the operating field temporarily.

SYPHILITIC OPTIC PERINEURITIS

JAMES A. RUSH, M.D.,
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We examined a patient who had syphilitic perioptic neuritis, a rarely diagnosed inflammation of the sheaths of the optic nerves that may be confused with papilledema caused by brain tumor or pseudotumor cerebri. The visual acuity was typically normal, and the only visual-field abnormality was enlargement of the blind spots. After we excluded an intracranial mass lesion, the diagnosis was confirmed by cerebrospinal fluid serologic findings positive for syphilis and normal intracranial pressure. Anti-inflammatory and antisyphilitic therapy resolved all the symptoms in our patient.

Syphilitic perioptic neuritis is a bilateral inflammation of the optic nerve sheaths that spares the subjacent optic nerves.[1] Visual acuity is unaffected, but the optic disks are swollen. This condition may be mistakenly diagnosed as papilledema. The accurate diagnosis of syphilitic perioptic neuritis depends upon positive serologic findings and normal cerebrospinal

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fluid pressure. We recently treated a patient with syphilitic perioptic neuritis who responded to therapy.

Case report

A 34-year-old man was referred here on March 31, 1980, for evaluation of bilateral optic disk edema. A moderately severe, right-sided retro-orbital pain had developed one month previously. The frequent recurrences lasted several hours, and subsided spontaneously. Episodes of photopsia, resembling "bright lights and squiggly lines" and lasting a few seconds, had occurred sporadically in his right eye two weeks previously. On several occasions he had noted momentary blurring of the vision of his right eye.

He had had typical, right-sided migraine headaches between the ages of 21 and 28 years, but his current peri-orbital pains were unlike them. He was taking no medication and, despite the headaches, continued to work as an interstate truck driver. He had had frequent heterosexual episodes and, in December 1979, had been treated with nystatin cream for a painless penile lesion. One month later, a brief nonpruritic chest rash had developed, but he had not sought treatment and denied having had syphilis.

Visual acuity was 6/6 (20/20) in both eyes. Pupillomotor responses were brisk bilaterally, confrontation visual fields were full, and color vision was intact. Ocular movements were normal; there was no nystagmus. Optokinetic reflexes were symmetrical. Results of biomicroscopy and applanation tonography were normal bilaterally. Each optic disk was moderately elevated and had indistinct margins. A few peripapillary hemorrhages and dilated retinal veins were evident in each eye. An exudate was present at the 8 o'clock position on the optic disk margin of the right optic nerve (Figure). The retina was otherwise normal, and there were no vitreous cells. Goldmann perimetry and tangent screen disclosed enlarged blind spots bilaterally. Results of a neurologic examination were noncontributory. A hemogram and routine serum chemistry profile were normal. A serum VDRL was positive in a 1:64 titer and an absorbed serum fluorescent-treponemal-antibody absorption (FTA-ABS) test was reactive. Results of a cranial computed tomogram were normal. A clear cerebrospinal fluid specimen was obtained; opening pressure was 140 mm Hg. The total protein was 35 mg/dl. Protein electrophoresis was normal. Cellular analysis was not performed because of a laboratory error. The cerebrospinal fluid VDRL was positive in a 1:4 dilution and a cerebrospinal fluid FTA-ABS test was reactive. An India ink preparation and bacterial and fungal cultures were negative.

A short course of dexamethasone was administered while we were waiting for these results. After receiving the test results, we administered 9.6 million units of intramuscular benzathine penicillin during a four-week period. Six weeks after completion of therapy, the patient's visual acuity was 6/6 (20/20) in both eyes, visual fields were normal, and the optic disks were flat.

Discussion

A patient who has bilateral swollen disks and normal visual acuity is usually thought to have papilledema caused by increased intracranial pressure. If cerebrospinal fluid pressure is normal, the appearance of papilledema in a patient

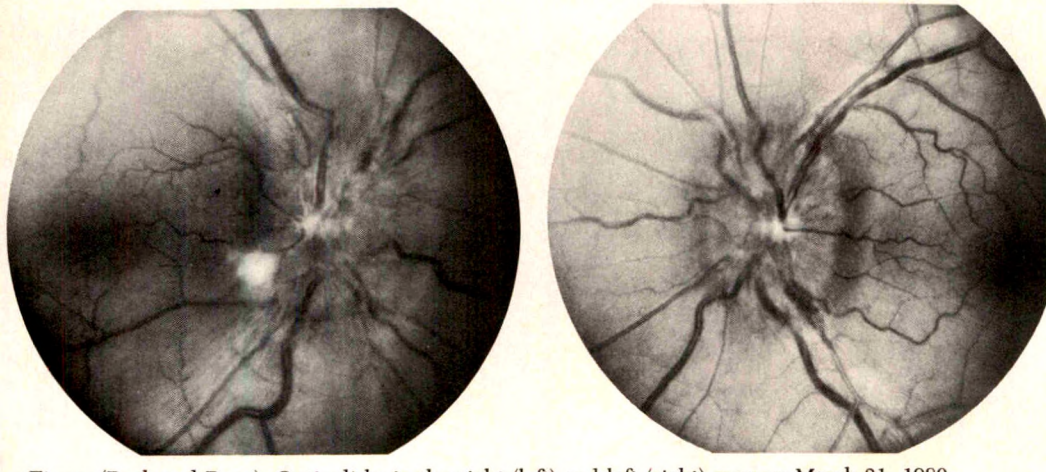


Figure (Rush and Ryan). Optic disks in the right (left) and left (right) eyes on March 31, 1980.

who has normal visual acuity suggests the possibility of perineuritis of the optic nerve.[2]

Optic perineuritis is an inflammation of the meningeal sheaths of the optic nerve and usually occurs as an extension of a diffuse basal meningitis that spares the optic nerve substance. It is rarely diagnosed and has been reliably documented only in syphilitic meningitis.[3] Because syphilitic meningitis frequently causes papilledema or optic neuritis, the development of optic perineuritis seems to depend on the vagaries of the inflammatory process, and the diagnosis is provisional until normal cerebrospinal fluid pressure and functionally intact optic nerves are demonstrated.

Symptoms are few in optic perineuritis. Retro-orbital headaches caused by inflamed meninges at the base of the brain occur, but meningismus is usually absent. Because optic nerve dysfunction does not occur in this condition, visual symptoms are nonspecific; the visual sensations in our patient simulated those of papilledema caused by pseudotumor cerebri.[4] In these circumstances the clinical distinction between optic perineuritis and pseudotumor cerebri can be difficult, but we know that syphilitic meningitis occurs four times more frequently in men than in women,[5] whereas idiopathic pseudotumor cerebri is usually a disorder of obese women.[6]

The correct diagnosis of optic perineuritis is achieved by finding a reactive cerebrospinal fluid VDRL test; cerebrospinal fluid cell counts and protein values can be normal.[7] Furthermore, cerebrospinal fluid FTA-ABS tests are sensitive more than 99% of the time[8] and give false-positive results in fewer than 1% of normal subjects.[9]

Six to 9 million units of penicillin G is usually adequate treatment for symptomatic neurosyphilis,[10] but there are no established dosages for syphilitic optic perineuritis. Our patient improved with 9.6 million units of penicillin, and Lorentzen[10] reported successful results with 6 million units of procaine penicillin G for the treatment of syphilitic optic neuritis.

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MEETINGS, CONFERENCES, SYMPOSIA

EDITED BY THOMAS CHALKLEY, M.D.

INTERNATIONAL OPHTHALMIC MICROSURGICAL STUDY GROUP—EIGHTH BIENNIAL MEETING

The Eighth Biennial Meeting of the International Ophthalmic Microsurgery Study Group was held Sept. 29 to Oct. 3, 1980, at Cabo de Formentor, Majorca. The group consists of 40 members and the same number of invited guests. The 1980 meeting was devoted to the following subjects.

The first day covered cataract-extraction and glaucoma (S. McPherson, Durham, United States); cataract-extraction and corneal graft (E. Malbran, Buenos Aires, Argentina); iridopexy (J. Barraquer, Barcelona, Spain); radial keratomy for axial myopia (R. Troutman, New York, United States); surgical management of epithelial ingrowths and cysts (W. Stark, Baltimore, United States); vitrectomy instruments in anterior segment surgery (U. Demeler, Hamburg, Germany and A. Fink, New York, United States); hyaluronic acid in anterior segment surgery (R. Troutman, New York, United States).

During the second day vitreoretinal surgery was discussed: microsurgery of retinal detachments (M. Bonnet, Lyon, France and A. Urrets-Zavalia, Cordoba, Argentina); microsurgery in diabetic retinopathy (A. Muiños, Barcelona, Spain); techniques of vitreous surgery in retinal detachment (E. Malbran, Buenos Aires, Argentina); experimental chalcosis (H. Paulmann, Cologne, Germany); surgical management of persistent hyperplastic primary vitreous (W. Stark, Baltimore, United States); reattachment of the retina by vitreous replacement (R. Winter and J. Draeger, Bremen, Germany); preretinal membrane formation and macular pucker (Y. Fisher, New York, United States); extended use of vitrectomy instruments (A. Lim, Singapore).

In the afternoon the glaucomas were considered: microsurgery of Schlemm's canal (R. Sampaolesi, Buenos Aires, Argentina); surgical therapy of persisting hypotony (G. Naumann, Erlangen-Nurnberg, Germany); ciliary body reattachment in ocular hypotony (M. Shea, Toronto, Canada); optic disk in congenital glaucoma surgery (A. Dominguez, Madrid, Spain); surgery of congenital glaucoma (R. Witmer, Zurich, Switzerland); anesthesia for microsurgery (M. Roper-Hall, Birmingham, England, and W. Henschel, Bremen, Germany); effect of changes in P_{CO_2} and body positions on intraocular pressure (S. Kessing, Copenhagen, Denmark).

The third day was devoted to prevention and control of astigmatism: results of crescent resection on keratotorus (F. Barraquer, Bogota, Columbia); corneal relaxing incisions (R. Troutman, New York, United States); control of astigmatism in keratoconus (R. Witmer, Zurich, Switzerland); influence of nylon sutures on postoperative astigmatism (S. McPherson, Durham, United States).

The next subject was intraocular foreign bodies: methods of intraocular foreign body extraction (E. Malbran, Buenos Aires, Argentina); changes in management (M. Roper-Hall, Birmingham, England); clinical management of copper foreign bodies (H. Paulmann, K. Heimann, and H. Neubauer, Cologne, Germany); microsurgery of nonmagnetic foreign bodies in the vitreous chamber (A. Muiños, Barcelona, Spain). Short reports on new instruments and microscopes followed.

The fourth day was devoted to lens

implant surgery: physiological optics applied to pseudophakia (C. Huber, Zurich, Switzerland); prevention of endothelial damage (J. Draeger, Bremen, Germany); vitrectomy techniques for managing selected intraocular lens complications (W. Stark, Baltimore, United States); long-term results of intraocular lenses (K. Schott, Essen, Germany); comparative results with different ocular lenses (K. Schott, Essen, Germany); comparative results with different lenses (K.-W. Jaboci, Giessen, Germany).

The afternoon session covered microsurgery of the cornea: optical minikeratoplasty in bad prognostic situations (G. Naumann, Erlangen-Nurnberg, Germany); histocompatibility antigens and corneal transplantation (W. Stark, Baltimore, United States); the challenge of large penetrating keratoplasties (J. Barraquer, Barcelona, Spain); keratoplasty in babies with sclerocornea (E. Gregersen, Copenhagen, Denmark); refractive effects of disparate diameter graft recipient technique (R. Troutman, New York, United States); keratomileusis and keratophakia (J. Barraquer, Bogota, Columbia); and experimental data on keratophakia (F. Hoffmann, Berlin, Germany).

MICHAEL J. ROPER-HALL

THE OCULAR MICROBIOLOGY AND IMMUNOLOGY GROUP—1980 MEETING

The 1980 meeting of the Ocular Microbiology and Immunology Group was held in the Crystal Room of the Palmer House in Chicago Nov. 1, 1980, immediately before the meeting of the Academy.

The morning session was moderated by Starkey Davis and Fred M. Wilson, Jr.

The first paper, entitled "Comparative Evaluation of Gentamicin, Gentamicin and Betamethasone, and Placebo Ointment in the Treatment of Marginal Blepharitis," was presented by W. B. Jackson, T. Connally, and T. Easterbrook. The investigators treated 45 cases of marginal blepharitis with gentamicin, combined gentamicin and betamethasone, or a placebo three times daily for two weeks. Gentamicin alone and the gentamicin-corticosteroid preparation were effective in eradicating the bacteria in 14 days.

Since most of the organisms were *Staphylococcus epidermidis*, one member of the audience asked whether the presumed infectious origin of these cases had been definitely established, and whether the long-term results should not also have been considered.

A similar study was carried out by Peter Donshik and J. Schulman. They reported that the inflammatory signs in their 150 patients were significantly diminished by a corticosteroid. There were many variables in this study (for example, the eyelids were scrubbed), which precluded the drawing of any definite conclusions.

F. M. Polack, T. Demong, M. Burdette, and A. Hubbard studied the ocular bacterial flora at the time of penetrating keratoplasty. Surgical scrubs reduced the number of organisms present. It was mentioned that the presence of a pathogen is only one factor in determining whether a patient develops endophthalmitis.

T. O. Wood discussed corneal ulcers after corneal transplantation. All of his patients were receiving topical corticosteroids at the onset of their infectious corneal ulcerations, many of which were caused by streptococci. Dr. Wood's laboratory personnel reported that polymyxin was effective against gram-positive organisms and that streptococci were sensitive to gentamicin. Several people questioned both these statements and the accuracy of the laboratory report.

M. J. Tapert, D. B. Jones, N. M. Robinson, and R. J. Wallace presented a case of *Mycobacterium chelonei* keratitis. Amikacin, tobramycin, and sisomicin all seemed to be effective against this organism, but rifampin was not.

J. Baum, A. Jordan, and H. Fedukowicz presented a survey of *Moraxella* corneal ulcers in a derelict population. *Moraxella* was the most common cause of bacterial corneal ulcers in this population, and all of its subspecies were incriminated. Although the organisms were sensitive in vitro to all antibiotics, some cases did not respond clinically to some antibiotics. It was suggested that other organisms may be concomitantly culpable in this immunosuppressed population.

D. M. O'Day, J. R. Patrinely, and J. H. Elliott discussed the management of *Staphylococcus epidermidis* andophthalmitis. Noninvasive antibiotic therapy had an effect comparable to that of combined intravitreal antibiotics and vitrectomy. However, the problem remains, how do we know the causal organism is S. *epidermidis*? If we wait for culture results, it may be too late. So the dilemma persists.

J. H. Brinser, L. D. Perry, and H. Kolodner isolated and identified cell-wall-deficient and aberrant bacteria from ocular sources. They found a modified acridine orange stain effective in identifying them. The role of these bacteria is still to be defined and is an exciting subject for investigation.

K. F. Tabbara, S. Sakuragi, and G. R. O'Connor discussed the effect of minocycline on murine toxoplasmosis. This semisynthetic tetracycline was therapeutically effective in toxoplasmosis and seems to have a synergistic effect when used with sulfadiazine.

W. W. Culbertson, K. F. Tabbara, and G. R. O'Connor presented an experimental model of acute ocular toxoplasmosis in subhuman primates. The major drawbacks of the model are (1) the expense and difficulty of handling monkeys, and (2) certain nonclinical steps required in producing the model, for example, penetrating the eye with a needle.

M. A. Galin, A. W. Tuberville, and A. E. Maghraby discussed the sodium hydroxide sterilization of intraocular lenses. A 5% solution of sodium hydroxide sterilized the lenses in 24 hours and apparently did not hydrolyze the lens surface.

R. A. Eiferman and J. Schnyder discussed a moderately effective antifungal drug they obtained in China. Unfortunately, they do not know the drug's name nor its chemical structure.

R. E. Smith and Dr. Tse presented a case of lymphoid granulomatosis. The patient had early lung lesions, chills, fever, and weight loss. The ocular signs were palsy of the sixth, seventh, and eighth nerves and sheathing of the peripheral retinal vessels.

S. Stenson and A. Brookner reported a case of endogenous bilateral necrotizing scleritis caused by *Aspergillus oryzae*.

R. Abel and A. Tishman discussed a case of traumatic rupture of a corneal wound that occurred 2½ years after keratoplasty.

G. A. Stern, S. Zam, and V. J. Gutgesell presented a case of viral keratitis. The patient developed amoeboid subepithelial opacities eight days after the onset of a mild punctate keratitis. Herpes simplex virus type 1 was recovered in cultures. Serum adenoviral titers were not determined.

Ronald Smith and Jay Krachmer moderated the afternoon session.

M. V. Darnieder, H. E. Ullman, M. J. Beeson, J. L. Taylor, and W. J. O'Brien discussed the chemotherapy of keratitis induced by mutant and wild strains of herpes simplex virus type 1 in rabbits.

Acycloguanosine, vidarabine, and trifluorothymidine were effective against these strains and prevented stromal disease. Overall, trifluorothymidine seemed to be the most effective.

H. E. Kaufman, E. D. Varnell, and Y. M. Centifanto-Fitzgerald discussed various aspects of herpetic disease. They felt that the virulence of the herpes strain, not the response of the host, was largely responsible for the severity of the disease, and that the fortunate patient was the one who first contracted an avirulent strain. They confirmed the special efficacy of trifluorothymidine.

D. H. King and S. L. Hutcherson studied Viroptic and Stoxil solutions. A clinical trial in which many investigators participated was performed, and both drugs were found to be effective against epithelial herpetic disease (95% and 75% respectively). Only minor adverse effects were noted in some 9% to 11% of the patients.

K. R. Wilhelmus, B. R. Jones, and D. Coster stated that minimal debridement of epithelial herpetic keratitis in man, followed by 3% acyclovir ointment, resulted in rapid healing and minimal adverse effects. The results of this combination were superior to either treatment alone.

G. Smolin, M. Friedlaender, M. Okumoto, N. Stebbing, and R. Friedlaender treated rabbits with herpetic keratitis with 10^6 U/ml of natural or clonal human leukocyte interferon A, topically or systemically. Only the topical applications were effective, and both types of interferon were equally effective.

T. W. Sery, L. Foster, and A. W. Vogel sensitized corneas with bovine serum albumin (BSA). Eight weeks later, when the sensitized corneas were challenged with herpes simplex virus, they were resistant to it. Unsensitized control animals developed the usual herpetic infection. It was assumed that the initial sensitization resulted in a nonspecific immune awareness of the BSA-injected corneas.

P. A. Asbell and R. M. Franklin injected immune complexes or antigens into the cornea, and antibody into the conjunctiva, in attempts to develop a disciform corneal lesion. Limited success was attained. These investigators assumed that immune complexes could be responsible for the disciform lesions seen in stromal herpetic keratitis. A member of the audience suggested that antibody-dependent cellular cytotoxicity or cell-mediated immunity might play a role in disciform keratitis.

R. B. Guss and J. P. McCulley discussed the abnormal immune responses in Wiskott-Aldrich syndrome. The patients had depressed cellular immunities and abnormal antibody responses. In particular, their suppressor cells seemed to be defective. In the eye the abnormal findings were episcleritis, marginal keratitis, infectious blepharitis, and atopic dermatitis of the eyelids.

T. O. Bennett, R. Epstein, and J. Sugar discussed tissue typing in outbred canines undergoing keratoplasty. Matching by tissue typing resulted in longer survival of the grafts (75% clear) compared with an unmatched group (0% clear). Inbred animals, even when the recipient has a vascularized bed, have a high rate of success. In the ensuing discussion, the following unanswered question arose: Are 1-a antigens present on endothelial cells?

J. W. Chandler and T. E. Gillette used hyperbaric oxygen to pretreat corneal allografts in order to lengthen graft survival in mice. This procedure seemed to alter the 1-a antigen present in the cornea. A discussion of the exact location of these 1-a antigens followed. Additionally, the location of the Langerhans cells has yet to be clearly defined in the cornea.

M. B. Abelson and I. J. Udell exam-

ined the role of conjunctival eosinophils in allergic ocular disease. Eosinophils are numerous in vernal keratoconjunctivitis but may be absent in 85% of the scrapings from patients with acute allergic ocular disease. Histologic studies confirmed that it requires time for the eosinophils to reach the surface.

R. M. Lang and M. H. Friedlaender examined conjunctival scrapings from vernal keratoconjunctivitis and hay fever conjunctivitis with similar results.

M. R. Allansmith, K. J. Bloch, R. S. Baird, and K. Sinclair induced ocular anaphylaxis by the local conjunctival injection of antigen. The vascular permeability increased rapidly and lasted for an hour. Minor anaphylactic changes were also present in both the injected and contralateral eyes.

E. L. Stock, E. E. Schwartz, W. Watson, and D. M. Meisler developed a model of immediate hypersensitivity in the guinea pig by injecting it with ovalbumin. The clinical signs and local eosinophilia were typical of immediate hypersensitivity.

A. Sugar and R. F. Meyer noted giant papillae on the upper tarsal conjunctivas of 11 patients whose penetrating keratoplasties were performed with interrupted nylon sutures. Removal of the exposed sutures led to resolution of the papillae.

J. V. Greiner and M. R. Allansmith reported increased mucous secretion in patients with contact lenses who had associated giant papillary conjunctivitis. They thought the excessive mucus might be caused in part by mechanical trauma as well as by immune mechanisms.

The 1982 meeting will be held in San Francisco, California, on the Saturday before the International Congress opens.

GILBERT SMOLIN

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THE JOURNAL—1981

Readers who review THE JOURNAL back to front rather than front to back will be disconcerted to find that the summaries in this issue are no longer at the conclusion of the article, but rather at the beginning. This permits the orthodox front-to-back reader to tell at a glance whether he wishes to read the entire article or limit his attention to the summary. Additionally, it permits mechanical reproduction of a single page to provide the title, the summary, the name of the journal, the page number, and the year. This change is one of many initiated since my meeting with a youthful group of ophthalmologists (at least youthful in my eyes) in the JOURNAL offices last November during the meeting of the American Academy of Ophthalmology. Among their strong recommendations was publication of the summary before the body of the article. (I have perhaps a vain hope that this will result in the complete disappearance of those summaries in which the title is either repeated or paraphrased.)

The group suggested that Notes, Cases and Instruments include research notes of studies that did not justify an extended article. They believed too that the Correspondence section should be markedly strengthened. I have been trying to do this recently by sometimes suggesting that authors encapsulate material in a Letter to the Editor. The group also recommended strongly that the titles and authors of Letters to the Editor be printed on the title page so as not to be lost.

Opinions on the Abstracts section were mixed. I think this is to be expected because these leaders maintain a strong awareness of current developments in ophthalmology. They all approved publishing abstracts from nonophthalmic periodicals. They also recommended that the address of the senior author be given so one could write for reprints. We had done this previously and I discontinued it to save printing costs. We have decided to publish this information again because its usefulness should more than justify the costs.

Many recommended that the main editorial be related to an article in THE JOURNAL, thus putting the article in con-

text. We have done this on occasion, but some authors do not want other experts in their field reading their material before it is published. This procedure, though, seems to work well for "The New England Journal of Medicine" and "The British Journal of Ophthalmology."

There was concern regarding the large number of competent papers that are rejected. THE JOURNAL has the largest circulation of any journal in its field. Because of this, typescripts flood the office, since the authors know their views will get international attention. Even with the help of skilled referees, it is not possible to publish all of the meritorious material submitted. This is disheartening to authors and oftentimes in choosing between two equally meritorious papers, I select the one that requires the least editorial attention. This seems arbitrary and capricious, and frustrates some authors submitting to THE JOURNAL for the first time.

One participant asked that THE JOURNAL sell bound volumes so that readers could obtain their bound volumes at a minimum cost. Investigation indicated that the increased clerical and bookkeeping costs would make this unrewarding.

A variety of other topics were discussed, and readers will see gradual changes introduced in THE JOURNAL reflecting the recommendations that were made. I would be most interested in receiving suggestions from others who value THE JOURNAL.

FRANK W. NEWELL

OBITUARY

ARLINGTON COLTON KRAUSE 1896–1980

Arlington C. Krause was born in Chicago, July 27, 1896, and died in Knoxville, Tennessee, May 3, 1980. He earned his B.A. at Lawrence College (1918), and his M.A. (1921) and Ph.D. (1923) in chemistry at the University of Wisconsin. In 1928, he received an M.D. from Yale University. Because of his doctorates in chemistry and medicine, he was able to contribute to our knowledge of the chemistry of various portions of the eye. He was an assistant instructor at the University of Wisconsin from 1919 until 1923. After moving to Yale University, he was an instructor and a research assistant in surgery from 1925 until 1928. He then became an instructor at Johns Hopkins University in ophthalmology from 1929 until 1935. At the University of Chicago he began as an assistant professor in 1935 and became the Chairman of the Department of Ophthalmology before he left the university in 1956. He then went to the Veterans Administration Hospital in Memphis, Tennessee, as chief of the Department of Ophthalmology from 1956 until 1966. Dr. Krause wrote 95 articles, one monograph, and one book. His book, "The Chemistry of the Eye," was published by the Johns Hopkins Press in 1934. By 1945 he had written more than 50 articles on the chemistry of the eye. He then became interested in congenital malformations and wrote extensively on retrolental fibroplasia. He was involved in extensive research to find its cause.

In 1946, Dr. Krause wrote his classic paper on congenital encephalo-ophthalmic dysplasia, sometimes called "the Krause syndrome."

Dr. Krause was not a professor of mine, nor did we write a paper together. I did, however, spend a great deal of time with him in my early studies as a resident and regarded him as a mentor. Because of this, I dedicated my first book, "Ocular Differential Diagnosis," to him. Dr. Krause was able to look at a problem and to see each of its facets and ramifications. I don't know how many other ophthalmologists Dr. Krause influenced, but I can say that he had a profound influence on me and guided my efforts in ophthalmology.

It seems to me a man can be judged in two ways. One is by the influence he has on those around him; the other is by his published works. I think Dr. Krause met both criteria in a superb fashion.

F. HAMPTON ROY

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Hand-Held Fundus Cameras

Editor:

Hand-held fundus cameras, such as the Kowa RC-2, are extensively used by ophthalmologists but require very close approximation to the cornea if a reasonable field of view is to be achieved and corneal reflexes avoided. It does not seem to be widely known that these cameras (and presumably others) can be used successfully at distances of 50 cm or more by treating them as monocular indirect ophthalmoscopes and using a standard condensing lens. All the advantages of the indirect ophthalmoscope are then available to the observer—variable extent of field depending on lens used, easy visualization of the periphery, and variable magnification—and everything that is seen can be photographed immediately.

The internal illumination system provides adequate brightness for viewing but some trials may be necessary to determine the correct exposure for photography. Fast film should be used.

R. M. YOUNGSON, M.B.
London, England

Intraocular Lens Power Formulas

Editor:

The theoretical formulas used for intraocular lens power calculation confuse some ophthalmologists. The three most popular theoretical formulas are those of Binkhorst and Loones,[1] Colenbrander,[2] and Fyodorov, Galin, and Lonksz.[3] These three formulas are simply related and after some algebraic manipulation have the same form:

$$P = \frac{n}{A - D - g(t)} - \frac{n}{\frac{n}{K^1} - D - g(t)}$$

where P is the power of the intraocular lens, n is the refractive index of aqueous, A is the axial length of the eye, K is the cornea power in diopters, D is the cornea to intraocular lens distance, and g(t) is a function of intraocular lens thickness.

In the most general formula—that of Binkhorst and Loones—the intraocular lens is assumed to be thick with both anterior and posterior curvature. The function g(t) is complicated, but is a small number when evaluated. If one assumes that the posterior surface of the intraocular lens is flat, there is a considerable simplification in g(t). Because most intraocular lens are made this way, it is reasonable to make this assumption. When the thickness is chosen and appropriate indices of refraction are used for the aqueous and the lens materials, the formula can be further reduced to the structure given by Colenbrander. In this case g(t) becomes a constant (0.00005 m) reflecting a fixed thickness of the lens. A furthur simplification is obtained when the thickness is reduced to zero and a thin lens approximation is used. In this case the equation of Fyodorov, Galin, and Lonksz results from that of Colenbrander, when g(t) is equal to zero rather than 0.00005.

Using one of these formulas rather than another makes little practical difference. The equation of Fyodorov, Galin, and Lonksz and that of Colenbrander agree within 0.1 diopter for all but the most bizarre of corneal curvature and axial length combinations. Because the accuracy in fabrication of intraocular lenses is

considerably less than 0.1 diopter, it is not particularly important which of these formulas is used.

KARL J. FRITZ, M.D.
Chicago, Illinois

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Temporary Increase in Myopia After High-Altitude Climbing

Editor:

We examined a patient who experienced a temporary increase in his myopia in the absence of any other physical and ocular signs and symptoms after an expedition to the Himalayas.

The patient, a 51-year-old healthy businessman, a veteran mountain climber, and a physicist (optics) by training, noticed a sudden impairment of distant vision two days after returning to sea level after climbing in the Himalayas (up to 20,000 ft). His visual acuity had remained constant for the previous ten years; corrected visual acuity in both eyes had been 6/4.5 (20/15) and uncorrected visual acuity was R.E.: -2.25 -0.75×162 and L.E.: -0.75 -1.75×27 with a 1.5 bifocal addition for near. During the expedition he developed 2- to 3-mm moons across the entire width of his fingernails. The fingernails were clearly thinner in these areas and there were yellowish discolorations underneath. His toenails also showed similar thinned areas. The thinned areas gradually became normal.

When he returned to Canada (on the fourth day after he had first noticed that he could not see well with his spectacles), he underwent repeated ophthalmologic and medical examinations. His corrected visual acuity at that time was 6/9 (20/30) in both eyes and his uncorrected visual acuity was R.E.: -3.25 -0.75×165 and L.E.: -1.75 -1.75×35. He had no other ocular signs that could be attributed to the 1-diopter increase in myopia. Medical examinations showed no signs of diabetes or any other disease that could have been related to the visual problem. However, within seven weeks of his arrival in Canada, his corrected visual acuity returned to the pre-expedition level.

The patient described the sequence of events in a precise way, and there was no reason to doubt that what he said was genuine. He was certain that his vision for distance failed while he was in the Delhi airport on the day he left India for Canada, and that the impairment was sudden rather than gradual. The cause and mechanism involved in the sudden 1-diopter increase in myopia and its subsequent reversal remain a mystery. If we assume that the increase in myopia was related to high-altitude climbing, the time lag before the onset of the ocular symptom is puzzling. Although we cannot provide an answer, in view of the uniqueness of the case we suggest that mountain climbers, particularly individuals who are middle-aged or older, be closely monitored for similar problems.

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Burlington, Ontario, Canada
PRASANTA K. BASU, M.B.,
Toronto, Ontario, Canada

Retinal Pigment Epithelial Degeneration Associated With Leukocytic Arysulfatase A Deficiency—Correction

Editor:

In the article "Retinal pigment epithelial degeneration associated with leukocytic arylsulfatase A deficiency" by J. J. Weiter, M. Feingold, E. H. Kolodny, and S. S. Raghaven (Am. J. Ophthalmol. 90:768, 1980), the following correc-

tion should be made. On page 771, second paragraph, the fourth through sixth sentences should read as follows:

> They found increased lipofuscin in the retinal pigment epithelium as well as in the ganglion cells. Light microscopy demonstrated acid phosphatase in pigment epithelial cells as well as in ganglion cells. (Both lipofuscin[16] and acid phosphatase are normal constituents of the adult retinal pigment epithelium, but both of these entities were believed by the authors[14,15] to be increased.)

JOHN J. WEITER, M.D.
Boston, Massachusetts

Pharmacologic Localization in Horner's Syndrome

Editor:

In their article, "Evaluation of the causes and accuracy of pharmacologic localization in Horner's syndrome" (Am. J. Ophthalmol. 90:394, 1980), W. F. Maloney, B. R. Younge, and N. J. Moyer have made a worthwhile contribution to pupillary drug testing in Horner's syndrome by pointing out that when the affected pupil fails to dilate with hydroxyamphetamine, there may be something else wrong with the sympathetic innervation of the eye, in addition to a defect of the postganglionic neuron. I have been wrong if I have implied that, in such a case, the damage to the postganglionic neuron must be the only lesion in the sympathetic pathway; of course, a preganglionic lesion could also be present. This would certainly be the easiest way to explain most of the "false clinical localizations" made by the authors. Many of these patients had major trouble in the lower neck (tumor in Cases 2 to 6, surgery in Cases 7 to 9, and radiation in Cases 4 and 6). These troubles could easily have damaged the blood supply to the superior cervical ganglion, in addition (perhaps) to destroying the cervical sympathetic chain.

As pointed out by the authors, Sears, Kier, and Chavis[1] suggested in 1974 that the superior cervical ganglion might be vulnerable to spontaneous infarction. The blood supply to the ganglion apparently rises in the neck via the ascending cervical arteries. Injury to or surgery on the lower neck might thus simultaneously produce both preganglionic and a postganglionic Horner's syndrome. Sears, Kier, and Chavis believed that this might explain some "paradoxical" drug responses.

Weinstein, Zweifel, and Thompson[2] have recently shown that impaired dilation with hydroxyamphetamine often occurs in congenital Horner's syndrome when the distribution of the sweating loss suggests a preganglionic lesion; this is probably caused by transsynaptic dysgenesis of the postganglionic neuron after a preganglionic lesion. At least one of the 14 false localizations found by Maloney, Younge, and Moyer seems to fit into this category.

Maloney, Younge, and Moyer ask us to consider the possibility that the postganglionic lesion co-exists with, or is perhaps caused by, a covert preganglionic lesion whenever the pupil of a patient with Horner's syndrome fails to dilate with hydroxyamphetamine, This may happen, they say, in 16% of patients with Horner's syndrome. Come to think of it, one of my original six patients with surgical preganglionic lesions responded in just this way, and the pupil failed to dilate with hydroxyamphetamine.

H. STANLEY THOMPSON, M.D.
Iowa City, Iowa

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Reply

Editor:

We appreciate Dr. Thompson's remarks about false clinical localization in our series of patients with Horner's syndrome. We rewrote the first draft of the paper in an attempt to explain these paradoxical responses, and we agree with Dr. Thompson's theory of multiple neuromal involvement in at least several of these cases.

BRIAN R. YOUNGE, M.D.
Rochester, Minnesota

Photocoagulation in Toxoplasmic Retinochoroiditis

Editor:

I was greatly interested in the article, "Photocoagulation in active toxoplasmic retinochoroiditis" (Am. J. Ophthalmol. 89:858, 1980), by K. N. Ghartey and R. J. Brockhurst.

In September 1964, at the VII Pan American Congress of Ophthalmology in Montreal, I presented a report on xenon photocoagulation treatment of 25 cases of retinochoroiditis, performed either during an active primary attack or during an active recurrent episode. This report, along with a review of other studies, was published in Spain.[1] Spalter and associates[2] published a report the next year. My comments on their article appeared in 1966.[3]

At that time, photocoagulation was used to treat toxoplasmic retinochoroiditis in my country for the following reasons: (1) the high incidence of the disorder; (2) the social and economic factors that made it difficult for many people to obtain medical treatment; (3) the systemic complication and unproven efficacy, particularly in regard to cysts, of the many drugs used to treat the disease; (4) photocoagulation's ability to shorten the active phase of the attacks; (5) the possibility of avoiding satellite recurrences by destroying nearby cysts; and (6) the possibility of avoiding secondary complications (including glaucoma and cataract) produced by the inflammation.

During the 1960s, I studied 60 cases treated by this method. By the 1970s, however, I came to believe that active cases did not need photocoagulation for a number of reasons.

Active foci seem to heal spontaneously, although the amount of time required varies. Routine examinations disclose many instances of healed retinochoroiditis scars in patients unaware of ever having had the disease.

The disease can still recur, despite photocoagulation, near the treated area or in other parts of the retina, because all the cysts near the retinochoroiditis were not destroyed or because the cysts occurred far from the primary focus (D. Nicholson, personal communication, June 1980). For this reason, I have abandoned photocoagulation of inactive foci, something I originally recommended.

Two of the 60 cases became worse after photocoagulation, and the possibility of spreading the *Toxoplasma* organisms was raised.

In some cases, macular or extramacular tractional epiretinal membranes developed or worsened after photocoagulation. Vitreoretinal membrane contraction can lead to retinal holes and retinal detachment.

In eight cases, an iatrogenic hemorrhagic retinal vasculitis developed near the areas that underwent photocoagulation.[4]

At the present time, I rarely treat active retinochoroiditis with photocoagulation. I use this method no more than three or four times a year and only in cases where there is a danger of foveolar involvement or when the disease is extremely protracted.

Although xenon photocoagulation is still used, argon-laser photocoagulation and crycoagulation[5] have also been used in some cases.

I agree with the other findings of Drs. Ghartey and Brockhurst and congratulate them on their article.

ALVARO RODRIGUEZ, M.D.
Bogata, Colombia

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Reply

Editor:

First, I must state that I was unaware of Dr. Rodriguez's presentation at the Pan American Congress of Ophthalmology in 1964 regarding photocoagulation in toxoplasmic retinitis. Second, I am well aware of Dr. Spalter's work concerning prophylactic photocoagulation, a technique that proved to be ineffective in my hands. Third, I agree with Dr. Rodriguez that photocoagulation in active toxoplasmic retinitis is indicated only in protracted cases, refractory to conventional forms of therapy, and wish to congratulate him on his work.

ROBERT J. BROCKHURST, M.D.
Boston, Massachusetts

Senile Lens and Senile Macular Changes

Editor:

In their article, "Senile lens and senile macular changes in a population-based sample" (Am. J. Ophthalmol. 90:86, 1980), R. D. Sperduto and D. Seigel failed to find an association between senile lens changes and senile macular degeneration. When two diseases are so prevalent (with senile lens changes found in 42% to 91% of the sample and senile macular degeneration in 35% to 50%) but show no association, the presumption that senility causes both must be doubted. It is likely that macular degeneration is a disease of senility, whereas lenticular changes may be the result of prolonged exposure to harmful environmental factors. Environmental factors have been shown to play a significant role in cataract.[1,2] This hypothesis suggests that further investigation into the identity of environmental risk factors is needed if we are to prevent these lenticular changes.

S. P. DHIR, M.D.
Chanigarh, India

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2. Fuchs, A.: Geographic distribution of senile cataract. Am. J. Ophthalmol. 49:1039, 1960.

Reply

Editor:

Both senile macular and senile lens changes are increasingly prevalent at older ages. Analysis of our data indicated that they occurred independently, which implied separate causative mechanisms to us. Our data did not permit us to distinguish between environmental factors and those that are part of biological aging. Finally, the relationships between the various subgroups which make up these two broad groups of "senile" changes remain largely unexplored.

ROBERT D. SPERDUTO, M.D.
DANIEL SEIGEL, Sc.D.
Bethesda, Maryland

BOOK REVIEWS

Cataract Surgery and its Complications, 3rd. ed. By Norman S. Jaffe. St. Louis, C. V. Mosby Co., 1981. Hardcover,

611 pages, preface, index, 515 black and white figures. $69.50

The appearance of its third edition confirms this book's position as one of the classics in ophthalmology. Those who don't own the previous editions will certainly want to own this one. Those who own the second edition may wonder whether they need to buy the third. I think they do, but let me review the differences between them.

The cover has color illustrations and, therefore, is printed on heavy paper. In the two copies that I have seen, the corners have already worn through. Personally, I much prefer a cloth binding. The book otherwise represents the usual high quality of book-making expected from Mosby. The type is clear and easy to read, and the inserts that appeared in smaller type in the second edition have now been printed in full-size type.

The number of illustrations has been doubled: 1,283 in this new edition! The number of pages has been increased 20%. A small amount of this is the result of better spacing of material on the pages, but most is caused by the addition of new material. There is an entirely new chapter on keratophakia and keratomilousis by Jose Barraquer. As far as I know, this is the only detailed presentation of this material in English.

As far as I can tell, one half of the chapters have only minor changes; three are unchanged. This is disappointing, at least superficially, because I expect this treasured volume to be up to date on everything. This means that many of the references have not been updated since 1975 (reflecting the usual two-year time lag before the publication of the 1977 edition).

There has been some shift to an extracapsular approach, but the emphasis in this book remains on the scientific aspects of cataract surgery and the management of complications rather than on surgical techniques. Discussions on the use of vitrectomy instruments have been added where they are appropriate to the management of complications in cataract surgery, but the chapter on subluxation of the lens continues to discuss the Calhoun pin without comment. I think most people have abandoned this technique, and are now removing dislocated lenses with a coaxially illuminated microscope, with or without the help of a vitreous surgeon.

The section on intraocular lenses has been updated and includes a superb discussion. There is an excellent expansion of the chapter on cystoid macular edema, with references through 1979. The chapter on endophthalmitis and the section on the diagnosis of postoperative endophthalmitis have been expanded. I notice that Dr. Jaffe now prefers using prophylactic preoperative antibiotics. The discussion on antibiotics in these two sections is excellent. It seems strange that Table 21-5 is left over from 1964. Certainly much newer material has been presented but the rest of the chapter is now up-to-date except that, both in this chapter and in the introductory chapters, Dr. Jaffe fails to mention modern draping techniques. The drapes are wrapped around the eyelid margins, draping the eyelashes out of the field and eliminating all worries about mechanical and bacterial interference by the eyelashes during surgery. The operating room floor should certainly not be swept between cases; in my own operating room I even forbid mopping between cases, for the reasons that Dr. Jaffe enumerates.

The faults are minor; the work superb. Certainly this new edition belongs on the bookshelf of every ophthalmologist performing cataract surgery, or caring for postoperative cataract patients.

ROBERT C. DREWS

Nutritional Ophthalmology. By Donald S. McLaren. New York, Academic Press, Inc., 1980. Hardcover, 438 pages,

index, 132 black and white figures. $74.50

"Nutritional Ophthalmology" is a thorough, updated revision of Dr. McLaren's 1963 classic, "Malnutrition and the Eye."

One third of the book is devoted to vitamin A deficiency and xerophthalmia, reflecting the seriousness of the problem and Dr. McLaren's long-standing interest in it. The biochemical, epidemiologic, and clinical facets of the problem are explored in detail.

The section that follows deals with the B vitamins, and the difficulty in attributing significant ocular pathology to any one of them. This is particularly evident in the discussion of "nutritional amblyopia," contrasting sharply with our understanding of Wernicke's disease.

The last portion deals mainly with ocular manifestations of inborn errors of metabolism. Although not traditionally "malnutritional" in nature, their inclusion is justified on the basis that some respond to dietary manipulation.

The author continues his practice of clearly distinguishing animal from human data, and the two are better integrated than in the earlier version.

Dr. McLaren has succeeded, once again, in producing an admirable review of the relationship between nutritional status and the eye. The subject is pertinent to developing countries, where malnutrition remains endemic, and our own, where chronic wasting diseases, new surgical procedures, reducing regimens, and fad diets all increase the likelihood that the average ophthalmologist will encounter the clinical entities described in this volume.

ALFRED SOMMER

Vitreous Surgery. By Ronald G. Michels. St. Louis, C. V. Mosby Co., 1981. Hardcover, 462 pages, preface, index, 436 black and white figures. $65

This comprehensive book on vitreous surgery covers not only surgical techniques but also the embryology, anatomy, and biochemistry of the vitreous and pathophysiology related to vitreoretinal disease. A description of the development of vitreous surgery and the various instruments used is of historical interest.

This work is well organized, and covers the preoperative evaluation of patients, surgical objectives and techniques, indications and results, postoperative management, and complications. The presentation gives a perspective on current problems, anticipated future refinements, and unsolved or controversial issues. For a book of this kind, illustrations are of paramount importance and this volume fulfills this need splendidly. Each chapter is extensively and broadly referenced not only with respect to clinical subjects, but also in regard to histopathology and basic science.

Throughout the book, the author provides the views and techniques of others as well as his personal preferences. The summaries and conclusion present a digest of the current state of the art. This book is a thorough, up-to-date evaluation of vitreous surgery. No rigid or standardized type of operation or technique is suitable for every case. Careful study of this book and assimilation of its principles and concepts will permit the surgeon to choose among many techniques when dealing with individual cases and problems encountered.

This book should be read by all residents in ophthalmology and all practitioners interested in this aspect of ophthalmologic practice.

PETER H. MORSE

Hypnosis in Ophthalmology. By George E. Morgan. Birmingham, Alabama, Aesculapius Publishing Co., 1980. Hardcover, 106 pages, index. $25

Dr. Morgan has written an interesting volume on hypnosis as it applies to the practice of ophthalmology. The book is simply written in an informal style but communicates the author's ideas lucidly. No attempt is made to present the subject in depth. The focus is a pragmatic approach aimed at providing a valuable, ancillary tool in the day-to-day endeavor of caring for patients with eye disease.

The information is useful and I plan to incorporate some of these principles in my own practice. Particularly appropriate is the author's message that we should all be physicians and not just ophthalmologists—a principle all too often neglected in the current practice of ophthalmology and in the current teaching of ophthalmic residents.

RICHARD S. RUIZ

Clínica Oftalmológica. By Adalmir Morterá Dantas. Rio de Janeiro, Editora Guanabara Koogan S.A., 1980. Hardcover, 987 pages, index, 1036 figures, many in color.

This is a basic text for medical students, beginning ophthalmology residents, and physicians in other specialties who have Portuguese as their primary language. The book is divided into four sections: (1) Basic Embryology, Anatomy, and Physiology; (2) Ocular Pathology; (3) Systemic Diseases; and (4) Ancillary Diagnostic Studies. Professor Dantas has provided the intended audience with a detailed, profusely illustrated, well-referenced introduction to medical ophthalmology. Additionally, this book is an excellent source of up-to-date ophthalmic terminology for interpreters and translators.

DON H. NICHOLSON

Goodman and Gilman's The Pharmacological Basis of Therapeutics, 6th ed. Edited by Alfred Goodman Gilman, Louis S. Goodman, and Alfred Gilman. New York, Macmillan Publishing Co., 1980. Hardcover, 1,843 pages, index. $45

The first edition of this classic textbook was published nearly 40 years ago. That it is in its sixth edition is a tribute to the quality of its editors and contributors. An additional beauty of this book is that it is continually changing, not only by updating chapters but also by adding new subjects. In this edition, for example, discussions of the principles of therapy, pharmacokinetics, drug interactions, neurohumeral transmission in the central nervous system, neural transmission in the autonomic nervous system, and toxicology have been added. Pharmacology is such a rapidly expanding field, with our knowledge of the mechanisms of action and the emphasis on risk/benefit ratios increasing, that a textbook such as this is a must for anyone interested in pharmacology. Still, expecting every ophthalmologist to purchase this book may not be realistic, but it is essential to have easy access to a copy. The book is basically written from the generalist's viewpoint and contains surprisingly little ocular pharmacology. Even so, for a volume of this size, the editors have done a superb job of making it a practical book and the classic text on pharmacology.

F. T. FRAUNFELDER

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Albrecht von Graefes Archiv für Klinische und Experimentelle Ophthalmologie

EXPERIMENTAL CRYOSURGICAL LESION OF THE OPTIC NERVE. Loewenstein, J. I., and Lessell S. (Depts. Ophthalmol., Neurol. and Anat., Boston Univ., 80 E. Concord St., Boston, MA 02118). Albrecht von Graefes Arch. Klin. Ophthalmol. 215:97, 1980.

The authors were stimulated to perform this study by a case where cryopexy at the posterior pole was followed by blindness. In the present study the right optic nerves of three cynomolgus monkeys were frozen under direct visualization after a modified Krönlein operation. Left optic nerves were subjected to an identical procedure without freezing. Postoperatively, visual-evoked responses could not be obtained by stimulating right eyes, while electroretinograms from both eyes and visual-evoked responses obtained by stimulating left eyes were normal. Extensive lesions found in the right optic nerves of all animals were histologically similar to those obtained by freezing in the brain. (5 figures, 7 references)—David Shoch

IMPORTANCE OF ORIENTATION ON VISUAL AND NEURONAL RESOLUTION. (German) Harms, G., and Rassow, B. (Abteilung für Medizinische Optik der Universitäts-Augenklinik, Martinistr. 52, D-2000 Hamburg 20, Budesrepublik Deutschland). Albrecht von Graefes Arch. Klin. Ophthalmol. 215:123, 1980.

Spatial resolution of gratings of different orientation has been evaluated in 39 patients, by means of projected slides and by laser interference fringes. Both methods reveal a significantly higher resolution (about 12%) for vertical or horizontal bars compared to oblique ones. The similarity of findings for both methods is indicative of the neuronal origin of this orientation-dependent effect and exclude an optical origin. (4 figures, 11 references)—Authors' abstract

American Journal of Clinical Pathology

ORBITAL AND ADNEXAL LYMPHOMAS. Astarita, R. W., Minckler, D., Taylor, C. R., Levine, A., and Lukes, R. J. (Depts. Pathol., and Med., Univ. Southern California, Los Angeles, CA). Am. J. Clin. Pathol. 73:615, 1980.

The application of immunologic, cytochemical, and electron-microscopic technics to the study of lymphoid lesions of the orbit and adnexa was found to enhance the accuracy of diagnoses of malignant lymphoma in five cases. The immunologic characterization of these lymphoproliferative disorders is emphasized as an especially useful diagnostic parameter. The occurrence of false-negative diagnoses of pseudolymphoma of the orbital region may be partially explained by a failure to recognize the relatively frequent plasmacytoid lymphocytic lymphoma, a distinctive entity that may mimic a reactive inflammatory process. All of the cases of malignant lymphoma reported represented stage I or stage II disease, suggesting that primary lymphoma of the orbital region may be a disease with a favorable prognosis. (6 figures, 4 tables, 31 references)—Authors' abstract

Archives of Ophthalmology

RACQUETBALL AS AN OCULAR HAZARD. Doxanas, M. T., and Soderstrom, C. (Dept. Ophthalmol., Greater Baltimore Med. Ctr., 6701 N. Charles St., Baltimore, Md 21204). Arch. Ophthalmol. 98:1965, 1980.

Thirty-seven cases of ocular injury incurred while persons were playing racquetball were found in reviewing records of 1,071 emergency room patients in a three-month period. These can be divided into ball-induced and racquet-induced injuries. Fifteen ball-induced injuries occurred, ranging from a corneal abrasion to vitreous hemorrhage, in a patient with sickle cell retinopathy. Twenty-two cases of racquet-induced brow lacerations that occurred when the player struck his (or her) own forehead with the racquet were found. During the same interval, five tennis-related ocular injuries were observed. Ocular safety devices are strongly recommended to help prevent racquetball injuries. (6 references)—Authors' abstract

THE LONG-TERM VISUAL EFFECTS OF SHORT-TERM BINOCULAR OCCLUSION OF AT-RISK NEONATES. Hoyt, C. S. (Dept. Ophthalmol., Room 751-A, Univ. California Med. Ctr., San Francisco, CA 94143). Arch. Ophthalmol. 98:1967, 1980.

A retrospective study of 50 five-year-old children whose eyes were patched bilaterally to treat neonatal jaundice was compared with a study of a similar group of 50 five-year-old children who were treated in the intensive care nursery but whose eyes were not patched. No difference in the incidence of strabismus or loss of stereoacuity was established in these two groups. Despite the experimental evidence documenting changes in the visual cortex and interocular alignment in animals binocularly deprived of visual stimulation near birth, the clinical practice of binocularly patching the eyes of neonates with jaundice does not seem to increase the incidence of subsequent strabismus or loss of stereoacuity. (3 tables, 20 references)—Author's abstract

LOXOSCELISM OF THE EYELIDS. Edwards, J. J., Anderson, R. L., and Wood, J. R. (Dept. Ophthalmol., Univ. Iowa Hosp. and Clinics, Iowa City, IA 52242). Arch. Ophthalmol. 98:1997, 1980.

Loxoscelism is a reaction to the bite of spiders of the genus *Loxosceles*. Several species have been found in the United States; the most commonly encountered is *L. reclusus*, the small brown recluse spider. Two types of reactions occur from the bite. In the localized type, necrotic loxoscelism, a cutaneous lesion with extensive gangrene develops. About 25% of patients have systemic manifestations of viscerocutaneous loxoscelism. There is the same local reaction, but, in addition, fever, chills, vomiting, joint pain, and hematologic abnormalities occur. Hemoglobinemia and hemoglobinuria suggest severe involvement. Deaths are believed to be caused by massive intravascular hemolysis. The authors describe a 61-year-old man who was bitten on the periorbital region; viscerocutaneous loxoscelism with gangrenous involvement of the eyelids developed. Severe laryngeal edema from regional, massive swelling of his neck was life-threatening. (10 figures, 16 references)— Authors' abstract

AGING OF THE OPTIC NERVE. Dolman, C. L., McCormick, A. Q., and Drance, S. M. (Pathol. Lab., Vancouver Gen. Hosp., 855 W. 12th Ave., Vancouver, British Columbia, Canada V5Z 1M9). Arch. Ophthalmol. 98:2053, 1980.

Histologic studies were carried out on 300 optic nerves covering ages from birth to 96 years. The optic nerve is small and nearly unmyelinated at birth. It rapidly grows and becomes medullated. With advancing years, the leptomeninges and fibrous septa become broader and occupy an increasingly larger proportion of the cross-sectional area of the nerve. The axons progressively diminish, and this probably reflects primarily a loss of ganglion cells and would contribute considerably to reduced visual acuity in the older population. Other age-associated degenerative changes are corpora amylacea and lipofuscin in astrocytic cytoplasm. In the older population, scars, swollen axons, and Schnabel's cavernous degeneration become common, indicating a high incidence of vascular impairment. (10 figures, 2 tables, 25 references)—Authors' abstract

Berner Beiträge zur Geschichte der Medizin und der Naturwissenschaften

THE IRIS IN EIGHTEENTH-CENTURY PHYSIOLOGY. Mazzolini, R. G. (Hans Huber Publishers, Bern, Switzerland). Berner

Beiträge zur Geschichte der Medizin und der Naturwissenschaften 9, 1980.

Twentieth century ophthalmologists take for granted the fact that the iris has two muscles and that these control the size of the pupil. It may come as a surprise to find that this fact was not accepted at all in the 18th century and the most ingenious kinds of explanations were given to avoid the recognition that there were muscles present. This may in part have come about from the statement of von Haller that "all muscles are irritable . . . the iris . . . has no irritability." On this basis, the muscular origin of the pupil reflex was ignored. Only later was it agreed that muscles must be involved and this was purely on the basis of a syllogism such as the following: (1) all structures that contract contain muscles; (2) the iris contracts; (3) therefore the iris contains muscles. Students of the history of ophthalmology will find this monograph interesting. (37 figures, 4 tables, 485 references)—David Shoch

Brain

THE NEUROLOGY OF VISUAL ACUITY. Frisén, L. (Dept. Ophthalmol., Univ. Göteborg, Sahlgren's Hosp., S-413 45 Göteborg, Sweden). Brain 103:639, 1980.

A series of patients with well defined lesions of various parts of the visual pathways was studied in an attempt to illuminate the neuropathophysiology of visual acuity. Acuity was found to remain normal in all cases with unilateral retrochiasmal lesions, including those of the optic tract. Bilateral retrochiasmal lesions involving the foveal nerve fibers on both sides impaired acuity to the same degree in both eyes. Lateral chiasmal lesions regularly produced impaired acuity in the ipsilateral eye. Midchiasmal lesions commonly led to an impairment of visual acuity in both eyes, usually asymmetrically, and roughly proportionate to the severity of the visual field defect. Compression optic neuropathy was found to reduce acuity in rough proportion to the severity of compression. It was concluded that acuity remains normal as long as either the crossing or the non-crossing neural outflow from the retinal fovea remains intact: acuity fails only when both sets of nerve fibers are compromised. A properly executed acuity test seems to be a powerful tool for detecting such conditions. The lower limit of normal acuity should never be set below 1.0 or 20/20: even this level is clearly subnormal in many subjects. (12 figures, 1 table, 120 references)—Author's abstract

British Journal of Ophthalmology

OCULAR RETICULUM CELL SARCOMA. Simon, J. W., and Friedman, A. H. (Dept. Ophthalmol., Mount Sinai Med. Ctr., 1 Gustave L. Levy Pl., New York, NY 10029). Br. J. Ophthalmol. 64:793, 1980.

Reticulum cell sarcoma has been reclassified and is now known as histiocytic lymphoma. It is a malignancy composed of large cells resembling histiocytes. The disease presents a recognizable clinical pattern and the authors report three cases, all patients in the sixth and seventh decades of life. Usually the patients report a gradual visual loss and examination discloses uveitis with a great deal of vitreous debris. Medical therapy is ineffective. Reticulum cell sarcoma in the central nervous system or other systems may precede or accompany the ophthalmic presentation. If the disease is suspected the appropriate diagnostic modality is vitreous aspiration. (8 figures, 17 references)—David Shoch

Investigative Ophthalmology and Visual Science

VISUAL ACUITY SCREENING OF PRETERM INFANTS. Dobson, V., Mayer, D. L., and Lee, C. P. (Dept. Psychol., NI-25, Univ. Washington, Seattle, WA 98195). Invest. Ophthalmol. Vis. Sci. 19:1498, 1980.

Visual acuity was screened in 36 healthy infants born four or more weeks prior to term. Preterm infants tested at eight and 12 weeks of postnatal age showed significantly poorer performances than those shown by eight- and 12-week-old full-term infants. However, no differences in performance were found when

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the scores of preterm infants tested at four, eight, and 12 weeks of postterm age (i.e., four, eight, and 12 weeks from due date) were compared with scores of four-, eight-, and 12-week-old full-term infants. The results suggest that visual acuity is more closely correlated with age from conception than with age from birth and that visual acuity screening in preterm infants should be carried out with acuity gratings appropriate for the infant's postterm age rather than with acuity gratings appropriate for the infant's postnatal age. (3 figures, 1 table, 19 references)—Authors' abstract

THE EFFICACY OF ASCORBATE TREATMENT AFTER SEVERE EXPERIMENTAL ALKALI BURNS DEPENDS UPON THE ROUTE OF ADMINISTRATION. Pfister, R. R., Paterson, C. A., Spiers, J. W., and Hayes, S. A. (1720 8th Ave., South, Birmingham, AL 35233). Invest. Ophthalmol. Vis. Sci. 19:1526, 1980.

Alkali burns were produced in a series of rabbit eyes with a 1N sodium hydroxide solution applied for 35 seconds over a 12-mm area. In one experiment the treated group received daily subcutaneous injections of ascorbic acid while the control group received no treatment. At the end of 30 days there was no significant difference in the number of eyes that had perforated. In a second experiment the burned rabbits received 10% ascorbic acid topically while the control eyes were given the vehicle only. In this group 80% of the control eyes ulcerated or perforated while only 30% of the ascorbate treated group showed ulcerations or perforations. The difference was probably caused by the level of ascorbate achieved in the aqueous humor (2 tables, 6 references)—David Shoch

UTILITY OF THE ARDEN GRATING TEST IN GLAUCOMA SCREENING: HIGH FALSE-POSITIVE RATE IN NORMALS OVER 50 YEARS OF AGE. Sokol, S., Domar, A., and Moskowitz, A. (Dept. Ophthalmol., Tufts-New England Med. Ctr., 171 Harrison Ave., Boston, MA 02111). Invest. Ophthalmol. Vis. Sci. 19:1529, 1980.

The Arden grating test was administered to 64 subjects between six and 82 years of age without ocular pathology, to 20 glaucoma patients, and to 21 ocular hypertensives. The results show an age effect, with significantly higher scores (lower sensitivity) in normal subjects over 50 years of age. In addition, there was no significant difference in performance between age-matched normals, glaucoma patients, and ocular hypertensives on the Arden gratings. The results show a high percentage of false-positives in older normal subjects. The Arden gratings should be used cautiously when one is testing patients over 50 years of age. (1 figure, 2 tables, 7 references)—Authors' abstract

Lancet

ERYTHROCYTE GLYCERALDEHYDE-REDUCTASE LEVELS IN DIABETICS WITH RETINOPATHY AND CATARACT. Crabbe, M. J. C., Halder, A. B., Peckar, C. O., and Cheng, H. (Univ. Oxford, Nuffield Lab. Ophthalmol., Walton St., Oxford, OX2 6AW England). Lancet 2:1268, 1980.

The activity levels of glyceraldehyde-reductase and aldose-reductase-like enzyme present in erythrocytes were determined in 104 diabetics. Some of these patients had retinopathy, some had cataracts, some had neither, and some had both. In general, patients with both retinopathy and cataracts had significantly higher enzyme activity than normal controls and this also held true for diabetics with only retinopathy or only cataracts. If neither cataracts nor retinopathy were present, then there was no significant difference in the glyceraldehyde-reductase levels. Juvenile diabetics had a significantly higher enzyme level than adult-onset diabetics. The increase in the levels of erythrocyte glyceraldehyde-reductase was caused by increased amounts of active enzymes rather than to elaboration of new kinetic pathways. (2 figures, 40 references)—David Shoch

Ophthalmic Surgery

REACTIONS TO AN ANTERIOR CHAMBER LENS—TWO YEARS AFTER IMPLANTATION. Wolter, J. R., Croasdale, R. E., Bahn, C. F. (Depts. Ophthalmol. and Pathol., Univ. Michigan Med. Ctr.,

Ann Arbor, MI 48109). Ophthalmic Surg. 11:794, 1980.

For two and a half years this 99-year-old man had done well with his anterior chamber lens of the Choyce type. He donated the eye for pathological evaluation after his death. The eye exhibited some superior corneal edema, distortion of the lower iris with formation of pockets to hold the inferior feet of the implant, some iris atrophy, slight iritis, minimal bleeding, slight neovascularization, and vacuolic degeneration of the pigment epithelium. The observation of a callus-like formation of connective tissue on all those parts of the iris surface that have had contact with the lens implant is the most important finding. This indicates that the iris can not only adjust to the foreign substance of the implant, but it can also give it firm and lasting support. (11 figures, 17 references)—Authors' abstract

Ophthalmology

EYELID RETRACTION TREATED BY LEVATOR MARGINAL MYOTOMY. Grove, A. S. (Massachusetts Eye and Ear Infirm., 243 Charles St., Boston, MA 02114). Ophthalmol. 87:1013, 1980.

Upper eyelid retraction in patients with ophthalmic Graves' disease may be due to: (1) overaction of Müller's muscle; (2) overaction of the levator muscle; (3) contraction of the levator; and (4) subcutaneous adhesions anterior to the levator. A new operation is described to lengthen the levator by performing partial width (marginal) myotomies. If present, subcutaneous adhesions can also be divided during this operation. Tissue removed from contracted levators at the time of marginal myotomy showed muscle damage with atrophy. There was no histologic evidence of acute inflammatory change in either the levators or Müller's muscle. (14 figures, 23 references)—Author's abstract

BOTULINUM TOXIN INJECTION INTO EXTRAOCULAR MUSCLES AS AN ALTERNATIVE TO STRABISMUS SURGERY. Scott, A. B. (Smith-Kettlewell Inst. Vis. Sci., San Francisco, CA 94115). Ophthalmol. 87:1044, 1980.

Sixty-seven injections of botulinum A toxin were given to patients for correction of strabismus. No systemic complications of any kind have occurred. The maximum time of paralysis occurs four to five days following the injection, and then gradually diminishes, depending on the dose. The maximum correction of strabismus has been 40 prism diopters. The maximum follow-up following injection is six months. Injection of botulinum A toxin into extraocular muscle to weaken the muscle appears to be a practical adjunct or alternative to surgical correction. (5 figures, 2 tables, 3 references)—Author's abstract

LASER SHRINKAGE OF THE CILIARY PROCESSES: A TREATMENT FOR MALIGNANT (CILIARY BLOCK) GLAUCOMA. Herschler, J. (Div. Ophthalmol., Univ. Arizona Health Sci. Ctr., Tucson, AZ 85724). Ophthalmol. 87:1155, 1980.

Based on the observation of the unusual relationship between the ciliary processes and lens equator in patients with "malignant" glaucoma, a hypothetical cause for the shallowing of the anterior chamber has evolved. Using the concept of a relative block of forward aqueous flow at the lens equator as the inciting element in malignant glaucoma, the clinical response to treatment can be explained. In five of six cases where maximal medical therapy failed to relieve the malignant process, laser shrinkage of several ciliary processes was successful in reversing the posterior secretion of aqueous and restoring the anterior chamber. (1 table, 7 references)—Author's abstract

C-REACTIVE PROTEIN IN GIANT CELL (CRANIAL, TEMPORAL) ARTERITIS. Eshaghian, J., and Goeken, J. A. (Medical Bldg., 6333 Wilshire Blvd., #510, Los Angeles, CA 90048). Ophthalmol. 87:1160, 1980.

C-reactive protein is a constituent of the serum originally found in patients with acute pneumococcal pneumonia. This protein forms a precipitin reaction with the capsular mucopolysaccharide of pneumococci. In this study, the C-reactive protein was increased in ten of 11 patients with biopsy-proven temporal arteritis but was positive only in 14 of 32 patients with negative temporal artery biopsies. Clinical correlation showed that C-reactive protein was more sensitive than the erythrocyte sedimentation rate in following the adequacy of corticosteroid dosages in temporal arteritis, probably because many more noninflamma-

tory factors influence erythrocyte sedimentation than influence the level of C-reactive protein. (2 tables, 44 references)—David Shoch

Survey of Ophthalmology

RETINAL VASCULAR ACCIDENTS IN GLAUCOMA AND OCULAR HYPERTENSION. Luntz, M. H., Schenker, H. I. (Dept. Ophthalmol., Beth Israel Med. Ctr., 10 Nathan D. Perlman Place, New York, NY 10003). Surv. Ophthalmol. 25:163, 1980.

The association of primary open-angle glaucoma with vein occlusions appears to be genuine. Primary open-angle glaucoma occurs in anywhere from 6% to 60% of patients with retinal vein occlusion, depending on the series one studies. The association is not as clear with branch vein occlusion. Conversely, central vein occlusions occur in 3.5% to 5% of patients with primary-angle glaucoma and in about 3% of patients with ocular hypertension. Since this association is documented, it is recommended that patients over 65 years old who have intraocular pressures greater than 25 mm Hg have this pressure lowered even in the absence of true glaucoma. (1 table, 40 references)—David Shoch

Correction

Glen Gole, M.B., is the author of the letter to the editor, "Retrolental fibroplasia and the retinal vasculature" (Am. J. Ophthalmol. 90:435, 1980).

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

XX COLOMBIAN CONGRESS OF OPHTHALMOLOGY

The XX Colombian Congress of Ophthalmology will be held Aug. 5–9, 1981, in Bogotá, Colombia. For further information, write Sociedad Colombiana de Oftalmologia, Carrera 7a, No. 66-10, Apto. 203, Apartado Aéreo 51632, Bogotá, D. E. Colombia.

RESEARCH TO PREVENT BLINDNESS: NEW SCHOLARS AWARD

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UNIVERSITY OF OKLAHOMA OPHTHALMOLOGY ALUMNI: FIFTH ANNUAL MEETING

The Fifth Annual Meeting of the University of Oklahoma Ophthalmology alumni will be held April 4, 1981. Registration fee is $20, which includes luncheon. For further information, write Wayne F. March, M.D., Dean A. McGee Eye Institute, 608 Stanton L. Young Blvd., Oklahoma City, OK 73104.

UNIVERSITY OF CALIFORNIA POSTGRADUATE COURSE: CLINICAL MANAGEMENT OF INFECTIOUS DISEASES OF THE EYE: NEW DEVELOPMENTS

The University of California will hold a postgraduate course, "Clinical Management of Infectious Diseases of the Eye: Newer Developments," June 11 and 12, 1981. For further information, write Extended Programs in Medical Education, Room 569-U, University of California, San Francisco, CA 94143.

AMERICAN SOCIETY OF OPHTHALMIC PLASTIC AND RECONSTRUCTIVE SURGERY: 1981 OFFICERS

At the 11th Annual Scientific Symposium of the American Society of Ophthalmic Plastic and Reconstructive Surgery in Chicago, Nov. 7 and 8, 1980, the following new officers were elected: John L. Wobig, chairman of the advisory board; Allen M. Putterman, president; A. Jan Berlin, president elect; Fay E. Millett, vice president; Richard P. Carroll, executive secretary; Joseph Flanagan, secretary; Eugene O. Wiggs, treasurer; and Clinton D. McCord, program chairman.

SOUTH DAKOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: OFFICERS FOR 1980–1981

The South Dakota Academy of Ophthalmology and Otolaryngology officers for 1980–1981 are as follows: Michael W. Pekas, president; and Vernon H. Stensland, secretary-treasurer. The meeting will be held May 28–31, 1981, in Sioux Falls, South Dakota. For further information, write Vernon H. Stensland, M.D., Central Plains Clinic Ltd., 2727 S. Kiwanis Ave., Sioux Falls, SD 57105.

AMERICAN ISRAELI OPHTHALMOLOGICAL SOCIETY: NEW OFFICERS

The new officers of the American Israeli Ophthalmological Society are as follows: Albert Hornblass, president; Ronald Burde, vice president; Myles Behrens, treasurer; and Philip Shelton, secretary. The following were elected to the Medical Advisory Board: Claes Dohlman, Kurt Gitter, Morton Goldberg, Herbert Katzin, Edward D. Norton, Steven M. Podos, Charles Schepens, David Soll, and Myron Yanoff.

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THE AMERICAN JOURNAL OF OPHTHALMOLOGY publishes original and timely articles dealing with clinical and basic ophthalmology. Every typescript submitted is evaluated by two or more scientific referees who recommend that the paper be (1) accepted as submitted, (2) returned for revision, or (3) rejected. Acceptance is determined by such factors as the originality, significance, and validity of the contribution; the suitability of the subject matter for subscribers of THE JOURNAL; and the editorial care with which the manuscript has been prepared. Manuscripts that require extensive editorial correction or mechanical preparation will be returned for that purpose.

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1. Fuxe, K., and Jonsson, G.: The histochemical fluorescence method for the demonstration of catecholamines. Theory, practice and application. J. Histochem. Cytochem. 21:293, 1973.

2. Bjorklund, A., Flack, B., and Owman, C.: Fluorescence microscopic and microspectrofluorometric techniques for the cellular localization and characterization of biogenic amines. In Berson, S. A. (ed.): Methods in Investigative and Diagnostic Encocrinology, vol. 1. London, North-Holland Publishing Co., 1972, p. 318.

3. Duke-Elder, S., and Leigh, A. G.: Diseases of the Outer Eye. Cornea and Sclera. In Duke-Elder, S. (ed.): System of Ophthalmology, vol. 8, pt. 2. St. Louis, C. V. Mosby, 1965, pp. 811-856.

Abbreviations of periodicals are listed in Index Medicus: Am. J. Ophthalmol.; Surv. Ophthalmol.; Br. J. Ophthalmol; and the like. If there is any doubt about an abbreviation, the name of the publication should be spelled out completely.

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A Manual of Style, 12th ed. Chicago, University of Chicago Press, 1969.

Strunk, W., Jr., and White, E. B.: The Elements of Style, 3rd ed. New York, Macmillan Co., 1979.

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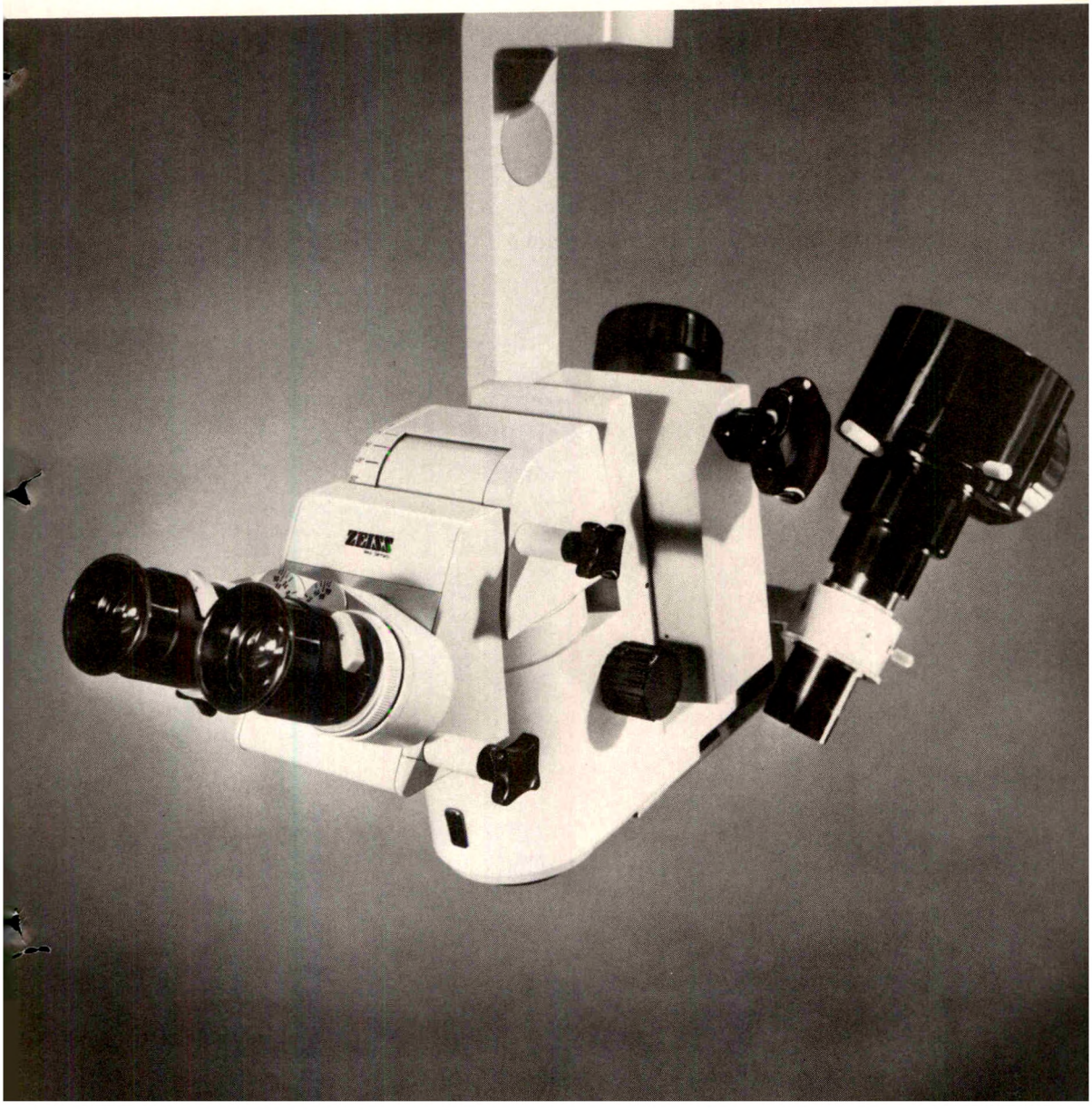
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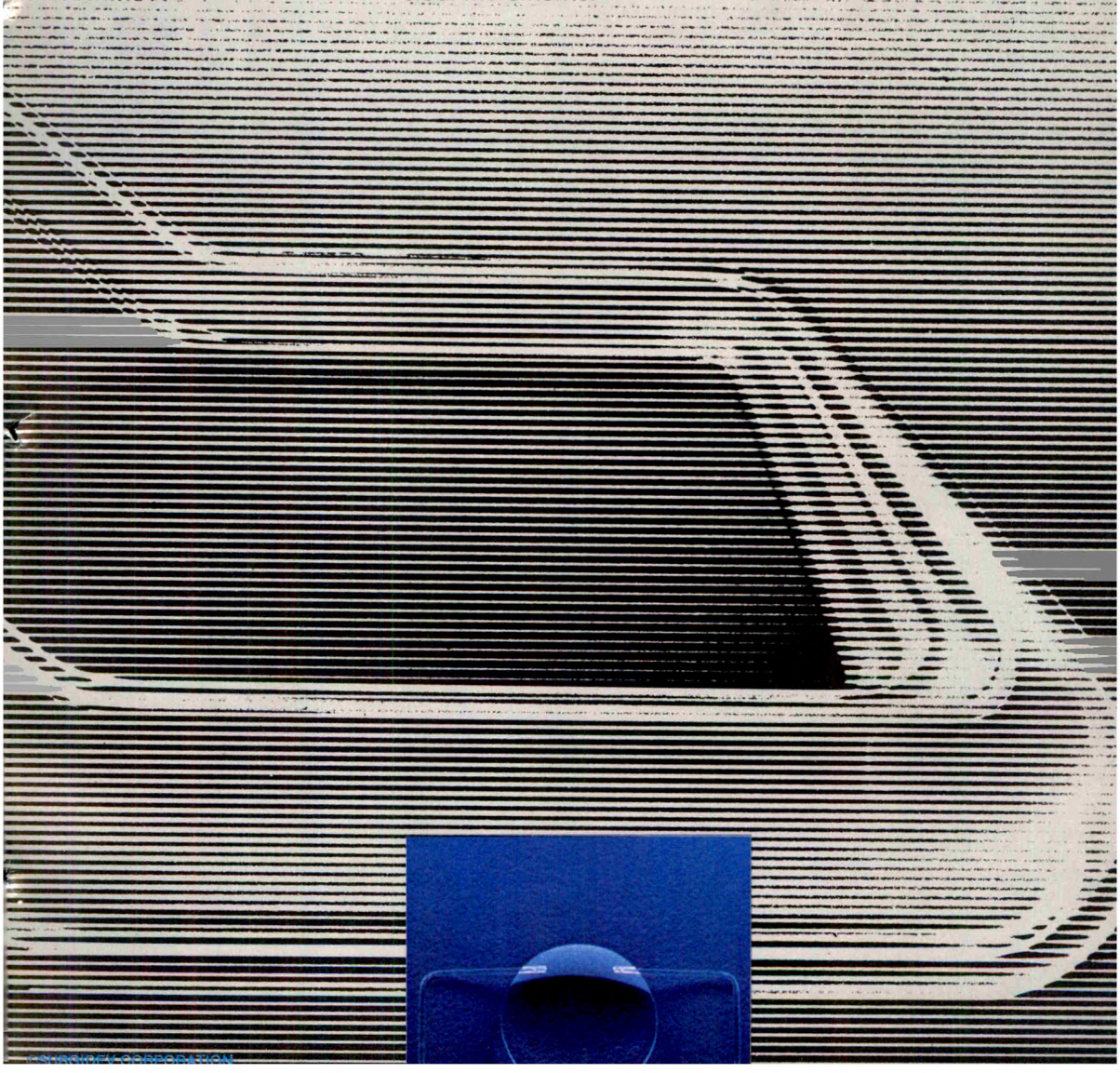
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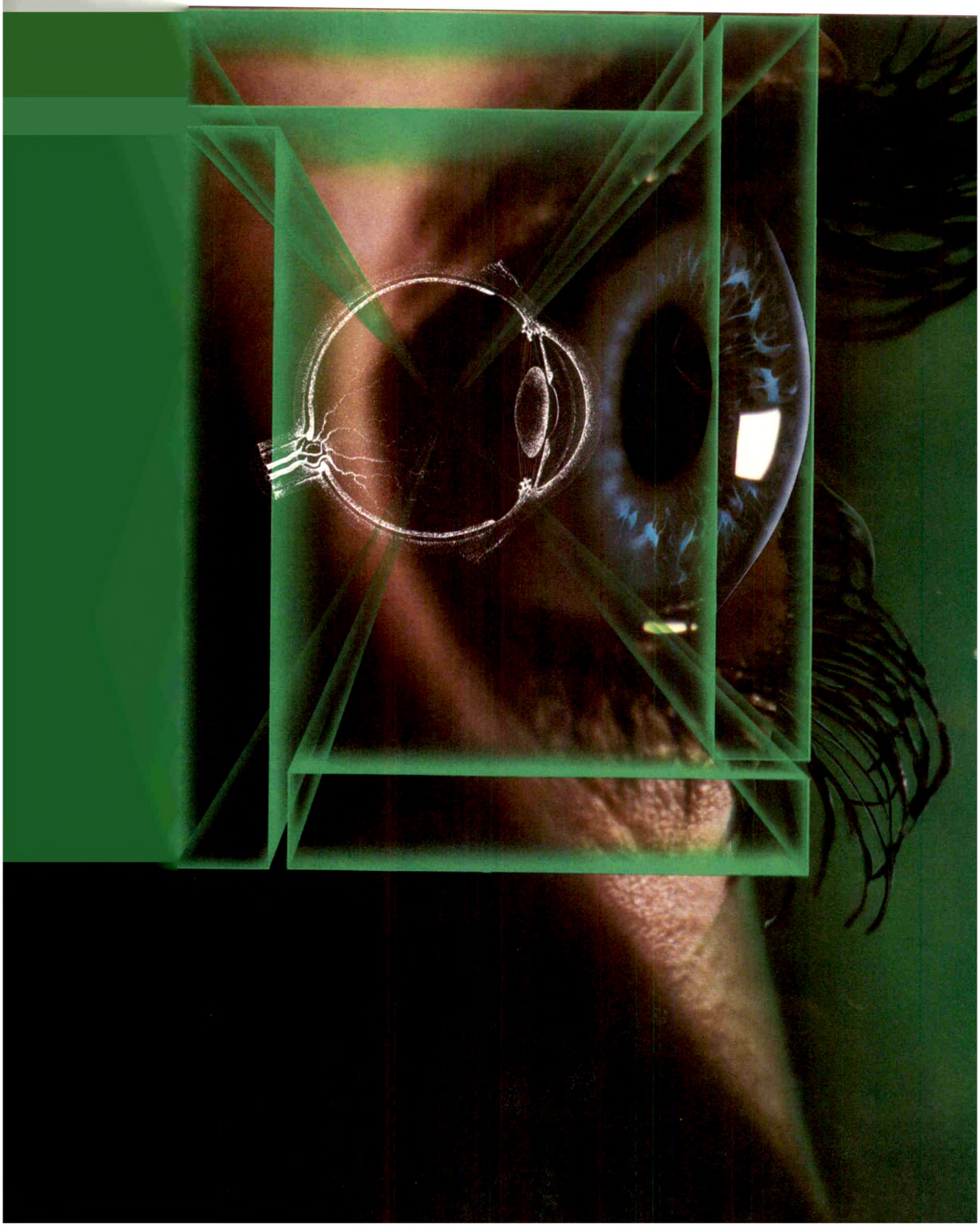
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2. Zimmerman, T. J., Ophthalmology Audio Digest, Vol. 17, No. 1

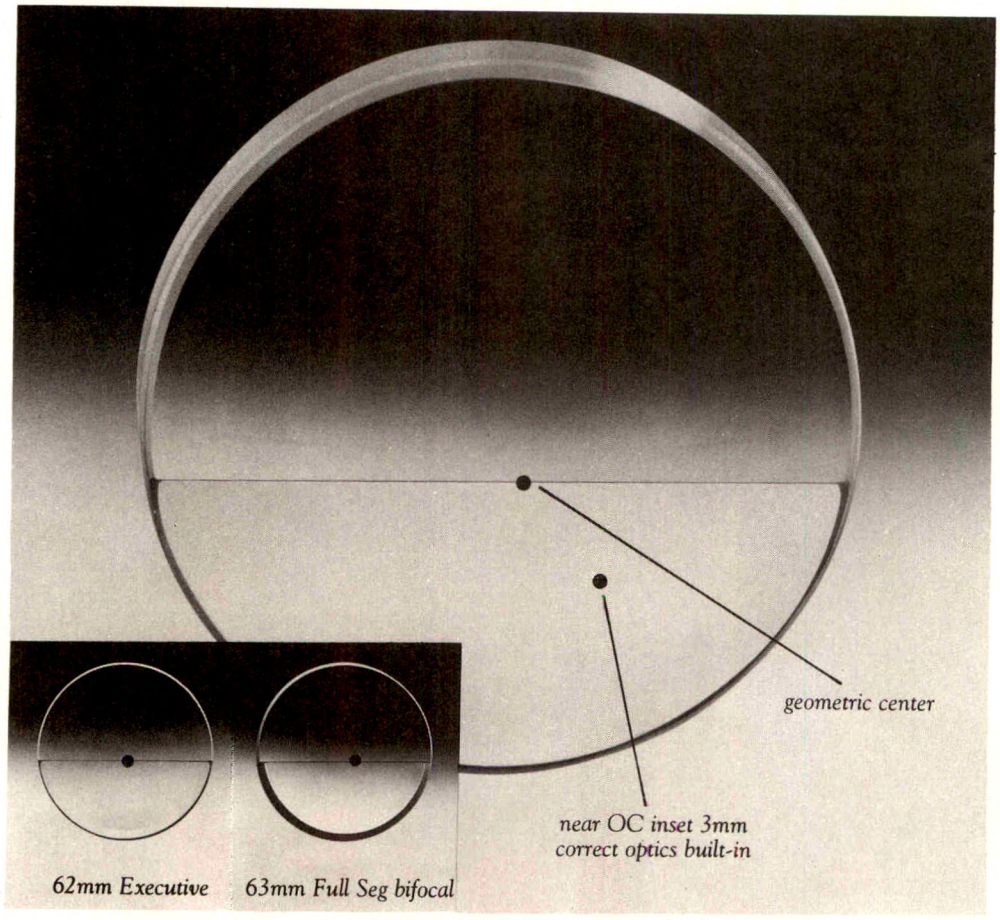
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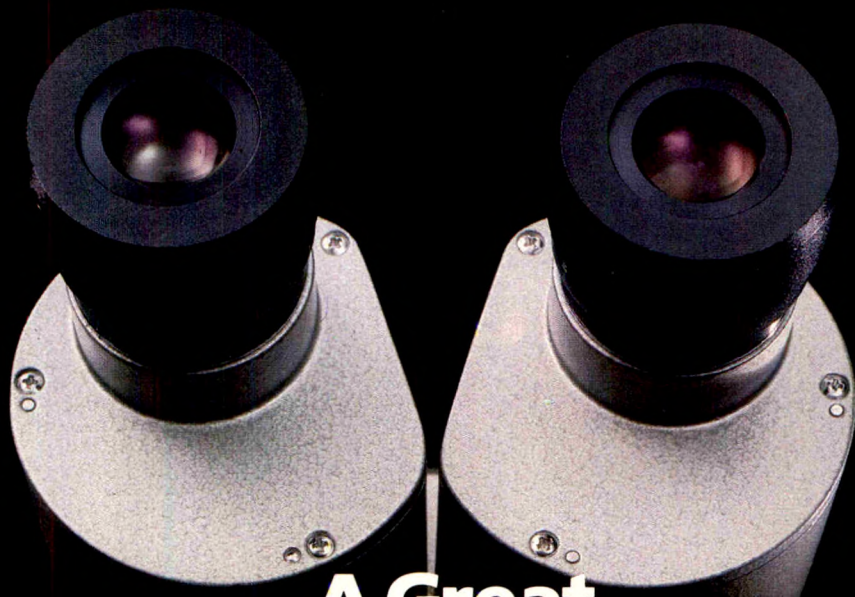
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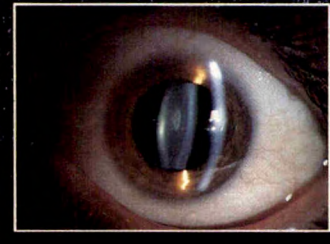
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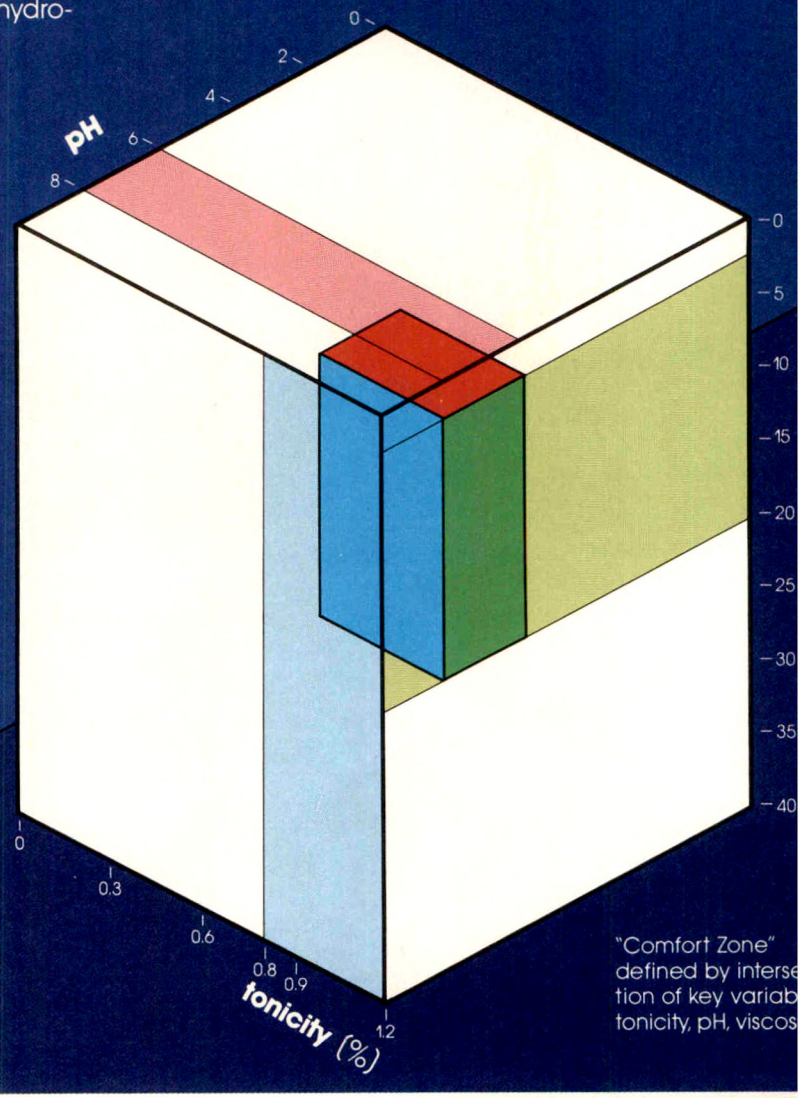
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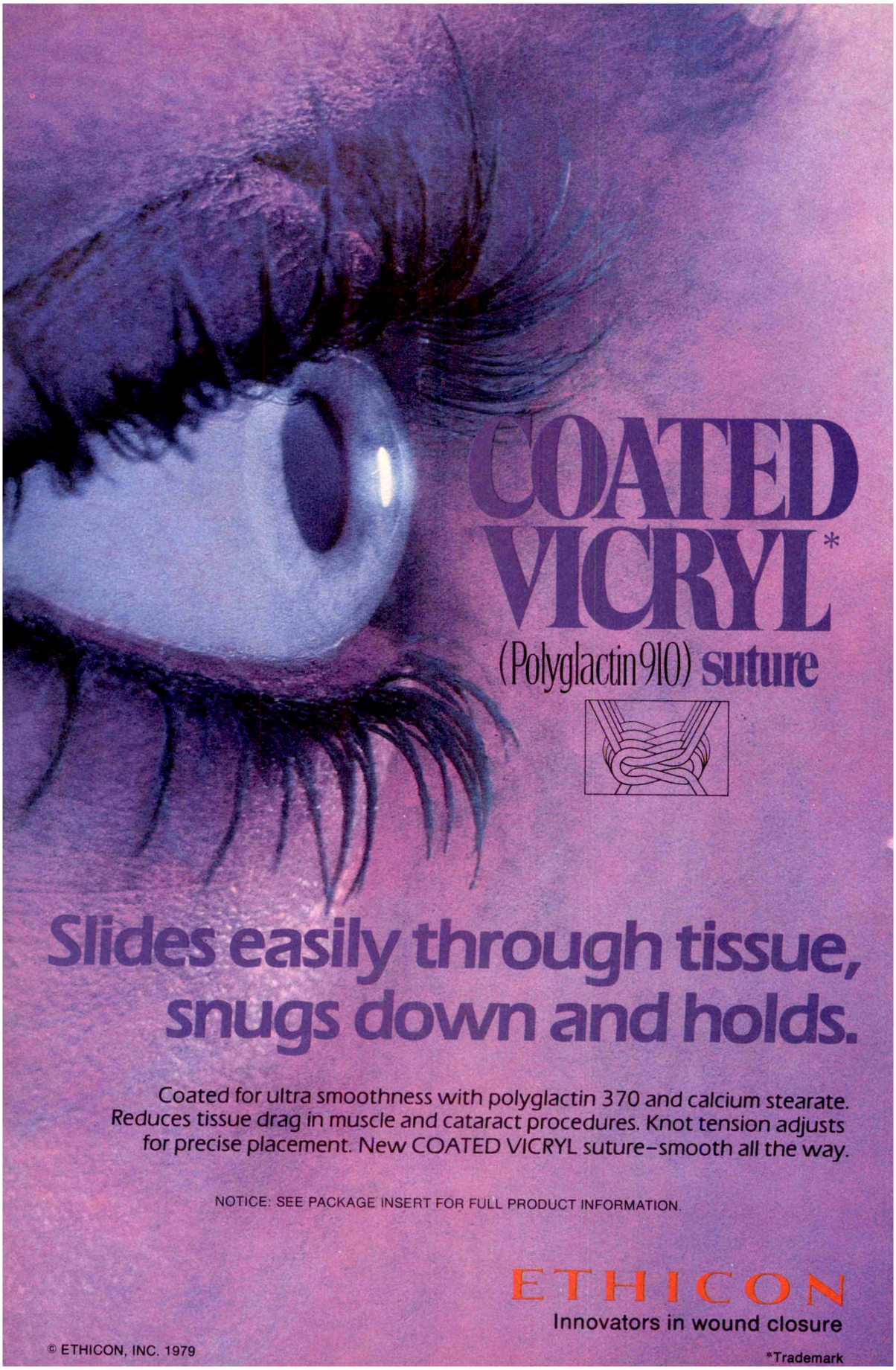
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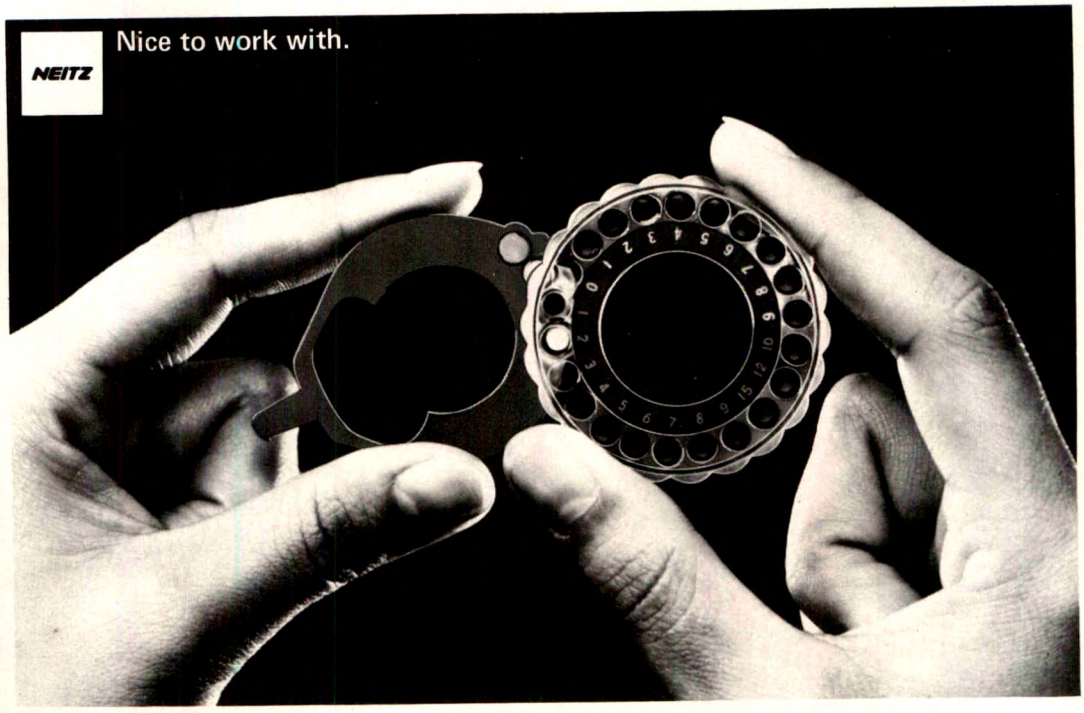
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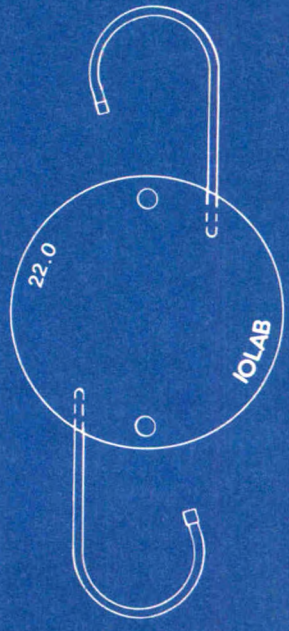
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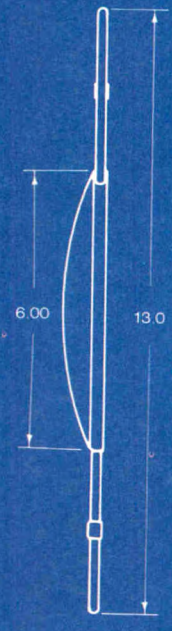


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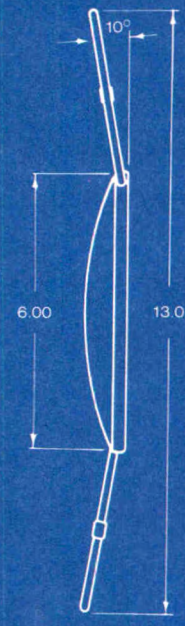
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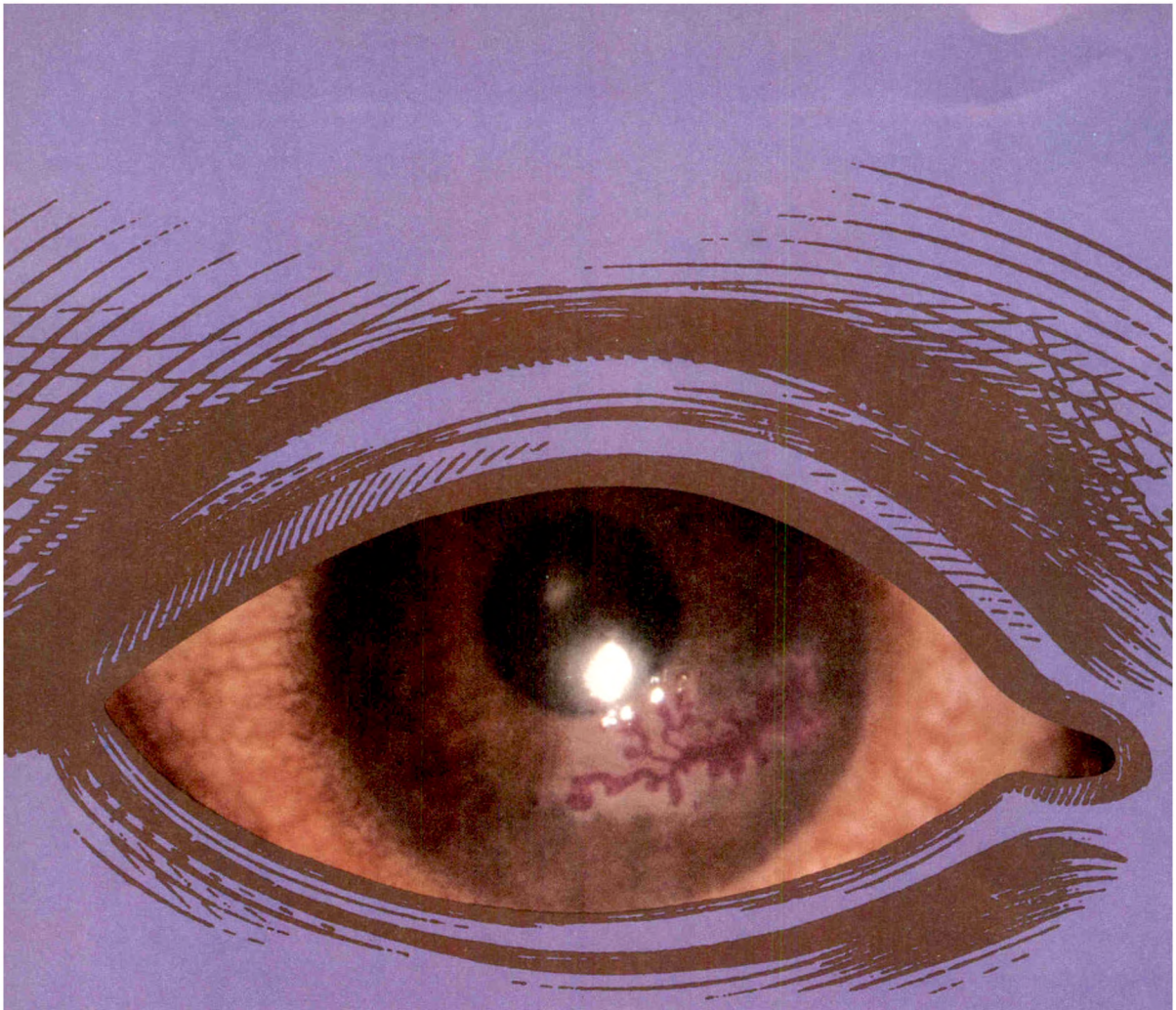
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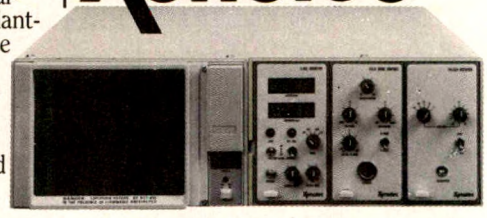
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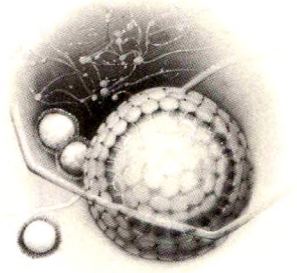
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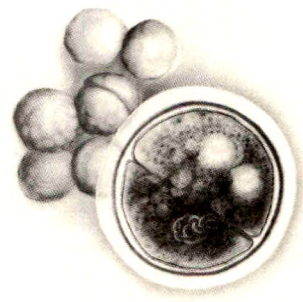
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REFERENCE: 1Antibacterial Agents. Monograph I. *Office Seminars in Ophthalmology.*℠ Irving H. Leopold, ed. Allergan Pharmaceuticals, 1976, p. 5.

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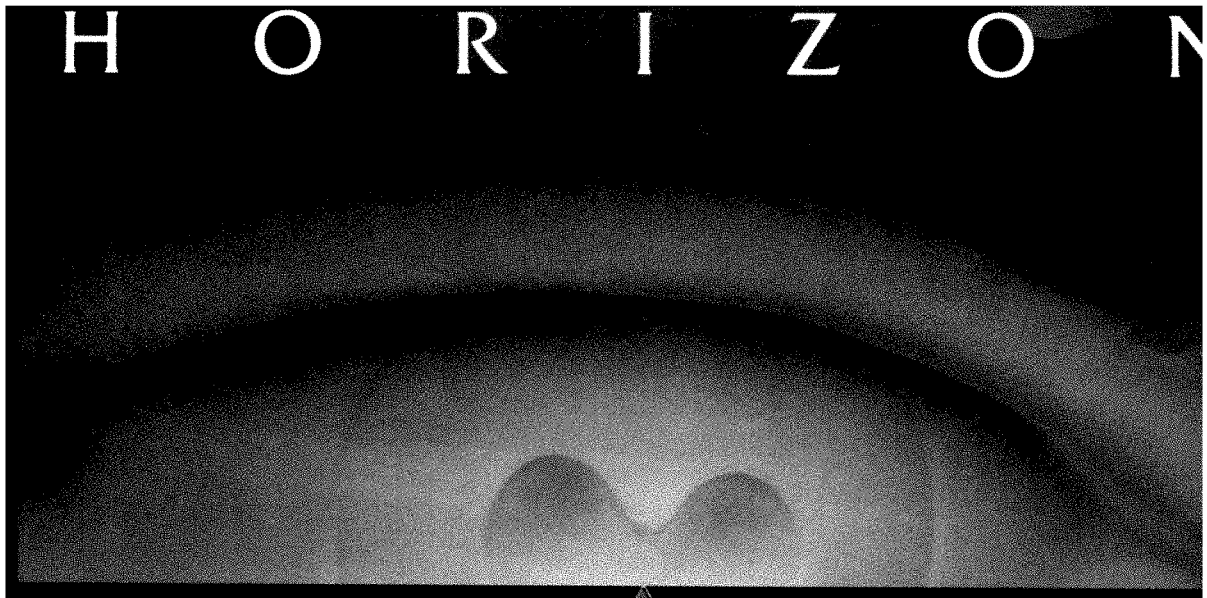
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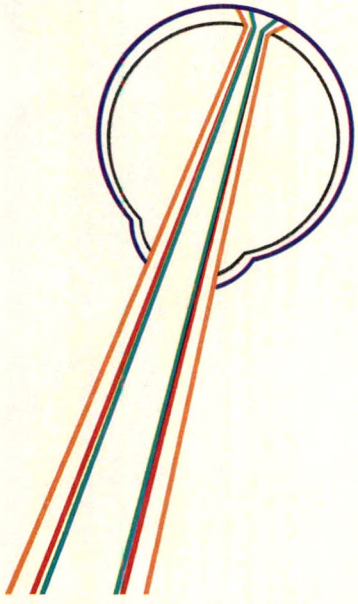
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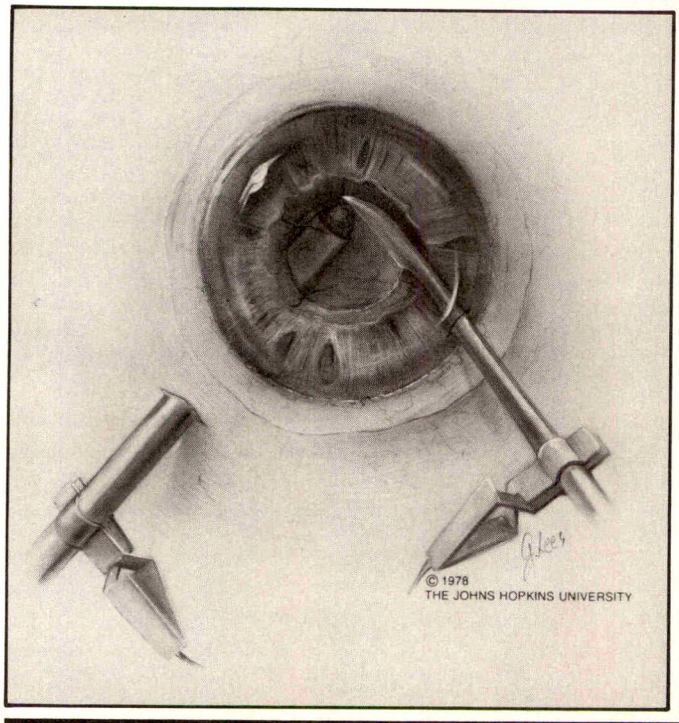
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Fig. 5-11. Pars plana and limbal incisions are com bined to facilitate bimanual, bipolar diathermy t treat vascularized pupillary membrane before exc sion with vitrectomy instrument. Vascularized tissue i positioned between instrument tips, which serve a electrodes to conduct diathermy current.

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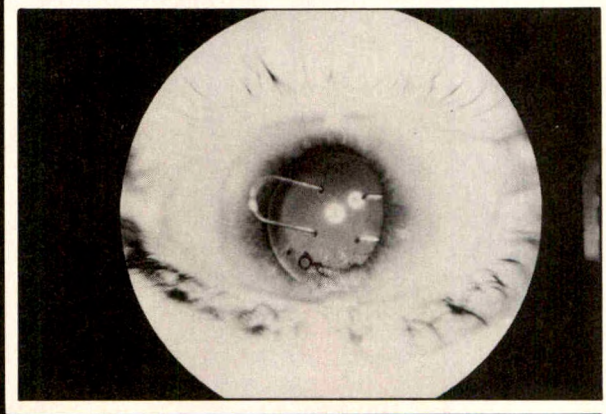


Fig. 109-1C, Iris suture lens with double loop dislocation and lens completely rotated. Lens is hanging by thin strand of iris and swings back and forth into vitreous. This is very difficult to repair. This lens was removed and replaced with Choyce-type lens.

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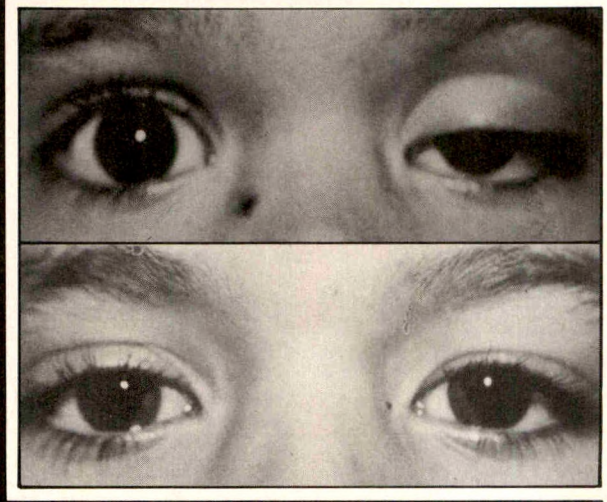


Fig. 13-20. A, Sever unilateral "congenital" ptosis. **B,** Apparent good result by fascia lata brow suspension.

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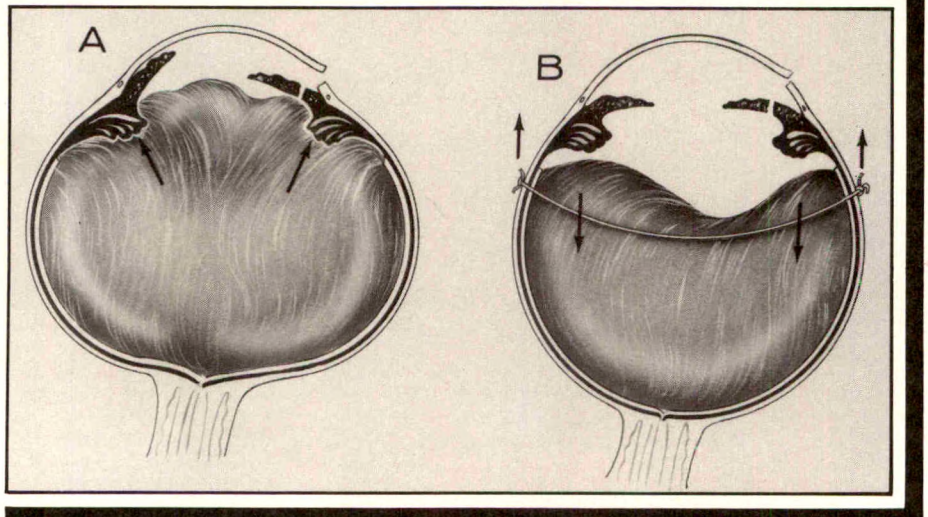
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Fig. 11-7. A, Collapse of globe after operative loss of vitreous. Angle of anterior chamber is closed by iris, which is pushed up from below. **B,** Application of Flieringa ring maintains shape of globe. Iris tends to fall back posteriorly as vitreous is removed, thus keeping angle open at all times.



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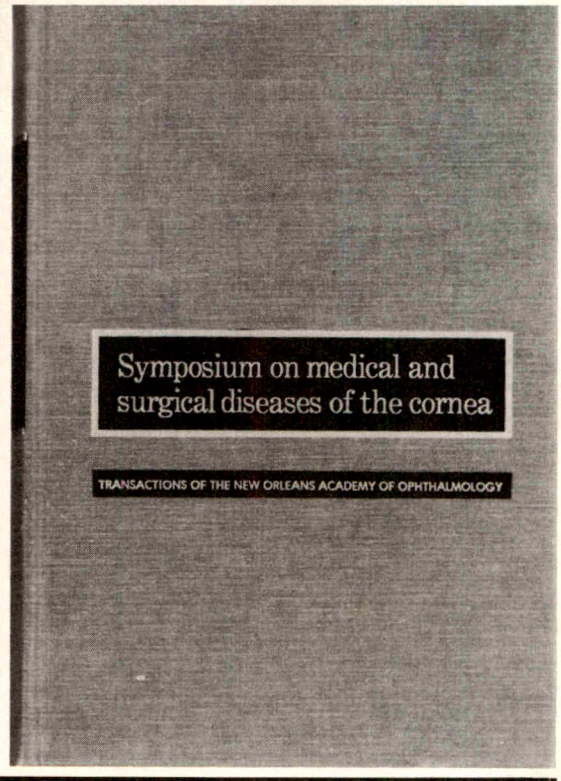
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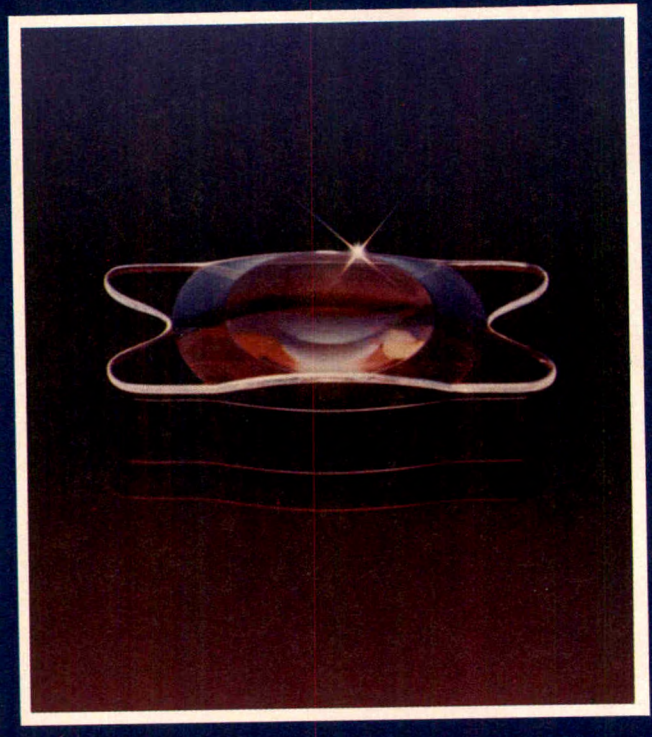
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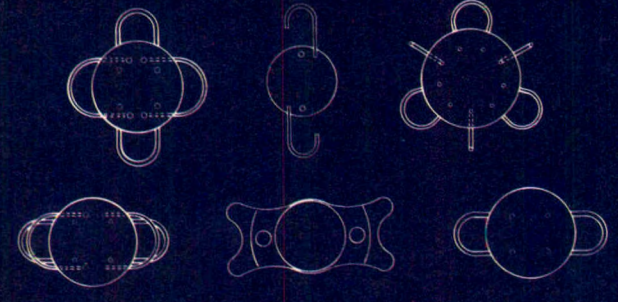
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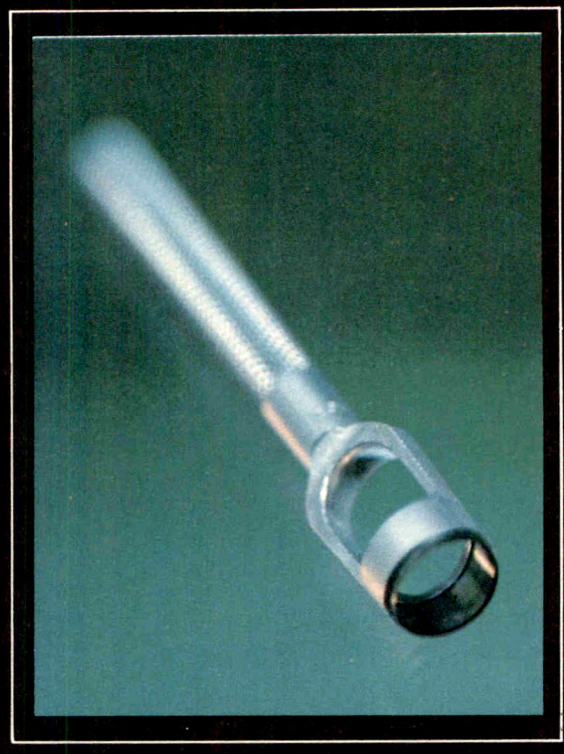
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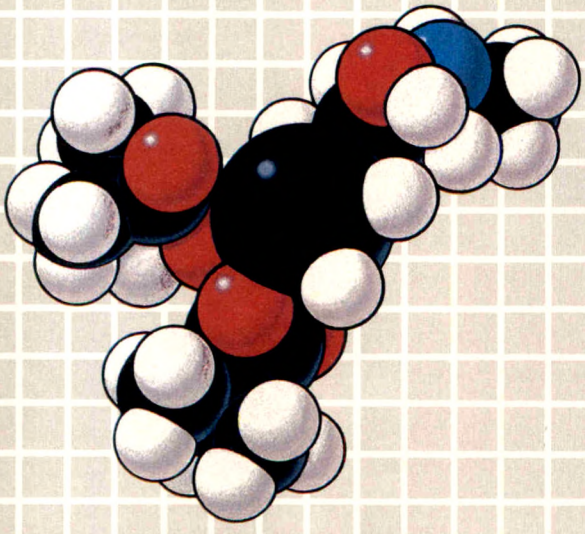
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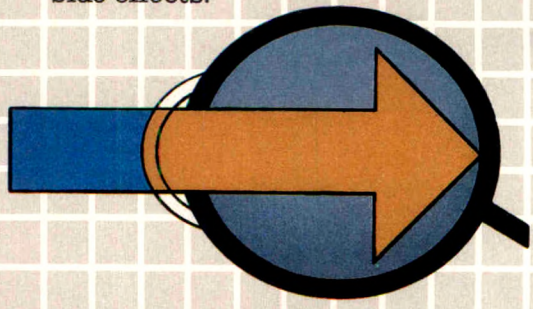
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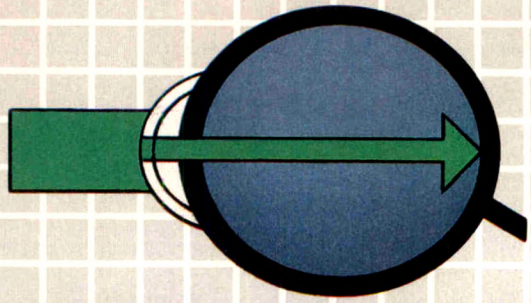
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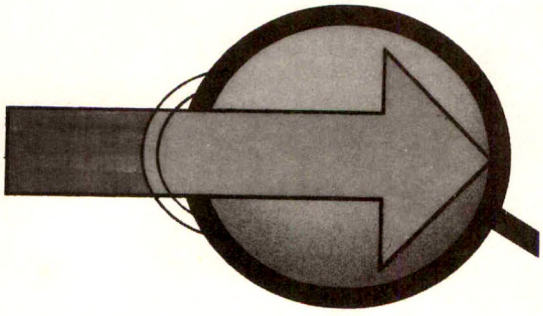
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1. Mandell AI. et al. Dipivalyl epinephrine: A new prodrug in the treatment of glaucoma. Ophthalmology 1978 Mar;85(3):268-275.
2. Kohn AN. et al. Clinical comparison of dipivalyl epinephrine and epinephrine in the treatment of glaucoma. Am J Ophthalmol 1979;87:196-201.
3. Kass MA. et al. Dipivefrin and epinephrine treatment of elevated intraocular pressure: a comparative study. Arch Ophthalmol 1979 Oct;97(10):1865-1866.
4. Allergan Report Series No. 168, 1980.
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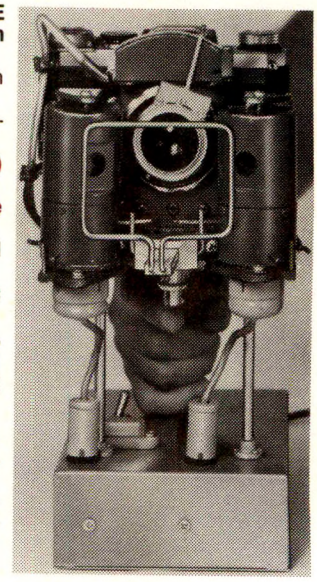
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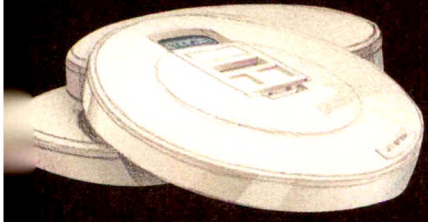
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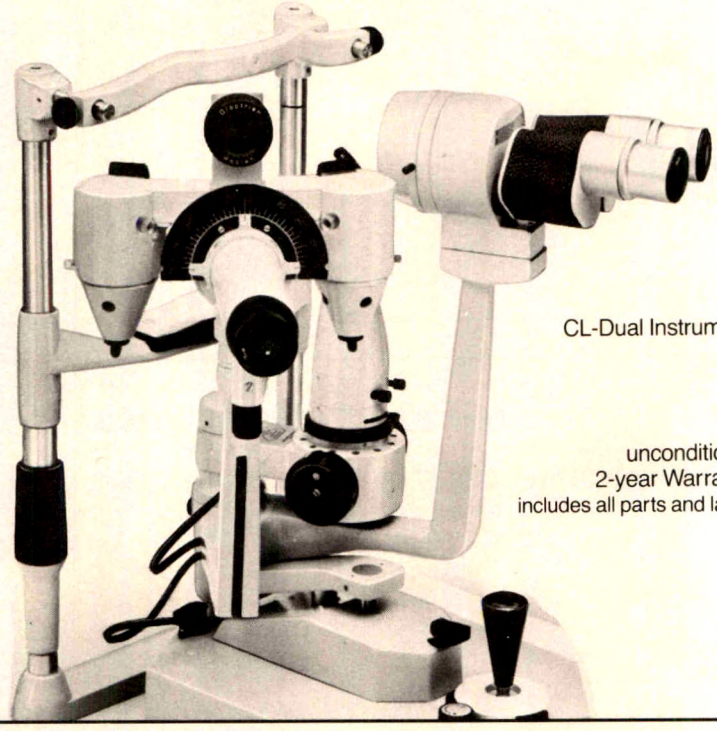
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Permalens® (perfilcon A)

Hydrophilic Contact Lens For Extended Wear

DESCRIPTION

The **Permalens*** (perfilcon A) Hydrophilic Contact Lens is a hemispherical shell which covers the cornea and may cover a portion of the adjacent sclera. The perfilcon A material is a terpolymer of 2-hydroxyethyl methacrylate, N-vinyl-2-pyrrolidone, and methacrylic acid, with ethyleneglycol dimethacrylate as a crosslinking agent. When fully hydrated in buffered normal saline, the water content is 71%. The refractive index is 1.38 and light transmission is at least 96% for the visible light spectrum. For the minus lenses the base curve ranges from approximately 7.7 mm to 8.6 mm, with a diameter of approximately 14.0 mm. Center thickness ranges from 0.1 mm to 0.24 mm. For the plus lenses the base curve ranges from approximately 8.0 mm to 8.9 mm, with a diameter of approximately 14 mm to 14.5 mm. Center thickness ranges from 0.3 mm to 0.43 mm. The gas permeability of **Permalens*** (perfilcon A) Hydrophilic Contact Lens has been determined to be: 42.0×10^{-11} $(cm^2/sec)ml\ O_2/ml \times mmHg$ at 36°C. The percent equivalent oxygen performance has been determined to be approximately 11.5% for a **Permalens*** (perfilcon A) Hydrophilic Contact Lens of 0.17 mm center thickness.

PERMALENS* (perfilcon A) Hydrophilic Contact Lenses are available in powers from − 20.00 to + 35.00 diopters.

ACTIONS

In its hydrated state, the **Permalens*** (perfilcon A) Hydrophilic Contact Lens is soft and pliable. When placed on the eye, the hydrated lens acts as a refracting medium to compensate spherical ametropias.

INDICATIONS (Uses)

The **Permalens*** (perfilcon A) Hydrophilic Contact Lens is indicated for extended wear use to improve vision in patients with healthy eyes who are not aphakic and who have no more than 2 diopters of corneal astigmatism.

CONTRAINDICATIONS (Reasons not to use)

The **PERMALENS*** Hydrophilic Contact Lens should not be dispensed for vision improvement if the patient has any of the following conditions:

- Acute and subacute inflammation of the anterior segment of the eye.
- Any eye disease which affects the cornea or conjunctiva.
- Any active corneal infection: purulent (pus) bacterial, fungal, or viral infection.
- Insufficiency of lacrimal secretion (dry eyes).
- Corneal hypoesthesia (reduced corneal sensitivity).
- Any systemic disease which may affect the eye or be exaggerated by wearing contact lenses.
- Allergy to any ingredient in the solutions necessary for care of the lenses. **Thimerosal,** used as a preservative in many solutions, is one cause of such allergy.

WARNINGS

SERIOUS DAMAGE TO THE EYE AND LOSS OF VISION MAY RESULT FROM PROBLEMS WITH CONTACT LENSES. IMMEDIATELY CONSULT YOUR EYE CARE PRACTITIONER TO IDENTIFY THE CAUSE OF ANY UNEXPLAINED EYE DISCOMFORT, WATERING, VISION CHANGE, OR REDNESS OF THE EYE AND TO BEGIN ANY NECESSARY TREATMENT.

PRECAUTIONS

You must take the following precautions **to prevent damage to your eyes or to your Permalens*** Hydrophilic Contact Lenses:

- Always wash and rinse your hands before handling your lenses. Eye irritation may result if cosmetics, soaps, lotions, creams, or deodorants come in contact with your lenses.
- Avoid using aerosol products such as hair spray while wearing your lenses. If a spray is used, keep your eyes closed until the spray has settled.
- Fluorescein should not be used while the lens is on the eye.
- Always follow the lens care system recommended for your **Permalens*** Hydrophilic Contact Lenses. (See Directions).
- Do not mix or alternate heat (thermal) and chemical (not heat) lens care systems.
- Use **only** the recommended lens care solutions. Do not use saliva or anything other than the approved solutions to wet (hydrate) the lenses. (See Directions).
- Avoid all harmful or irritating vapors and fumes while wearing your lenses.
- Do not swim with your lenses in place.
- Never use tweezers or other implements to remove the lens from the lens container. Pour the lens into your hand.
- Do not touch the lens with your fingernails.

ADVERSE EFFECTS (Possible problems)

The following adverse effects have been reported with the use of soft contact lenses, including **Permalens*** Hydrophilic Contact Lenses:

- Discomfort, or feeling of something in the eye (a foreign body, or an abrasion, or a scraped area)
- Eye infection
- Stinging, burning, itching (irritation)
- Excessive watering of the eye (tearing)
- Redness of the eyes
- Dry eyes
- Reduced sharpness of vision (poor visual acuity)
- Blurred vision
- Sensitivity to light (photophobia)

What to do if a problem occurs:

- First remove the lens.
- If the discomfort or problem stops, then look closely at the lens.
- If the lens is chipped, cracked, or otherwise damaged: **DO NOT** put the lens back on your eye. Return the lens to the storage case. Then contact your eye care practitioner.
- If the lens has dirt, an eyelash, or other foreign body on it, thoroughly clean and disinfect the lens using the method recommended by your eye care practitioner. (See Directions)
- If the problem does not stop, or if it recurs when you replace the lens, contact your eye care practitioner immediately.

DIRECTIONS (How to care for your lenses)

General Instructions

- **BOTH CLEAN AND DISINFECT THE LENSES EACH TIME THEY ARE REMOVED FROM YOUR EYES.**
- First wash and rinse your hands thoroughly.
- Use only the recommended system of lens care, either heat (thermal) or chemical (not heat).
- **DO NOT ALTERNATE OR MIX LENS CARE SYSTEMS.**
- Use the right solutions (see chart of approved solutions).
- After disinfection leave the lenses in the unopened storage case until ready to put the lenses on your eyes.
- Empty all of the solution from the storage case as soon as you remove your lenses.

CLEANING

- Place 3 drops of PREFLEX* Sterile Cleaning Solution on each surface of the lens and thoroughly clean between thumb and forefinger for at least 20 seconds to remove mucus and other deposits.
- Rinse both surfaces thoroughly for at least 10 seconds with a steady stream of PERMASOL™ Rinsing and Storage Solution, UNISOL* Preservative-Free Saline Solution, or FLEX-CARE* Sterile Solution for Rinsing, Storing and Disinfecting, depending upon the disinfection method used. (See Approved Solutions Chart.)

DISINFECTING

Heat (thermal) Lens Care System

- First clean and rinse the lenses. (See Cleaning).
- Place each lens in the correct section (Right or Left) of the storage case or vial and fill with the recommended rinsing solution; be sure the lens is completely covered.
- Close the storage case tightly.
- Place the storage case in the recommended low-heat disinfection unit.
- Disinfect according to the directions for operation of the low-heat disinfection unit.
- After disinfection, store the lenses in the **unopened** storage case until ready to place on the eyes.
- Empty the storage case after removing the lenses and allow the case to air dry.

EMERGENCY (ALTERNATE) METHOD FOR HEAT (THERMAL) LENS CARE SYSTEM

- If a low-heat disinfection unit is not available, place the storage case containing the lenses in a pan of already boiling water for 10 minutes (at altitudes above 7000 feet boil for 15 minutes).
- Remove the pan from the heat and allow to cool for 30 minutes to complete disinfection.
- USE OF THE RECOMMENDED LOW-HEAT DISINFECTION UNIT MUST BE RESUMED AS SOON AS POSSIBLE.

Chemical (not heat) Lens Care System

- First clean and rinse the lenses. (See Cleaning)
- Place each lens in the correct chamber (Right or Left) of your empty storage case.
- Fill the storage case with fresh FLEX-CARE* disinfection solution, completely covering the lens.
- Close the storage case tightly.
- NEVER HEAT THE PERMALENS* IN THE DISINFECTING SOLUTION.
- Leave the lenses in the solution for at least 4 hours.
- Remove each lens separately.
- Rinse thoroughly with FLEX-CARE* before putting the lens on the eye.

Lens Care Solutions

A complete list of solutions approved for use with the **Permalens*** (perfilcon A) Hydrophilic Contact Lens is given in the following table.

APPROVED SOLUTIONS FOR USE WITH PERMALENS* (PERFILCON A) HYDROPHILIC CONTACT LENS

SYSTEM PROCESS	HEAT (THERMAL) DISINFECTION SYSTEM	CHEMICAL (NOT HEAT) DISINFECTION SYSTEM
CLEANING	PREFLEX®	PREFLEX®
RINSING	PERMASOL™ UNISOL™	*FLEX-CARE®
DISINFECTING	PERMASOL™ used with approved low-heat disinfection unit. UNISOL™ used with approved low-heat disinfection unit	*FLEX-CARE®
LUBRICATING	CLĒRZ® ADAPETTES®	CLĒRZ® ADAPETTES®

*Chemical solution which disinfects. NOT TO BE USED WITH HEAT. The use of FLEX-CARE® with heat has a harmful effect on the lenses.

Product List:

ADAPETTES®	Sterile Lubricating Solution (Burton, Parsons & Co., Inc.)
CLĒRZ®	Lubricating and Rewetting Eye Drops (CooperVision, Inc.)
FLEX-CARE®	Rinsing, Storing and Disinfecting Solution (Burton, Parsons & Co., Inc.)
PERMA-CASE™	Contact Lens Storage and Carrying Case (CooperVision, Inc.)
PERMASOL™	Rinsing and Storage Solution (CooperVision, Inc.)
PREFLEX®	Sterile Cleaning Solution (Burton, Parsons & Co., Inc.)
UNISOL™	Preservative-Free Saline Solution, for rinsing and storing in the heat system (CooperVision, Inc.)

How to care for a dried out (dehydrated) lens

- Always keep the lens completely covered in the recommended storage solution when the lens is not being worn, to prevent the lens drying out.
- If a **Permalens*** Hydrophilic Contact Lens is left exposed to the air for 30 minutes or longer it will become dry and brittle.

To rewet your lens

- Handle a dried-out lens with care.
- First, place the lens in its storage case, and soak for at least one hour in one of the recommended rinsing and storage solutions: PERMASOL™ or UNISOL™, when using the heat (thermal) system, FLEX-CARE* when using the chemical (not heat) system.
- Next, clean and disinfect the rewetted (rehydrated) lens, using the lens care system recommended by your eye care practitioner. (See Directions)

DOSAGE AND ADMINISTRATION

Fitting Information

In general, the eye care practitioner will use conventional methods to fit the **Permalens*** (perfilcon A) Hydrophilic Contact Lens for vision correction. For a detailed description of the recommended fitting procedure refer to the **Permalens*** Fitting and Information Guide, available from CooperVision, Inc. (address below).

Dosage (Wearing schedule)

NOTE: Not every patient is able to wear the **Permalens*** Hydrophilic Contact Lens on an extended wear basis even if able to wear a lens on a daily wear basis. The eye care practitioner should determine the wearing schedule. Regular checkups, as determined by the eye care practitioner, are extremely important.

The eye care practitioner may recommend a limited wearing time for the first two days. However, clinical studies have established that a wearer's eyes adapt readily to wearing the **Permalens*** Hydrophilic Contact Lens on an extended wear basis, and that limited initial wear is usually not necessary.
The lens should be removed for cleaning and disinfection at least once every two weeks (14 days).

HOW SUPPLIED

Each lens is supplied sterile in a sealed glass vial containing a sterile buffered isotonic salt solution. The vial is marked with the dioptric power, base curve, diameter, lot number, and expiration date of the lens.

CAUTION: FEDERAL LAW PROHIBITS DISPENSING WITHOUT PRESCRIPTION

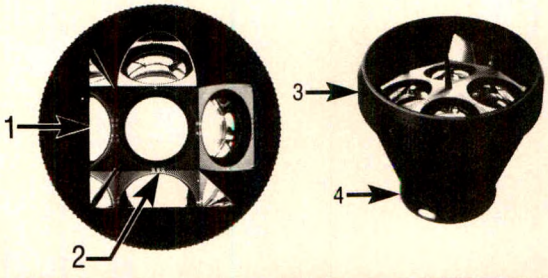
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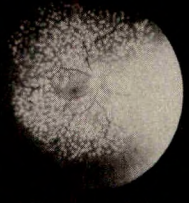
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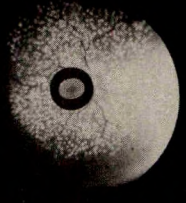
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- Either anti-reflective glass coating for laser use or highly polished plastic surface for office use.
- The 62° mirror is positioned for gonioscopy and photocoagulation of the chamber angle.



Panretinal Photocoagulation View with Laser Lens



Panretinal Photocoagulation View with Other Fundus Lenses (Note Unseen and Untreated Ring.)

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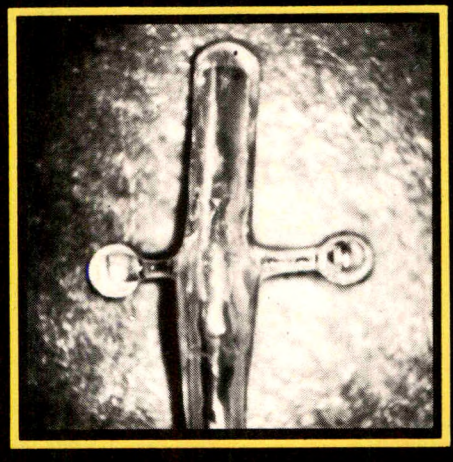
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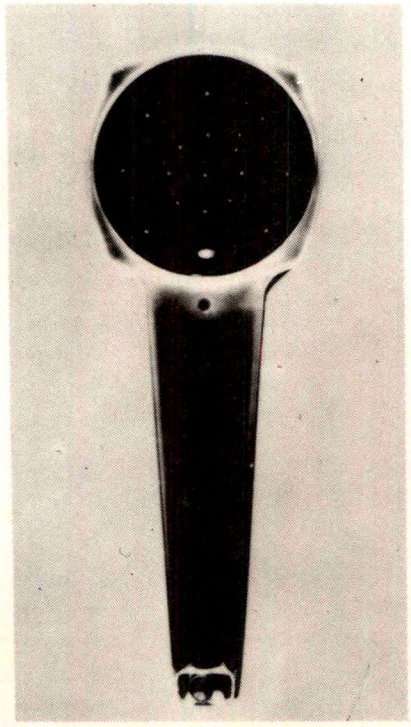
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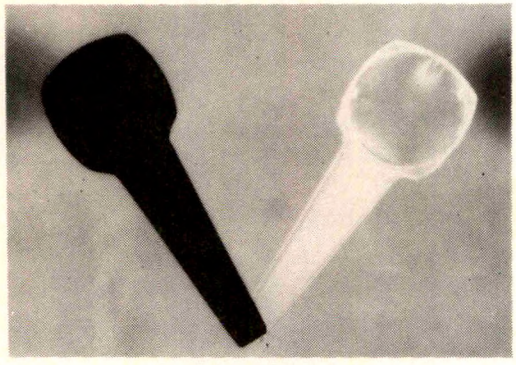
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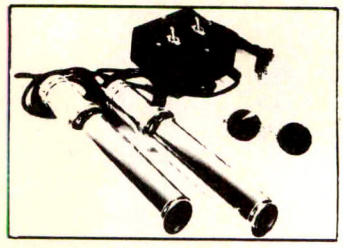
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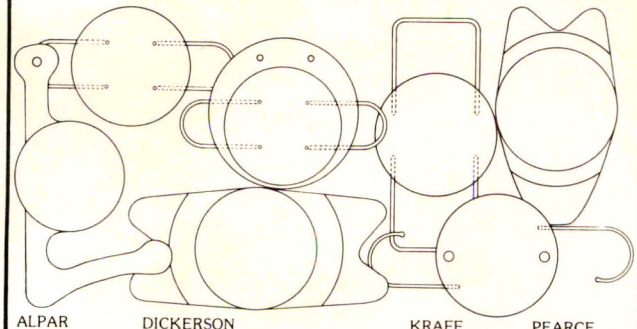
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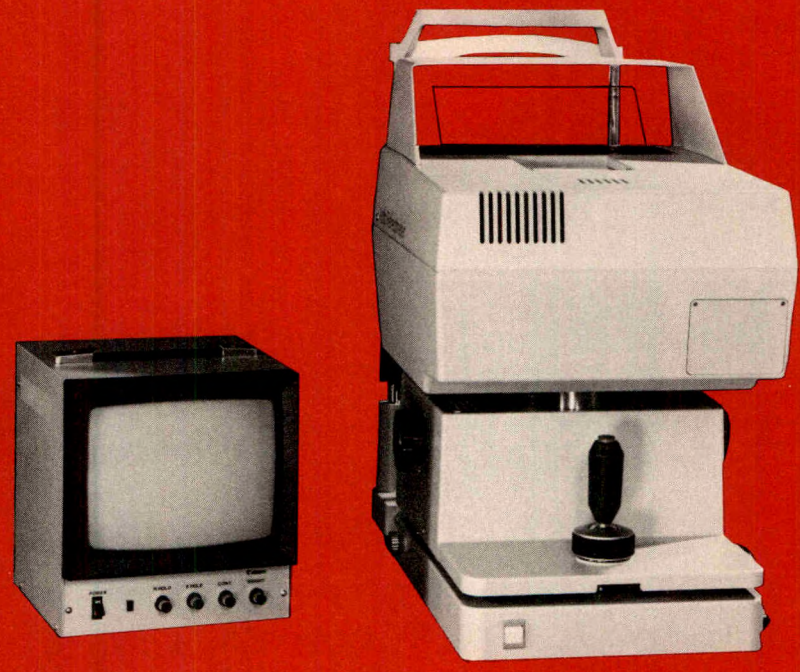
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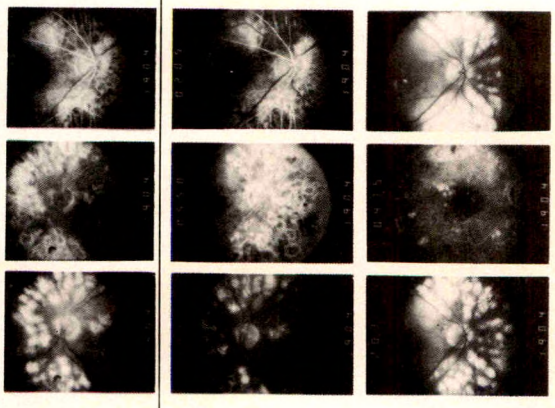
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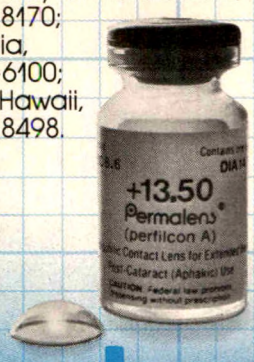
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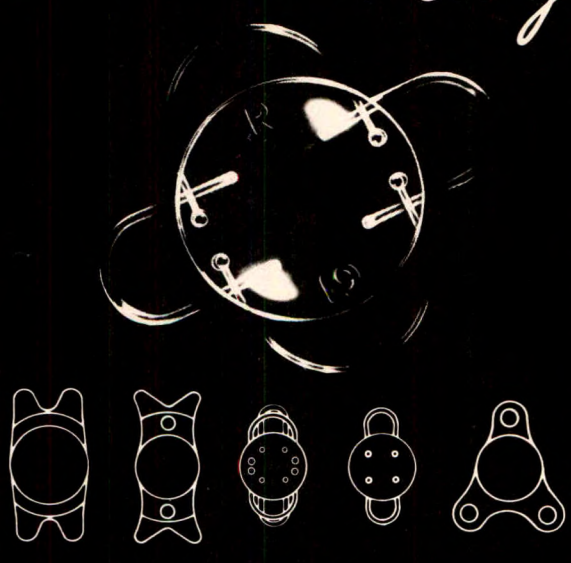
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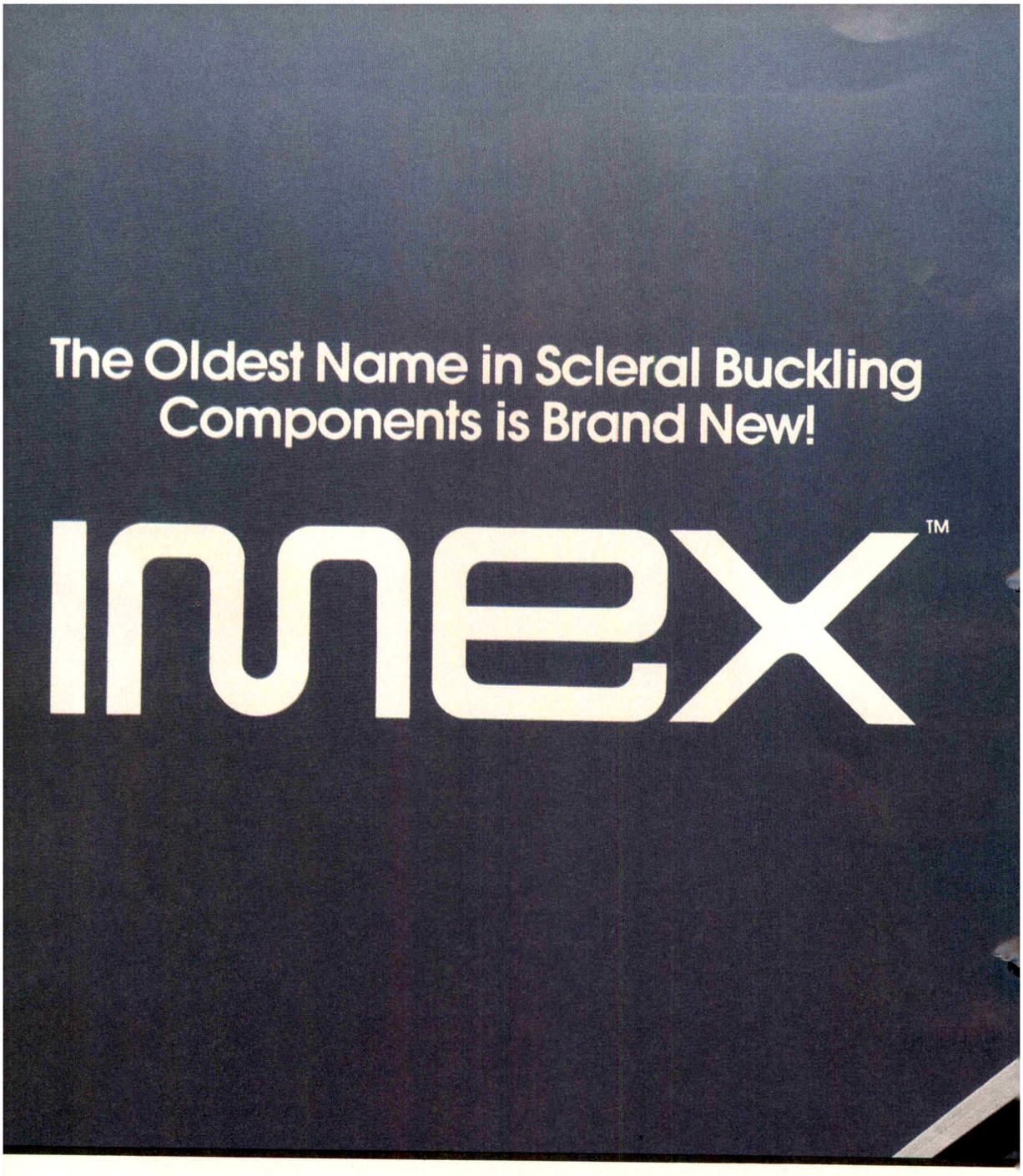
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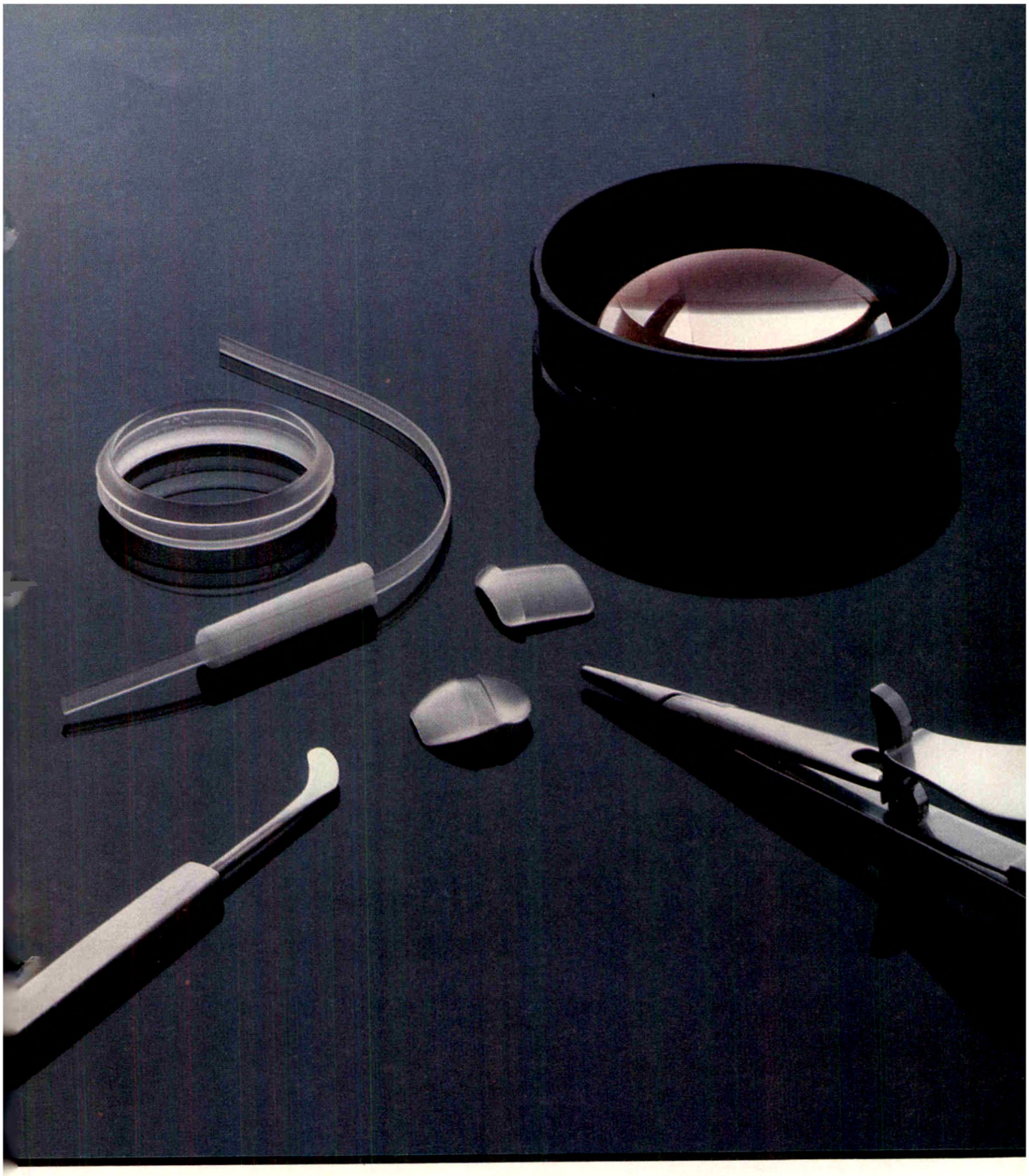
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DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′-deoxyuridine.

VIROPTIC sterile opthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

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CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

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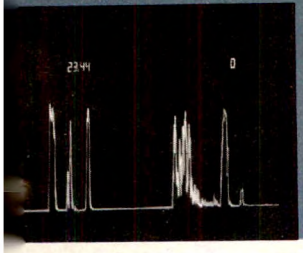
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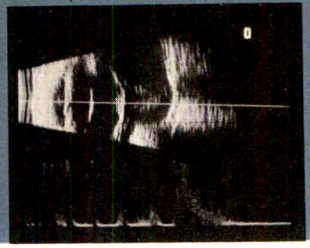


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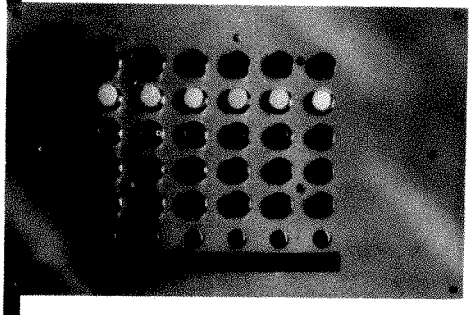
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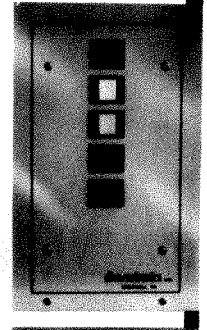
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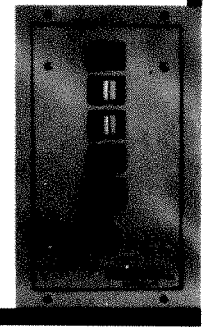
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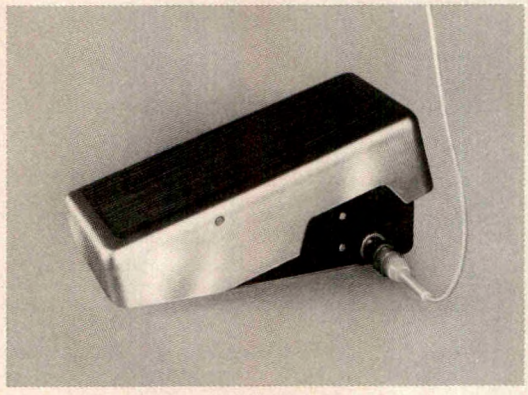
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ELECTRON MICROSCOPIC FEATURES OF EXPERIMENTAL CHOROIDAL NEOVASCULARIZATION

D. B. ARCHER, F.R.C.S., AND T. A. GARDINER, F.R.C.S.

Belfast, Northern Ireland

We produced choroidal neovascularization in the rhesus monkey by diminishing the blood supply to the inner retina and producing defects in Bruch's membrane by photocoagulation. The neovascular fronds which developed either infiltrated the subretinal space or proliferated through necrotic and gliotic retina into the vitreous cavity. Sequential electron microscopic sections of neovascular fronds in the subretinal space demonstrated that the advancing capillary sprouts were composed of primitive endothelial tubes surrounded by pericytes and enmeshed in a loose basement-membrane-like substance. More mature capillaries downstream to the growing front resembled normal choriocapillaris and displayed endothelial fenestrations and endothelial-pericyte membranous contacts. Large neovascular fronds developed major feeding vessels that closely resembled normal small choroidal arteries and veins. Retinal pigment epithelial cells in various guises were in constant association with proliferating neovascular networks.

Choroidal neovascularization has been produced in rhesus monkeys by diminishing the blood supply to the inner retina and simultaneously producing defects in Bruch's membrane by intense photocoagulation. Fourteen of 46 eyes so treated developed neovascular fronds identified by fluorescein angiography and light microscopy. The new choroidal vessels invaded the retina through defects in Bruch's membrane at the sites of photocoagulation marks. In nine eyes the new choroidal vessels infiltrated the subretinal space for various distances from their point of entrance; some developed major feeding vessels. In five eyes the new vessels passed directly through necrotic and attenuated retina at photocoagulation scars to invade the vitreous cavity and ramify in the preretinal region. The general morphologic, light microscopic, and fluorescein angiographic features of these new choroidal vessels have been described in another article.[1] In this study we describe the electron microscopic features of the new choroidal vessels and present a hypothesis as to their mode of propagation.

From the Department of Ophthalmology, The Queen's University of Belfast, Eye and Ear Clinic, Royal Victoria Hospital, Belfast, Northern Ireland. This work was supported in part by a grant from the Royal National Institute for the Blind, London (Dr. Archer).

Reprint requests to D. B. Archer, F.R.C.S., Department of Ophthalmology, Eye and Ear Clinic, Royal Victoria Hospital, Belfast, Northern Ireland.

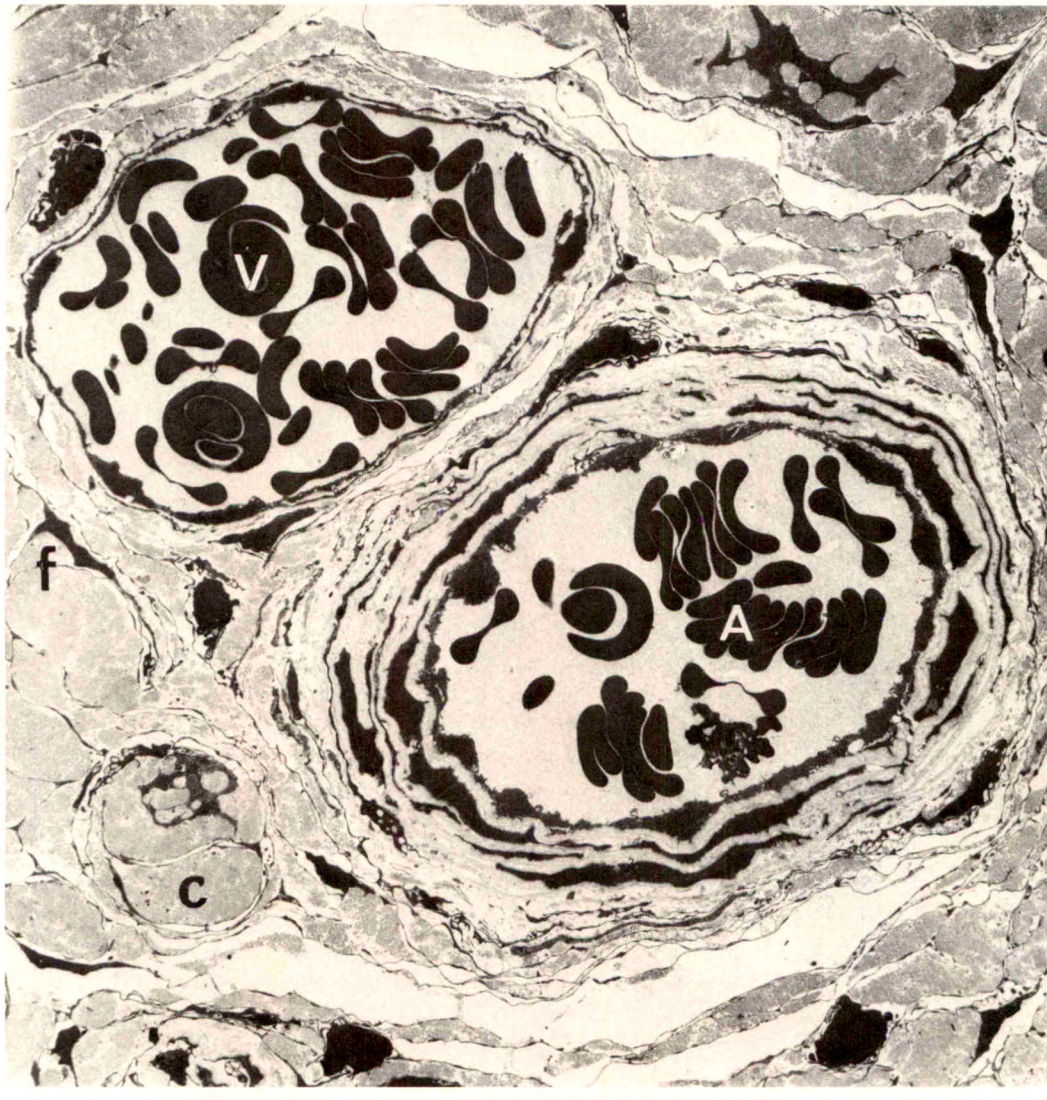


Fig. 1 (Archer and Gardiner). Feeding artery (A) and vein (V) service a network of new choroidal capillaries in the subretinal space. Endothelial cell nuclei bulge into the lumen. A well-developed muscularis is present in the artery. Transversely sectioned collagen bundles (C) and fibroblasts (f) are present in the adventitia (×2,000).

Material and methods

The techniques used for identification, preservation, and orientation of the neovascular networks have been described in our previous report.[1]

Results

Subretinal Neovascularization—Major new choroidal arteries and veins subserving neovascular fronds—Fronds of choroidal neovascularization that had been present for some time and had infiltrated the subretinal space for a considerable distance were serviced by well-defined feeding arteries and draining venules which closely resembled small choroidal arteries and veins (Fig. 1).

Both afferent and efferent vessels had luminal diameters between 40 and 60 μ, and frequently possessed a common adventitia composed of longitudinal collagen bundles interspersed with concentrically

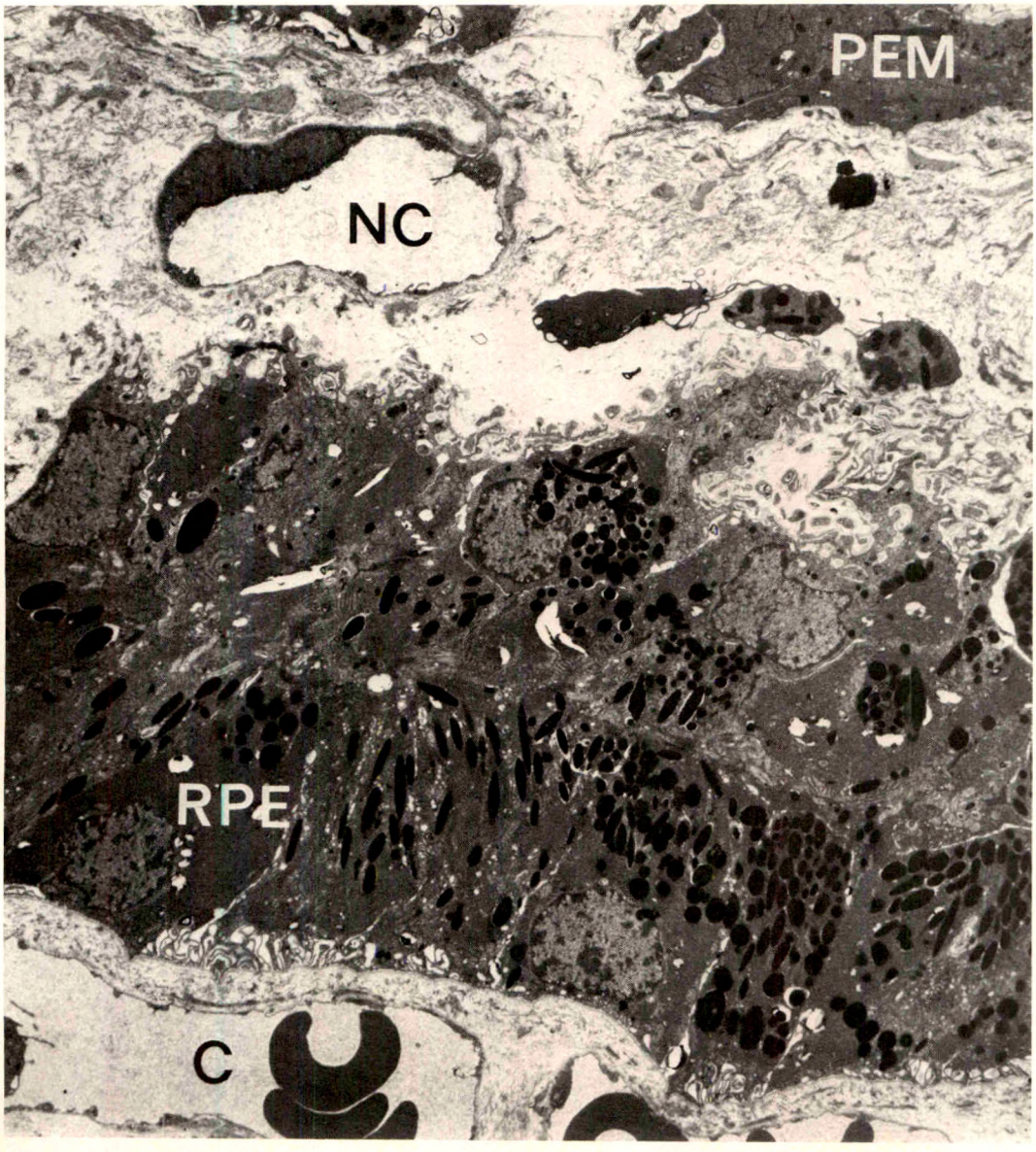


Fig. 2 (Archer and Gardiner). Outer retina and choriocapillaris (C) show invasion of subretinal space by new choroidal capillary (NC). There is reduplication of the retinal pigment epithelium (RPE) and formation of a retinal pigment epithelial membrane (PEM) adjacent to the new vessel (×3,000).

orientated fibroblasts. The peripheral adventitia merged with the residual glial tissue of the nearby atrophic retina.

The arteries were lined with a continuous layer of low endothelial cells whose nuclei characteristically protruded into the lumen. These cells were usually joined by adhering junctions, although focal regions of membrane occlusion were occasionally identified. The luminal plasma membranes showed scattered microvillous projections and a moderate degree of pinocytotic activity. At junctional complexes the area of contact between contiguous cells was increased by luminal cytoplasmic folds. Much of the endothelial cytoplasm was occupied by large bundles of intermediate filaments, 9 to 11 nm in diameter. Weibel-Palade bodies were a common component of the endothelial

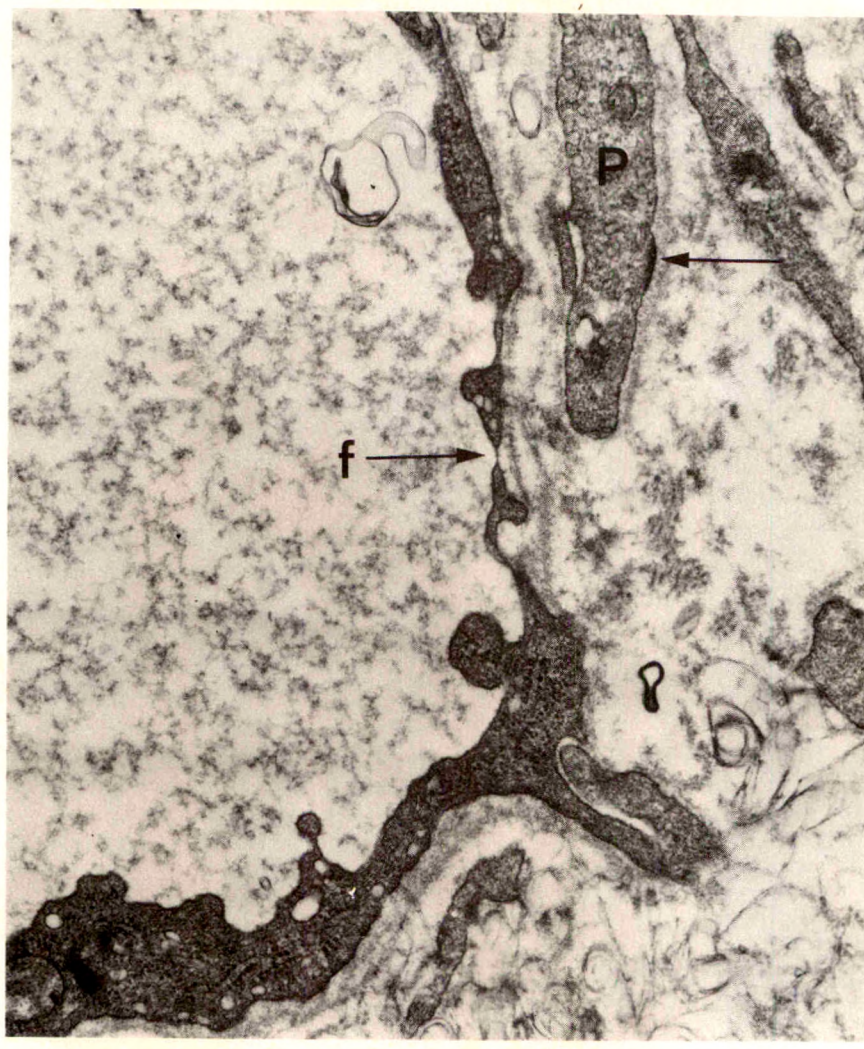


Fig. 3 (Archer and Gardiner). Endothelial fenestrae (f) in established new choroidal capillary. A pericyte process (P) demonstrates a prominent subplasmalemmal densification (upper arrow) (×40,000).

cells although their distribution was patchy. A fine basal lamina separated the endothelium from the media, which in the arteries were composed of three to four layers of well-differentiated smooth muscle cells.

Weibel-Palade bodies appeared to be more numerous in new choroidal veins than in arteries although we undertook no stereologic quantification. The endothelial cells in efferent veins were flatter, overlapped more, and showed fewer cytoplasmic filaments than those of fellow arteries. The media consisted of a discontinuous layer of pericytes around the endothelium. All major vessels examined were patent and perfused with blood.

ESTABLISHED CAPILLARIES—As the growing front of the neovascular complex was approached, the larger arteries and veins gave rise to well-constructed capillaries perfused with a plasma-like substance or red blood cells. These vessels closely resembled normal choriocapillaris and had a continuous endothelial layer, a scanty basement membrane, and attendant pericytes (Fig. 2). Many endothelial cells demonstrated characteristic fenestrations, 50 to 60 nm in diameter and closed by a diaphragm (Fig. 3). The

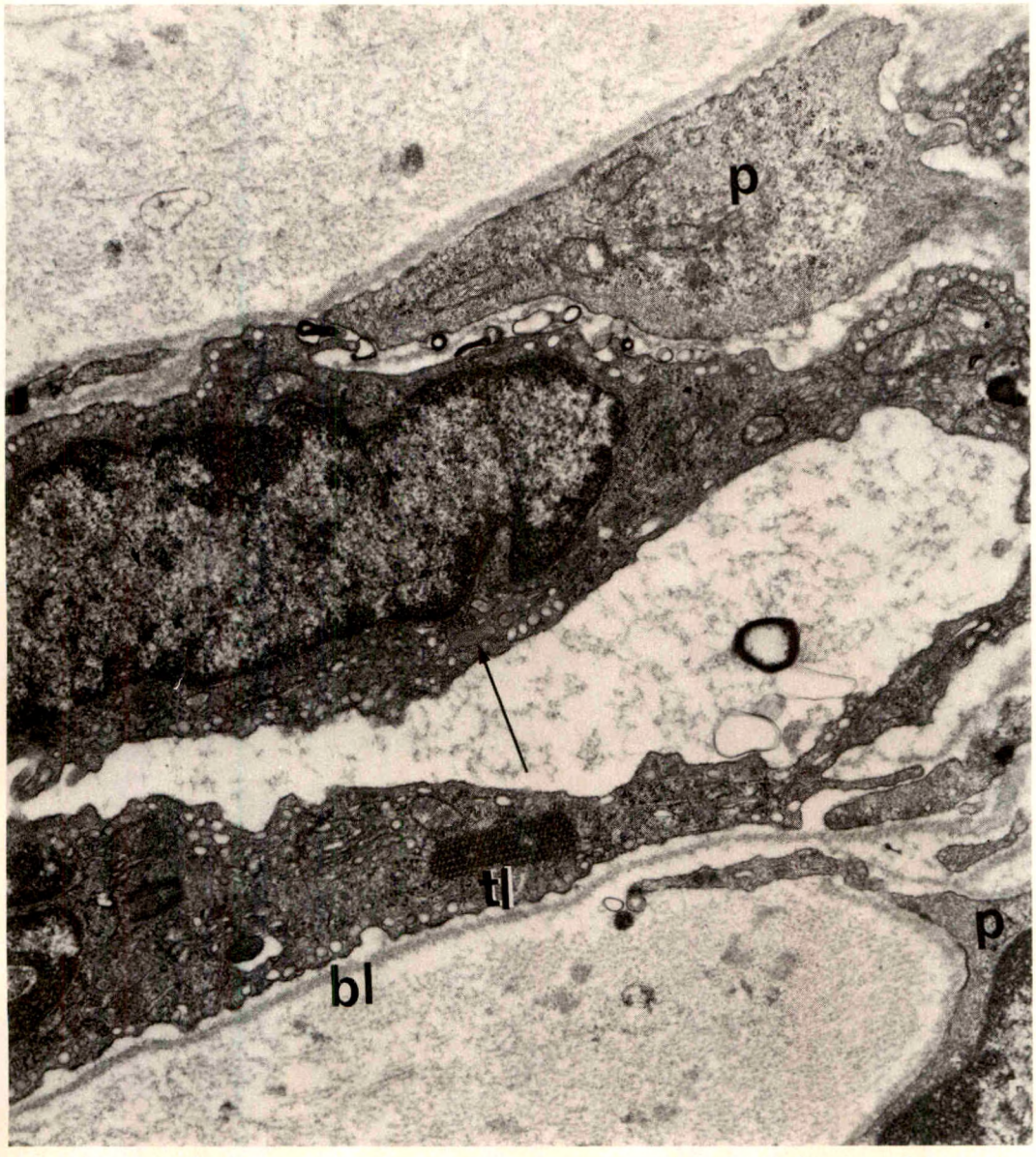


Fig. 4 (Ancher and Gardiner). Nonfenestrated segment of established new choroidal capillary shows a well-developed basal lamina (bl). Weibel-Palade bodies (arrow) and a multitubular lattice (tl) are present in the endothelium. Pericyte cell processes (p) are ensheathed by basal lamina (×20,000).

endothelial nuclei were large and the chromatin existed mainly in the form of heterochromatin. Cytoplasmic ribosomes were present as free or polyribosomes and were largely unattached to the endoplasmic reticulum, a few scattered foci of which could be seen throughout the cell. Mitochondria were small and the Golgi apparatus unremarkable. Unusual multitubular lattices were occasionally identified and Weibel-Palade bodies were present in limited numbers (Fig. 4).

The nature of endothelial cell contacts was difficult to determine because of the convoluted nature of these vessels, although occasional fortuitous profiles demonstrated adhering junctions.

Pericytes in the neighborhood of established capillaries were loosely apposed to the endothelial cells and separated from



Fig. 5 (Archer and Gardiner). Primitive new choroidal capillary shows bulky nonfenestrated endothelial cell and numerous large pericytes (p). Whorls of basal lamina surround the vessel (bl) (×8,000).

them by basement membrane. Cytoplasmic organelles were largely concentrated in the perinuclear region, the cell processes being predominantly occupied by fine cytoplasmic filaments (4 to 7 nm). Pinocytosis was infrequent in the pericytes, although fibrillar densifications of the type present in smooth muscle were typically present beneath the plasma membranes (Fig. 3). Endothelial pinocytosis was at a low level in these vessels and almost absent in regions where fenestrae occurred. Both endothelial cells and pericytes were enveloped by a fine regular basement membrane (60 to 100 nm) (Fig. 4). Many of these established capillaries were encased within a loose mesh of fine collagen-like fibrils. The origin of these fibrils was uncertain as fibroblasts were only sparsely distributed in these regions.

PRIMITIVE CHOROIDAL CAPILLARIES—



Fig. 6 (Archer and Gardiner). Higher-power view of primitive new vessel shown in Figure 5. Basal lamina (bl) ensheathes endothelial capillary and pericytes except at points of contact (arrows). Pericytes (P) show subplasmalemmal densifications (arrows) and secretory granules (sg) (×20,000).

These new capillaries were located near the advancing or peripheral margins of the neovascular fronds. They were characterized by bulky endothelial cells, and were rich in cytoplasmic organelles and closely ensheathed by many large pericytes showing a similar endowment of cellular inclusions (Figs. 5 and 6). The endothelial cell nuclei displayed prominent nucleoli, a high proportion of euchromatin, and deep invaginations of the nuclear envelope. A significant proportion of the cell cytoplasm was occupied by cisternae of the granular endoplasmic

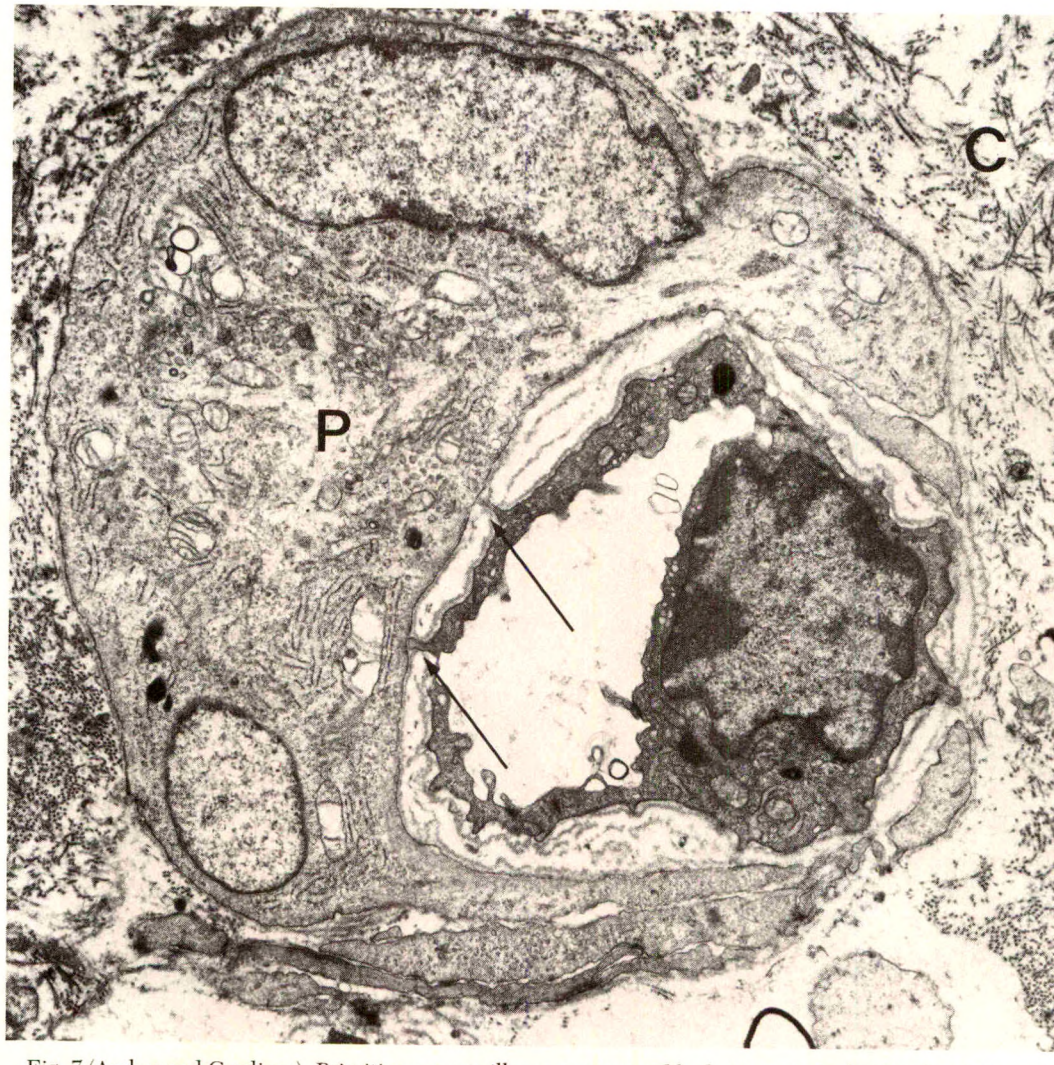


Fig. 7 (Archer and Gardiner). Primitive new capillary encompassed by large pericyte (P). Basal pseudopodia (arrows) of endothelial cell penetrate basal lamina to contact pericyte. Fine collagen-like fibrils (C) are present in the extravascular space (×12,000).

reticulum and there was an associated increase in the size and activity of the Golgi apparatus.

Multitubular lattices were numerous and appeared larger and less regular in outline than those in established capillaries. The apical plasma membrane showed an increase in the number of pinocytotic vesicles, and in some cells formed large microvillous processes. The basal cell surface often extruded small pseudopodia which became intimately apposed to the plasma membrane of a nearby or encompassing pericyte. Although close membrane contact and discontinuity of the basal lamina were obvious in such regions, any traditional type of junctional complex was impossible to identify (Figs. 7 and 8). Junctions between endothelial cells appeared to be of the zonulae adherens type, although definition was often poor. Endothelial fenestrae were



Fig. 8 (Archer and Gardiner). Higher-power view of primitive new capillary shown in Figure 7 shows zone of contact between pericyte (P) and basal pseudopodium of endothelial cell (E). The basal lamina (bl) is rounded off in this region (×80,000).

infrequent and usually solitary in these more primitive capillaries. The pericytes displayed a highly developed granular endoplasmic reticulum, many large mitochondria, and extensive arrays of fine filaments (4 to 7 nm), which were especially numerous in the more peripheral regions of the cells (Fig. 9). Such filaments were not a feature of endothelial cells. The pericytes also contained homogeneous membrane-bound granules of moderate electron density (Fig. 9). These bodies were round or oval (160 to 300 nm), had a faint microscopic halo, and were more numerous in cells at the distal region of the neovascular frond. We observed them in locations that suggested they originated in the Golgi apparatus, but,

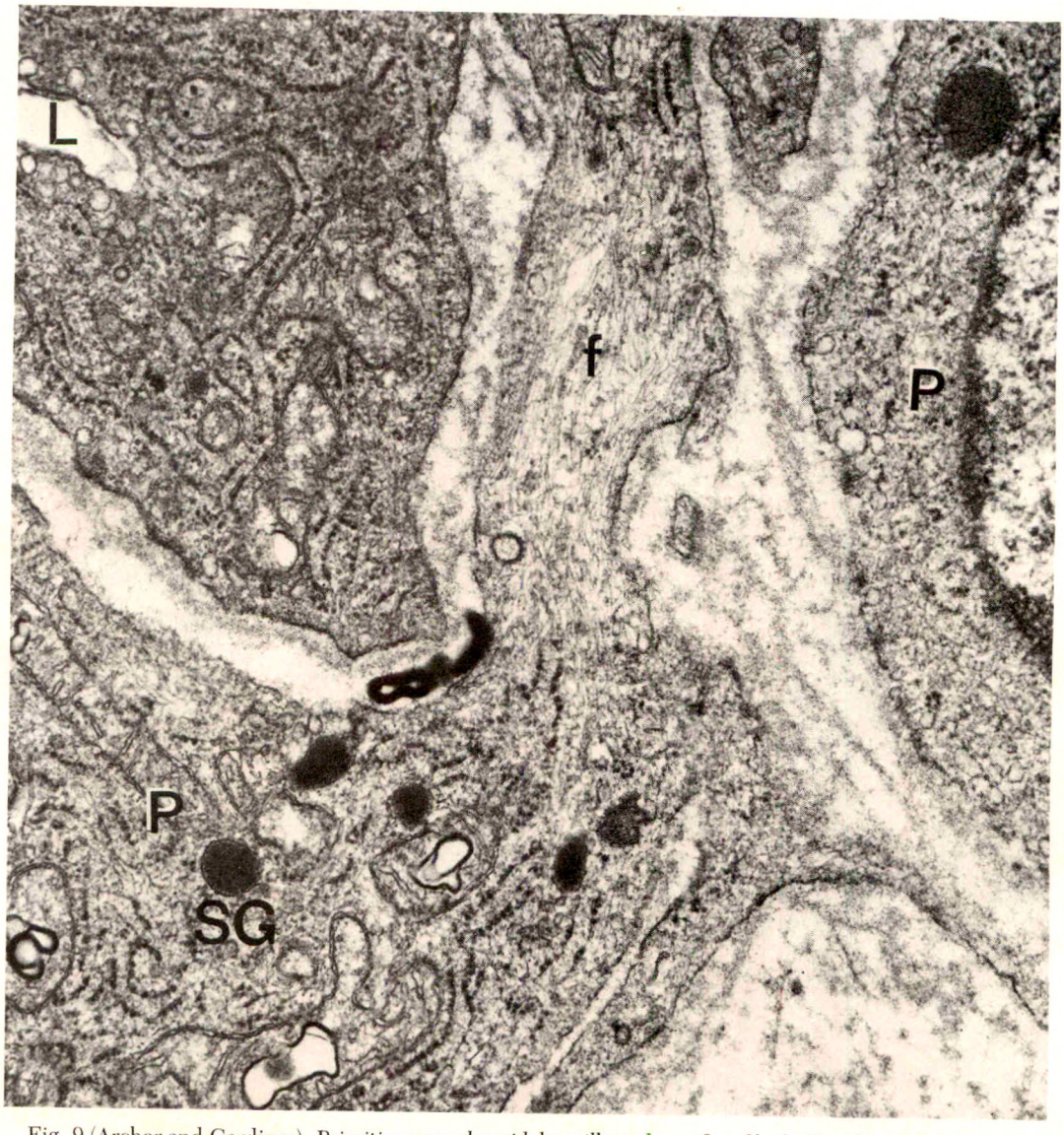


Fig. 9 (Archer and Gardiner). Primitive new choroidal capillary shows fine fibrils (f) and secretory granules (SG) in pericyte (P). L, Lumen of vessel (×40,000).

because the majority of such sites were on the outer periphery of the organelle, it was impossible to establish a definite relationship.

Occasional sections of these granules demonstrated fusion of the limiting membrane of the granule with the plasma membrane of the pericyte, confirming the extracellular destination of its product (Fig. 10).

CAPILLARY VASCULAR SPROUTS: ADVANCING CELLS—Vascular sprouts are the terminal vessels of the neovascular complex characterized by a slit-like lumen and unusual ultrastructural characteristics exhibited by both endothelial and pericyte components (Figs. 11 to 13).

The endothelial cells of these sprouts had more rounded profiles than those in vessels with established lumina. A high

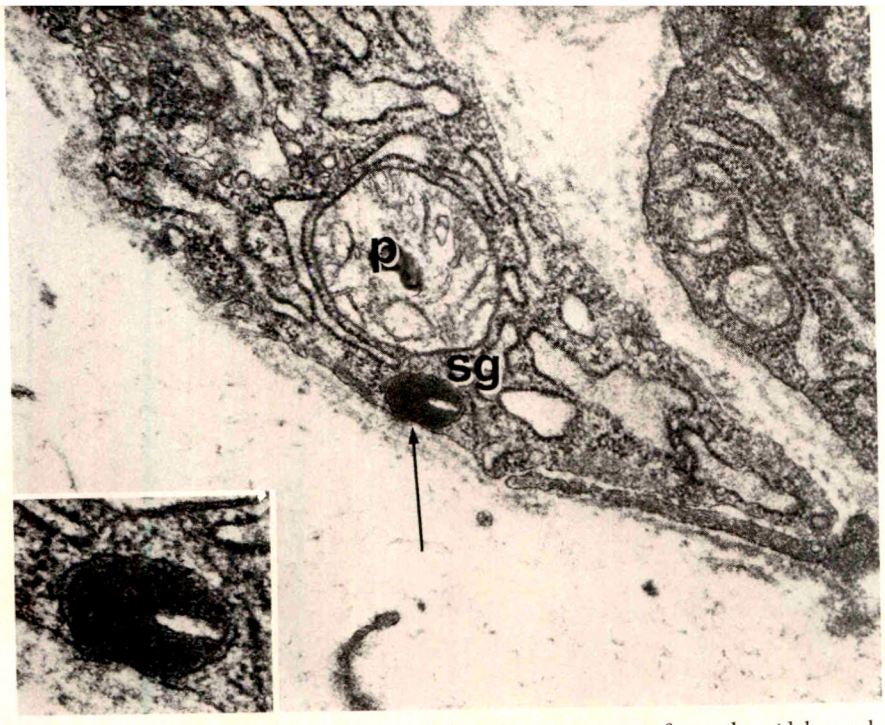


Fig. 10 (Archer and Gardiner). Pericyte (p) in advancing sprout of new choroidal vessel. A secretory granule (sg) is present and its limiting membrane has fused with the plasma membrane of the pericyte (arrows) (×35,000). Inset, Secretory granule (×70,000).

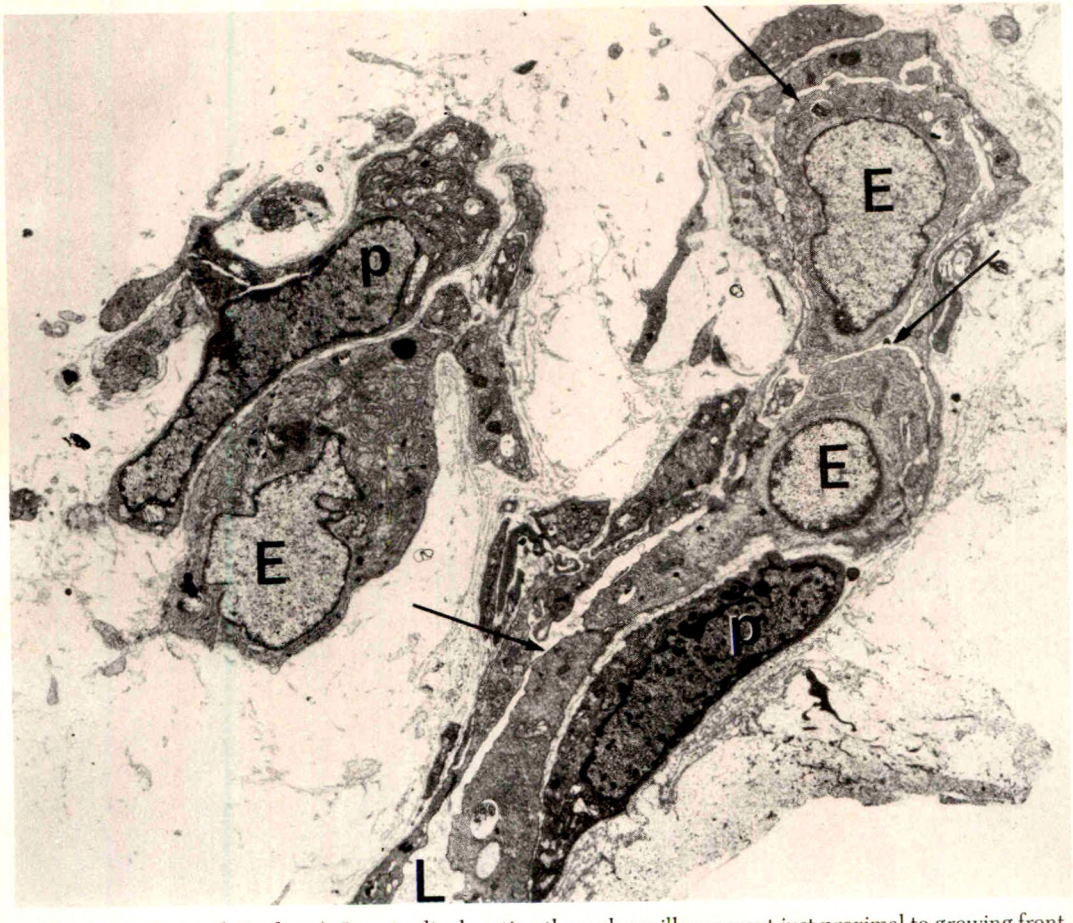


Fig. 11 (Archer and Gardiner). Longitudinal section through capillary sprout just proximal to growing front. The lumen (L) becomes slit-like as the end region of the vessel is approached. Pericyte nuclei (p) appear more flattened than those of endothelial cells (E) (×5,000).



Fig. 12 (Archer and Gardiner). Oblique section through capillary sprout shows euchromatic nuclei with nucleoli in both endothelial cell (E) and pericyte (P). A lumen (L) can be identified and an isolated endothelial cell process (ep) is present. The vascular cells are surrounded by a flocculent basal lamina (×8,000).

proportion of the endothelial cytoplasm was occupied by cisternae of the granular endoplasmic reticulum, which were distended by a fine granular proteinaceous material (Fig. 13). Multitubular lattices were very large and numerous (Fig. 14). Small Weibel-Palade bodies were also observed, although their disposition within the cells gave no indication of their eventual destination or function.

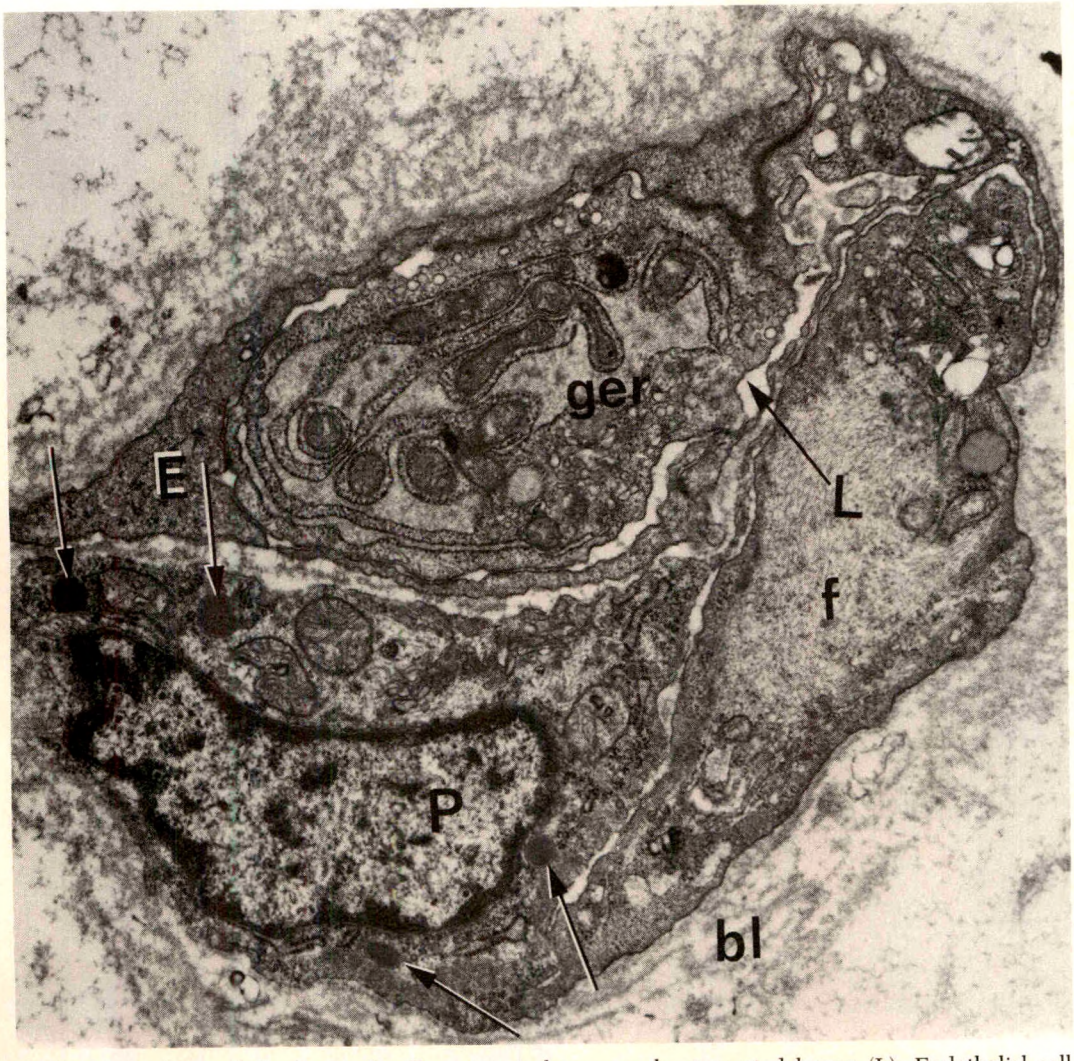


Fig. 13 (Archer and Gardiner). Capillary sprout shows grossly attenuated lumen (L). Endothelial cell cytoplasm (E) shows dilated cisternae of granular endoplasmic reticulum (ger) with many closely applied mitochondria. An adjacent pericyte (P) contains several large mitochondria, fine fibrils (F), and a number of secretory granules (arrows). A basal lamina (bl) is forming about the capillary sprout (×20,000).

Endothelial cell junctions were tenuous and ill-defined, and areas of cytoplasmic discontinuity were common. We identified occasional red blood cells in the subretinal space immediately proximal to a group of budding capillaries, suggesting that diapedesis may have occurred at these incompetent and discontinuous end regions (Fig. 15).

Serial sections disclosed that the leading cells of the growing sprouts resembled large polygonal pericytes (Fig. 16). The irregularly shaped nuclei of these cells displayed a high proportion of euchromatin and large nucleoli; however, the predominant feature was the exaggerated development of the granular endoplasmic reticulum (Figs. 15 and 16). Dilated cisternae of the granular endoplasmic reticulum enclosed a fine granular material and occupied much of the cell cytoplasm. The mitochondria were larger

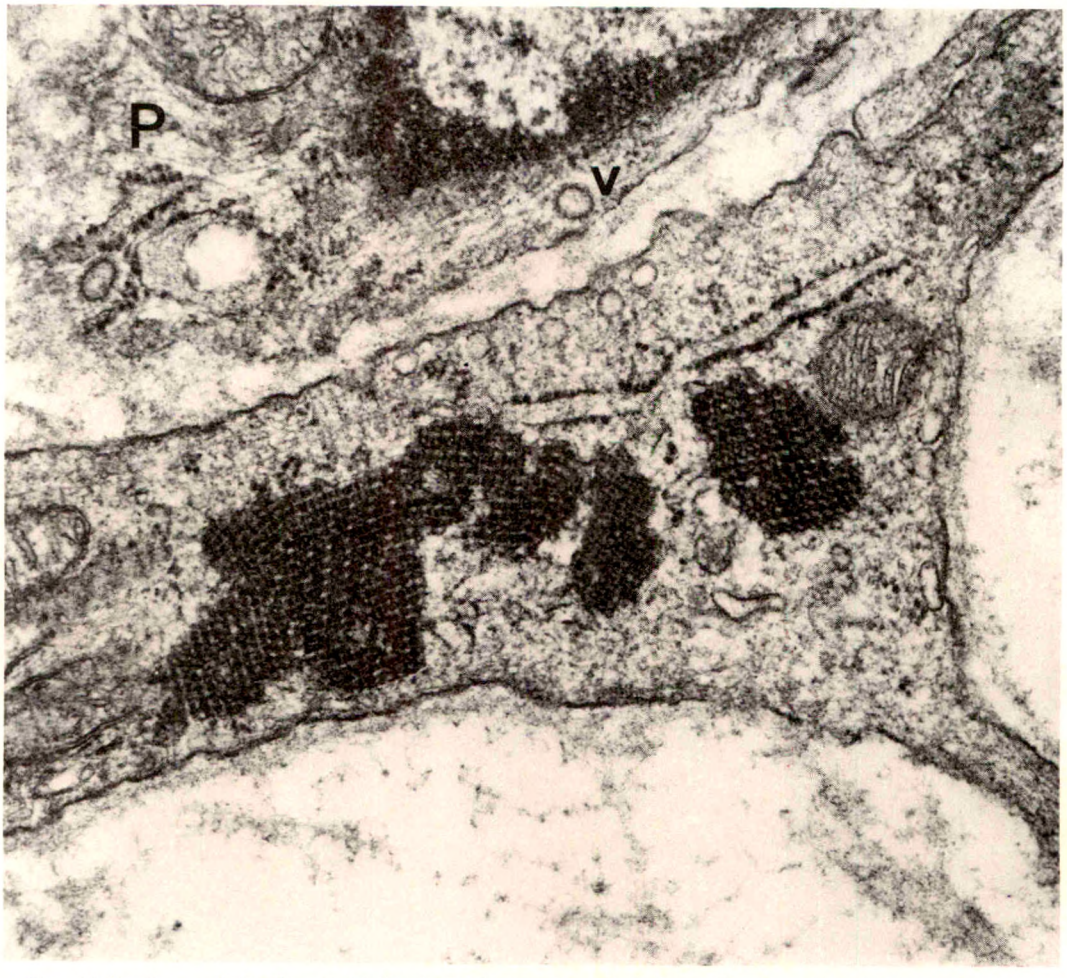


Fig. 14 (Archer and Gardiner). Multitubular lattice in endothelial cell process. Coated vesicles (V) are present at the plasma membrane of an adjacent pericyte (P) (×50,000).

and more numerous here than in any other cells of the neovascular complex. The pericyte secretory granules showed an uneven distribution but were always present (Fig. 17).

Capillary sprouts were invested with delicate basal laminal envelopes which appeared to condense from a diffuse cloud of basement-membrane-like material which enshrouded these developing vessels (Fig. 16).

Beyond the advancing frond, the gliotic retina contained macrophages, red blood cells, and degenerated outer receptors. Fibroblasts and collagen were not prominent in this region although retinal pigment epithelial cells were present, mostly as membranes or occasionally as giant cells.

Choroidovitreal vessels—VASCULAR OUTGROWTHS AT PHOTOCOAGULATION SITES—The choroidovitreal vascular outgrowths at photocoagulation sites were largely composed of irregularly intertwining vessels measuring 8 to 15 μ. Most capillaries had continuous endothelial linings; the individual cells were secured largely by adhering zonules

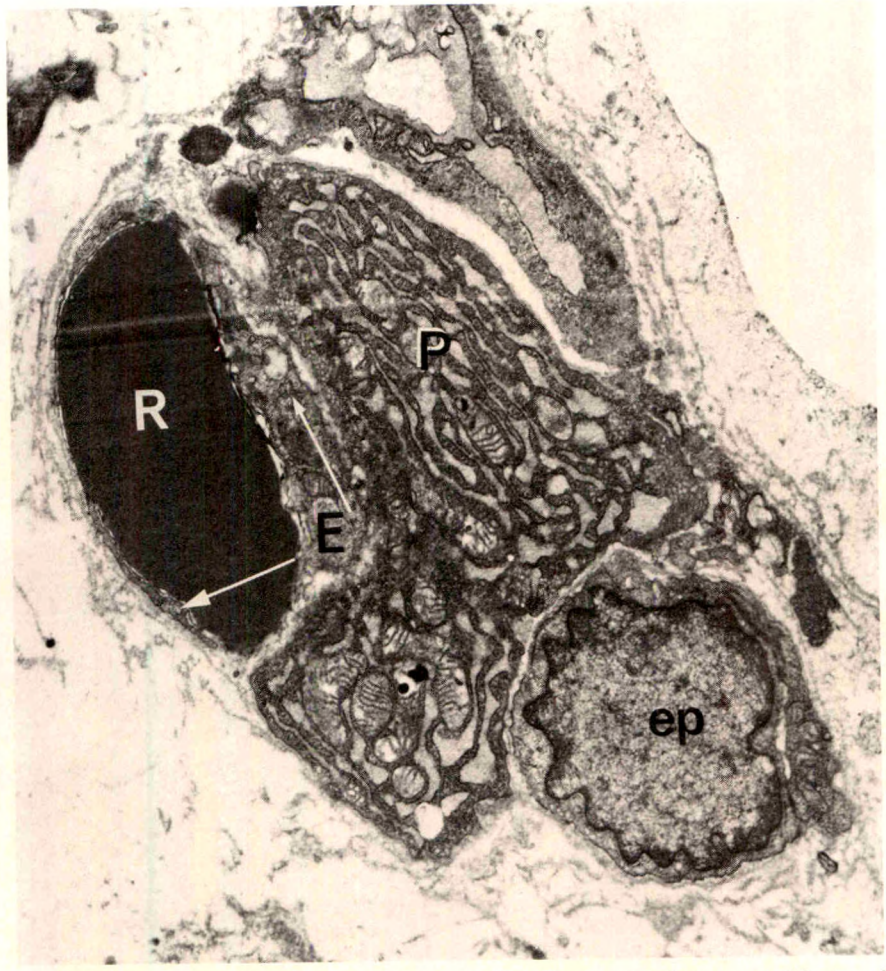


Fig. 15 (Archer and Gardiner). Distal region of capillary sprout shows a possible site of red blood cell diapedesis. A red blood cell (R) is partially surrounded by a discontinuous endothelial cell (E). The cytoplasm of an adjacent pericyte is filled by dilated cisternae of the granular endoplasmic reticulum and many mitochondria. An endothelial cell process (ep) containing a nuclear profile is also present (×10,000).

(Fig. 18). Plasma membranes demonstrated foci of intimate apposition with adjacent pericytes. The endothelial cytoplasm was unremarkable, although there was a high complement of intermediate filaments (9 to 10 nm) which ramified as large bundles throughout the cytoplasm (Fig. 18). Many Weibel-Palade bodies were present in addition to occasional multitubular lattices of the type found in subretinal neovascular fronds. In certain regions these lattices were clearly continuous with the smooth endoplasmic reticulum. Pericytes and their processes ensheathed the endothelial cells and were themselves surrounded by fibrocyte-like cells within a collagen matrix. Pigmented cells, probably of choroidal origin, occurred in large numbers at the sites of vasoproliferation.

Capillaries within the vitreous cavity showed a range of changes; some of these were interpreted as degenerative or regressive. Large areas of endothelial cyto-

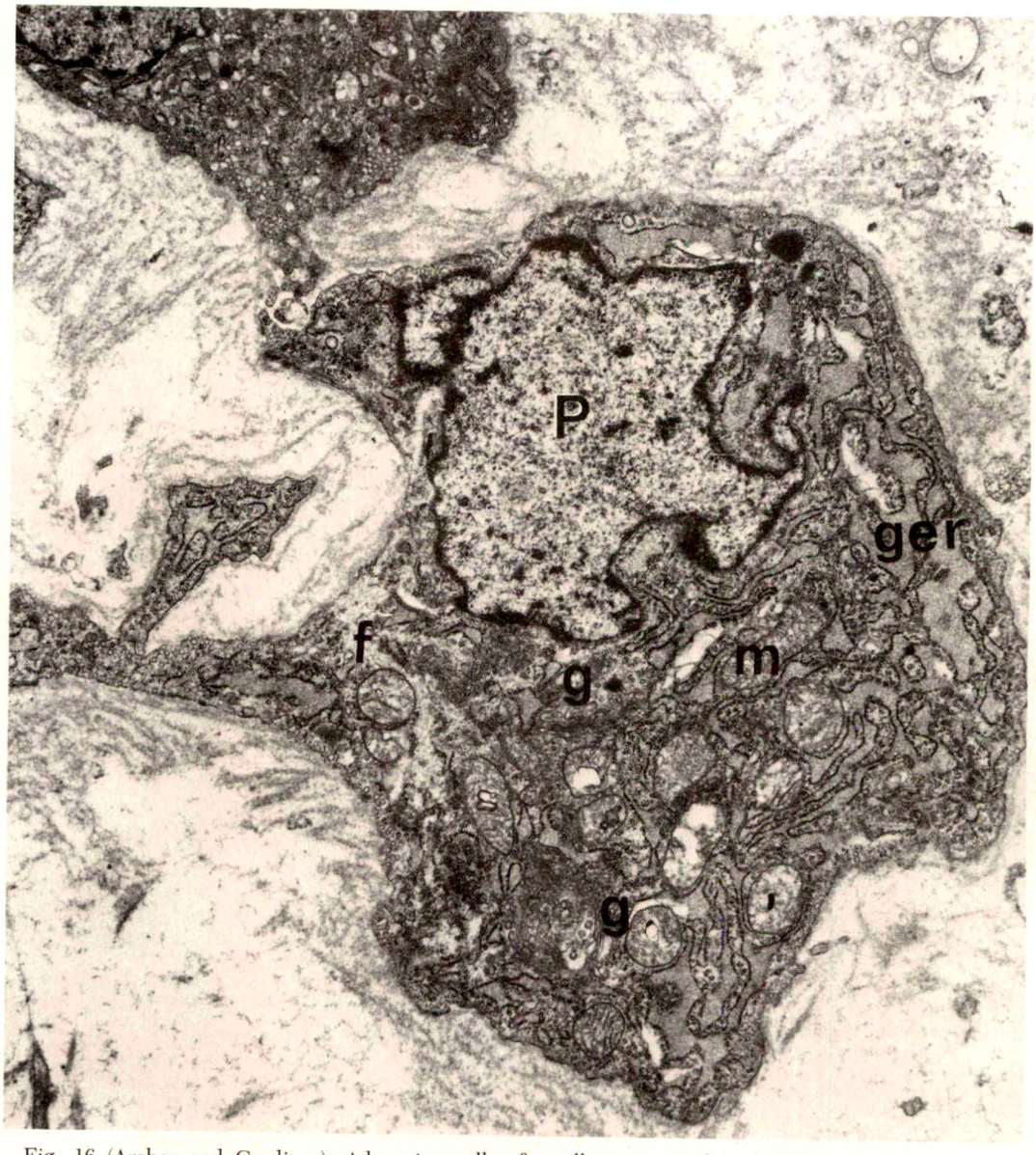


Fig. 16 (Archer and Gardiner). Advancing cells of capillary sprout show a large active pericyte (P). Numerous large mitochondria (m) and dilated cisternae of granular endoplasmic reticulum (ger) are present. Several dictyosomes of the Golgi apparatus (g) and a large bundle of fine fibrils (f) are also present. These cells are ensheathed by multiple layers of flocculent basal lamina material (×12,000).

plasm appeared to have undergone autophagic degeneration, whereas adjacent regions within the same cells remained healthy. These cells showed unusually high concentrations of Weibel-Palade bodies, many intact profiles of which could be discerned within the autophagic regions (Fig. 19).

Vessels having the structural features of small and precapillary arterioles were also identified near photocoagulation sites. The arterial endothelium demon-

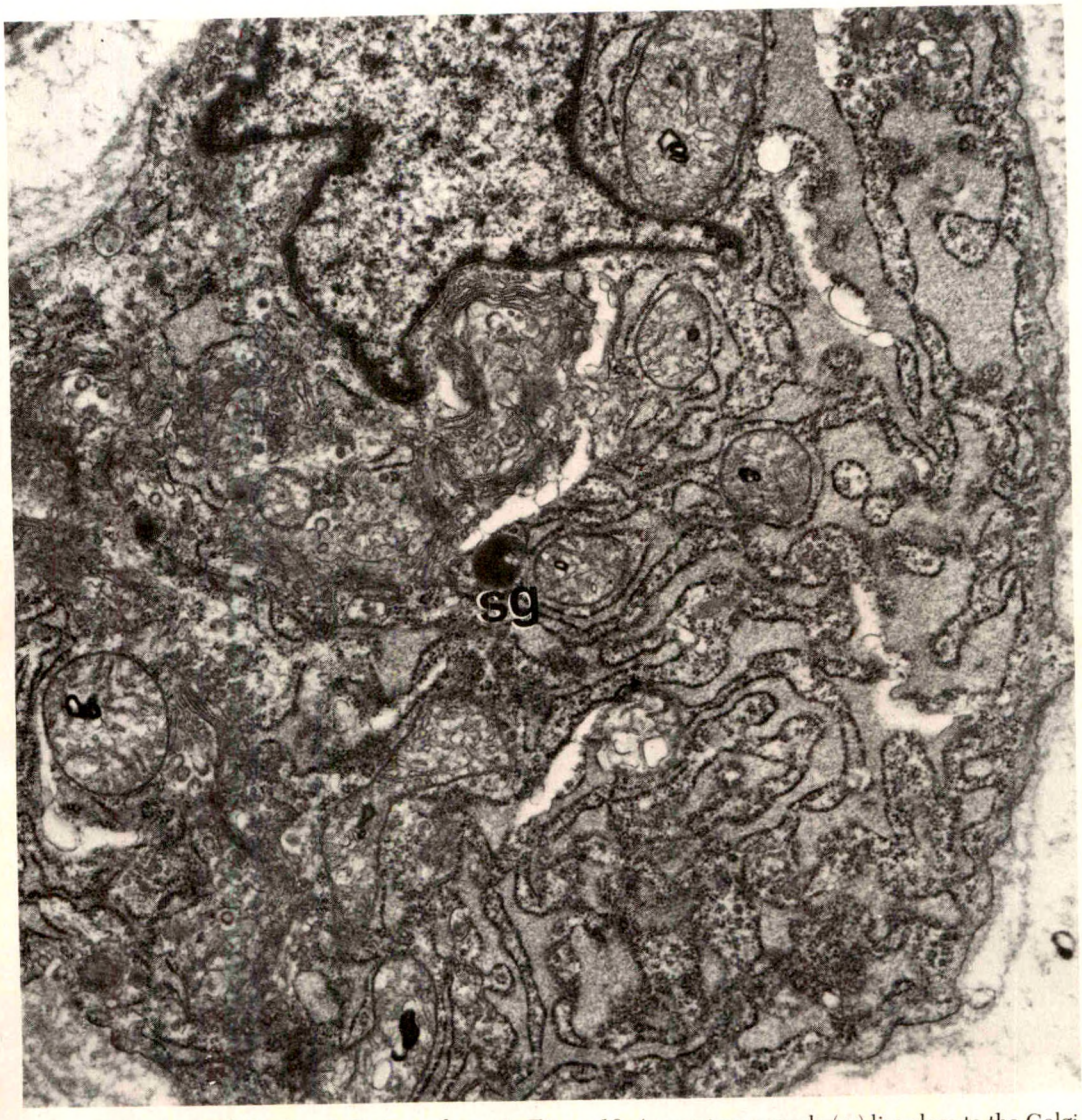


Fig. 17 (Archer and Gardiner). Pericyte shown in Figure 16. A secretory granule (sg) lies close to the Golgi zone.

strated essentially the same features as the capillaries, although they had even more prominent endothelial filaments. Only a single coat of smooth muscle cells was present, although many of these cells overlapped. Junctional zones between smooth muscle and endothelial cells were common.

TRUNK VITREOUS VESSELS—Large-caliber vessels communicating between vasoproliferative tissue at adjacent photocoagulation sites were identified in the vitreous cavity.[1] These vessels had large luminal diameters (25 to 30 μ) and were lined by many bulky endothelial cells that appeared to be orientated along the long axis of the vessel (Fig. 20).

The endothelial cells had large concentrations of intermediate filaments and many Weibel-Palade bodies, and were

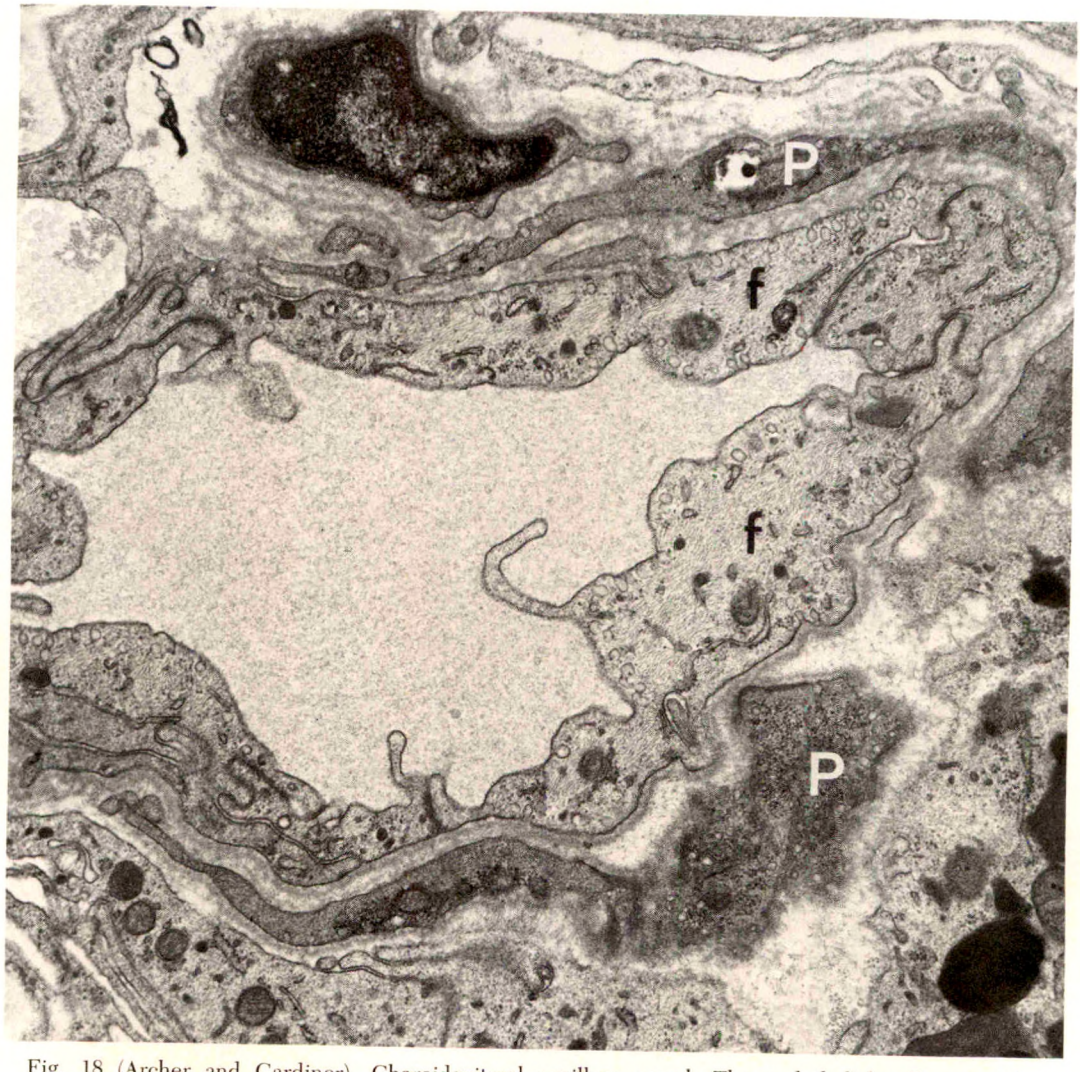


Fig. 18 (Archer and Gardiner). Choroidovitreal capillary vessel. The endothelial cells are rich in intermediate filaments (f) and are surrounded by several pericyte processes (P) (×20,000).

joined by adhering junctions. The nuclei of the endothelial cells were deeply invaginated with nuclear chromatin largely in the heterochromatic form. The endothelial layer was surrounded by one or two layers of smooth muscle cells that contained substantial accumulations of glycogen (Fig. 20). There were many points of contact between smooth muscle cells and the endothelium, although we could not define the exact nature of these junctions. The intravitreal vessels had ill-defined adventitia composed of fibroblasts, collagen bundles, and condensed vitreous fibrils. Cells containing pigment were occasionally incarcerated or entrapped within this loosely arranged adventitial tissue (Fig. 21). There was no evidence of intraluminar fibrin deposition or clot formation.

Discussion

The choroidal origin of these new vessels was confirmed by light microscopy

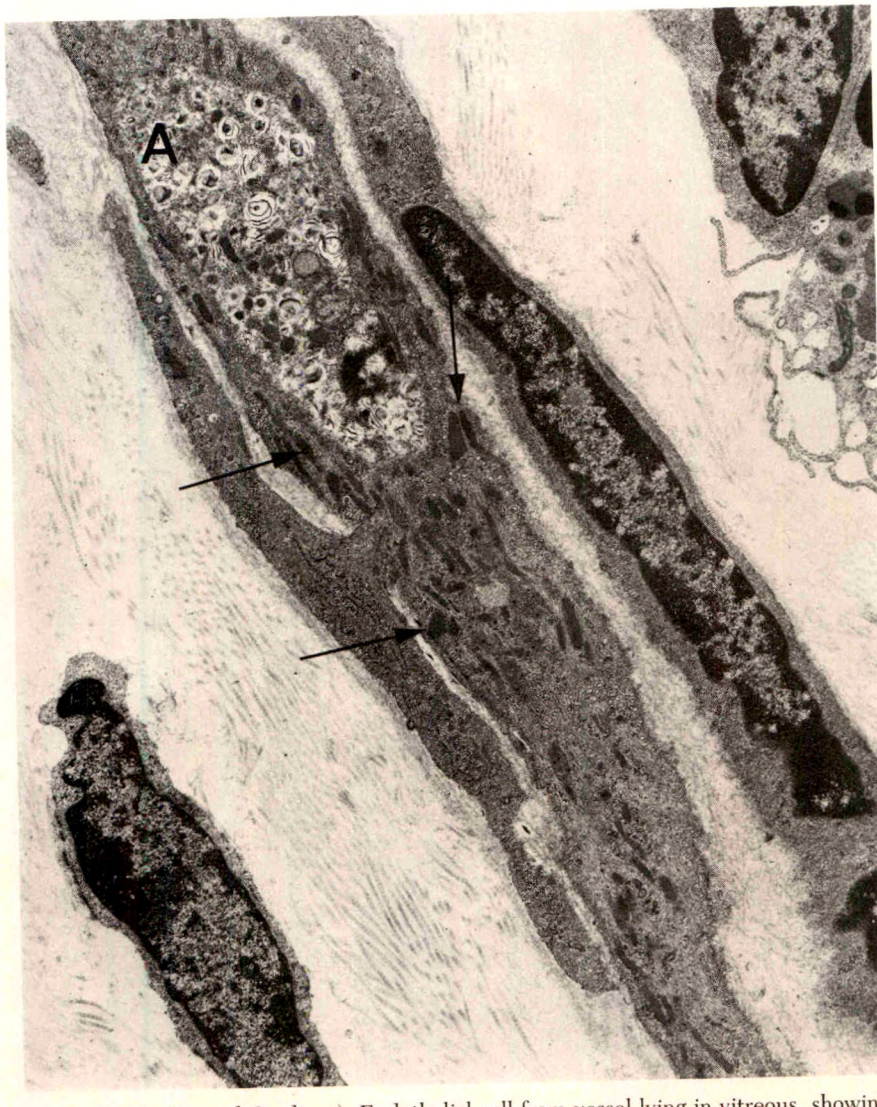


Fig. 19 (Archer and Gardiner). Endothelial cell from vessel lying in vitreous, showing numerous Weibel-Palade bodies (arrows) and a large autophagic region (A) (×10,000).

and fluorescein angiography,[1] although it was not possible to identify the precise points of origin of the new vessels from the existing choroidal vasculature as severe scarring at photocoagulation sites obscured their early development. The new vessels entered the subretinal space and, in some instances, the vitreous cavity via Bruch's membrane and the retinal pigment epithelium, through defects created by intense photocoagulation burns. We did not identify the factors that stimulated choroidal vasoproliferation, although the creation of intraretinal spaces and pathways by cellular necrosis and retinal detachment after retinal vascular occlusion favored the development of neovascular fronds and determined to some extent their morphologic characteristics and architecture.[1]

The new choroidal vessels in this experiment, whether ramifying in the sub-

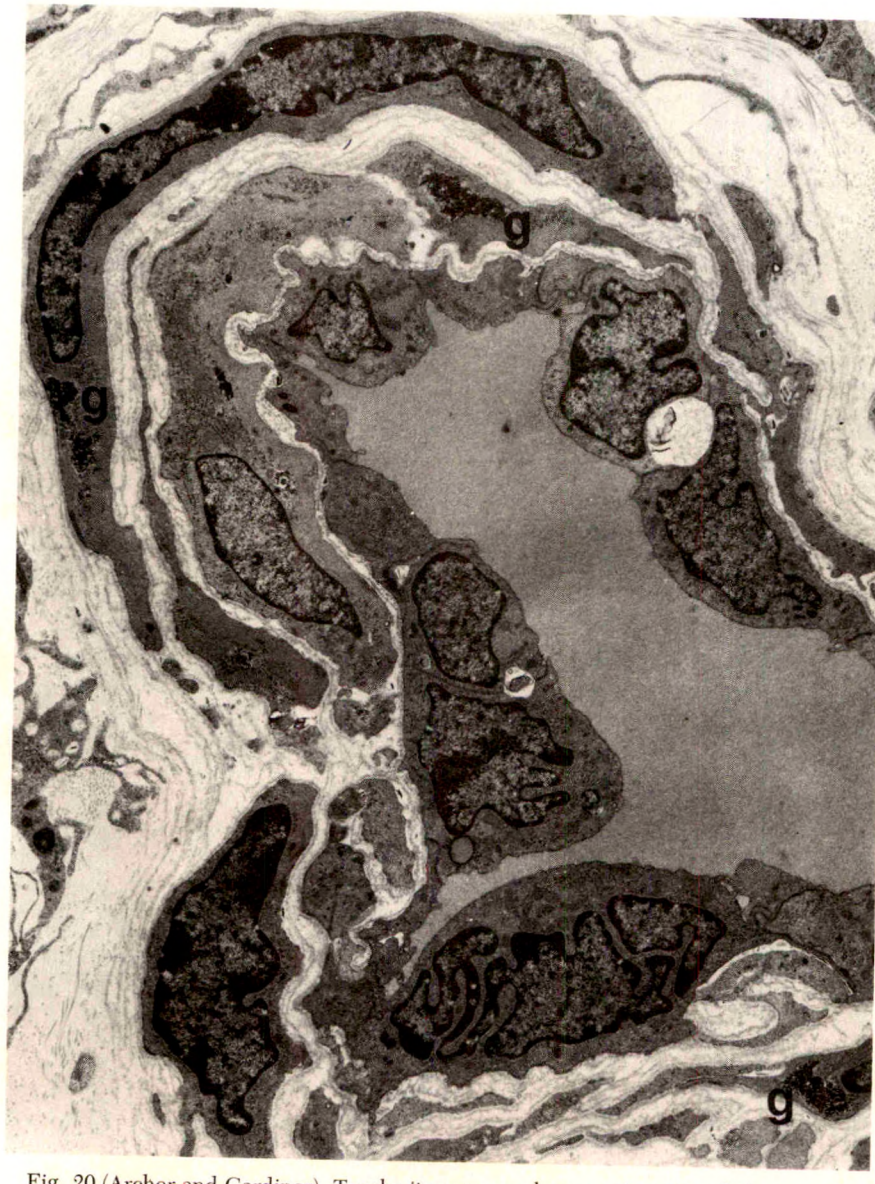


Fig. 20 (Archer and Gardiner). Trunk vitreous vessel connecting sites of photocoagulation. Many endothelial cell profiles are surrounded by a double layer of smooth muscle cells which contain large reserves of glycogen (g) (×8,000).

retinal space or vitreous cavity, had the same basic construction and similar angiographic properties. The choroidovitreal vessels, however, were more difficult to orientate and examine in sequential section and most of the data concerning choroidal vasoproliferation were obtained from serial analysis of many subretinal networks at varying stages of development.

The examination of capillary sprouts at the leading edges of neovascular fronds demonstrated that endothelial cells and pericytes were the key vascular components in choroidal angiogenesis. The endothelial cells at the terminal apices of

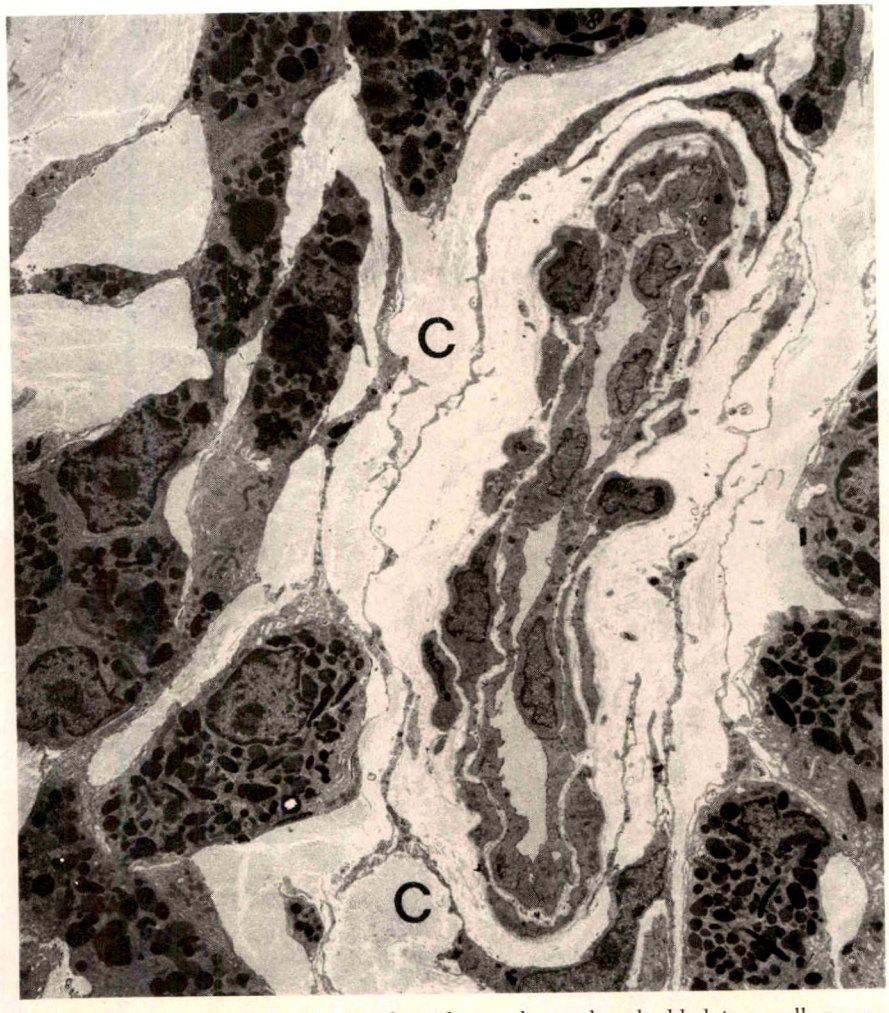


Fig. 21 (Archer and Gardiner). Choroidovitreal vessel embedded in a collagenous matrix (C) and surrounded by pigment cells. The vessel lumen is collapsed but not thrombosed (×2,500).

vascular sprouts were large and bulky and displayed unusual cytoplasmic profiles dominated by expanded cisternae of the rough endoplasmic reticulum. Such cells, although atypical, were always discernible as vascular endothelia by their relationship to the vascular lumen (often a rudimentary slit), the presence of a basal lamina, and the frequent occurrence of Weibel-Palade bodies, that is, organelles specific for vascular endothelia, in their cytoplasm[2] (Table). All endothelial cells in developing capillaries had definable junctions with adjoining endothelial cells and even the leading endothelial cells of vascular sprouts were not found in isolation.

The origin of these new endothelial cells or the sites of mitotic activity were not identified in this study; however, experiments in wound healing[3-5] and corneal vascularization[6] suggest that they probably derived from the surviving endothelial cells of residual choroidal vessels at the sites of photocoagulation and that they proliferated by a process of

TABLE

CRITERIA USED TO DEFINE ENDOTHELIAL AND PERICYTE COMPONENTS OF VASCULAR SPROUTS

Endothelial Cell	Pericyte
Basal lamina present	Basal lamina present
Junctional complexes with adjacent endothelial cells	No pericyte-to-pericyte contact but focal membranous contacts with endothelial cells
Weibel-Palade bodies common	No Weibel-Palade bodies
No subplasmalemmal dense bodies	Subplasmalemmal dense bodies, similar to those of smooth muscle cells, common
Multitubular lattices common	Tubular lattices not observed
Secretory granules not a feature	Secretory granules common
Association with vascular lumen	No direct association with vascular lumen

mitosis. The newly formed endothelial cells subsequently migrated to and budded in the subretinal space to form neovascular sprouts. The terminal portions of these sprouts were composed of solid or nearly solid cords of cells. The only cellular elements in immediate proximity to these vascular sprouts were degenerate rod and cone outer segments, retinal pigment epithelial cells, some of which formed membranes,[7] and a few red blood cells, probably the product of terminal erythrocyte diapedesis. The absence of any marked inflammatory infiltrate or significant fibrosis at the neovascular fronts made the identification and analysis of the vascular cells much easier. This study suggested that these endothelial cells did not develop from primitive mesenchymal cells, pericytes,[5] or macrophages, but rather that they developed exclusively from preexisting vascular endothelium. The strikingly developed rough endoplasmic reticulum and elaborate Golgi apparatuses of terminally located endothelial cells reflected their response to the synthetic requirements of rapidly growing vessels.

Pericytes were present in striking numbers at and beyond the advancing tip of neovascular sprouts, loosely encompassing primitive cords of endothelial cells and enmeshed in a basement membrane-like substance. We designated these perivascular and paravascular cells as pericytes on the basis of their juxtaendothelial position, the presence of basal lamina and subplasmalemmal densifications, and certain typical cytoplasmic features (Table). We did not determine the source of the pericytes involved in choroidal angiogenesis; however, we speculate that they were derived from the pericytes of the injured and exposed normal choroidal vasculature at photocoagulation sites, and that they migrated to accompany or precede the primitive neovascular tubes in intimate relationship with the endothelial cells. Cliff[8] has confirmed that mitotic division of pericytes occurs during wound healing, and Buzney and Frank[9] have shown that pericytes proliferate in abundance from explants of human retinal capillaries in culture. McCracken, Burger, and Klintworth[6] also demonstrated pericyte mitosis and proliferation in corneal neovascularization.

Active pericytes at and beyond the advancing capillary sprouts had markedly developed rough endoplasmic reticulum and elaborate Golgi apparatuses. Discrete membrane-bound granules were typically present in pericytes at this stage of angiogenesis and were considered to be secretory in nature, as their regular size and homogeneous character made any association with autophagy or hetero-

phagy unlikely. The fuzzy or diffuse halo present at the internal aspect of the limiting membrane of these secretory bodies was probably a glycocalyx, anticipating their future incorporation into the plasma membrane. Occasionally, fortuitous sections clearly demonstrated fusion of the limiting membrane of these secretory granules with the basement membrane of the pericyte, confirming an extracellular destination for their product. These bodies probably represent the precursors of the basement membrane that encapsulates these primitive vessels; such secretory bodies are not such a constant feature of pericytes in established capillaries where there is a well-structured basal lamina. That the pericyte is responsible for the production of its own basement membrane has been recognized for some time[10,11]; indeed, Rhodin[12] thought it might contribute to the formation of the endothelial cell basement membrane. The copious production of basal lamina by active pericytes at and beyond the advancing vascular sprouts may be attributable to the fluid environment of the subretinal space in which most of the neovascular fronds proliferate. The diffusion of secreted basal lamina precursor into this fluid medium possibly stimulates further basement membrane production to achieve the necessary perivascular concentrations for precipitation. Precipitation of basal lamina in a nonspecific manner around the capillary bud would then inhibit or limit further diffusion and allow a more substantial basal lamina to form in the immediate vicinity of the vascular cells. The influence of the basal lamina on cell growth and differentiation is now well recognized.[13-16] The evidence suggesting that certain cells require contact with a suitable substratum before mitosis can occur[17] may explain why endothelial mitoses in growing vessels are found downstream from the growing tip where an established basal lamina is present.[4,5,18] Perhaps an active front of preparatory cells is pushed along by a growing endothelium, which in turn slides along the tube of basal lamina extruded and left by the preparatory cells. To what extent the apical pericytes contribute to this vascular scaffolding is not known.

A phagocytic function or capability has also been attributed to pericytes, but we were unable to find any evidence of gross phagocytosis even in locations where dense concentrations of cellular debris occurred. Occasional secondary lyzosomes present in pericytes were generally considered to be autophagic as they frequently contained mitochondrial remnants. Whether pericytes can phagocytose or process exogenous protein[19] was not clarified by this study.

Most of the immature vascular cells in this study had a highly developed granular endoplasmic reticulum. This generally indicates the production of an extrinsic protein product, an activity usually reserved for differentiated cells. Our analysis of many growing fronds failed to disclose any primitive mesenchymal cells that might have represented the forerunners of pericytes or endothelial cells or both. We assumed that such cells, if present, would possess the ultrastructural features of stem cells elsewhere, in particular an undifferentiated cytoplasm showing an abundance of free ribosomes and little or no granular endoplasmic reticulum. Some investigators have demonstrated perivascular primitive mesenchymal cells in wound healing experiments, characterized by undifferentiated cytoplasm, a poorly developed granular endoplasmic reticulum, and an absent basement membrane.[3] Although no such cells were identified in this study, they may have been present during some transitory phase of vascular growth which was not identified or analyzed. Conceivably, rapidly proliferating pericytes have

an undifferentiated cytoplasm and absence of a basement membrane may indicate a stage of active migration.

On the basis of our findings, we suggest that new choroidal vessels are formed by proliferating endothelial cells originating in injured and exposed choroidal vessels at photocoagulation sites. The primitive endothelial cells migrate along low-resistance pathways in the subretinal space or through degenerate and gliotic retina toward the vitreous cavity. The advancing tip of the primitive vessel is composed of closely apposed endothelial cells which have adhering junctions but only rare fenestrations. A vascular lumen forms within one or two cells of the leading edge and is perfused with a plasma-like fluid. Occasional red blood cells extravasate through incompletely joined endothelial cells at the vascular tip and gain the extravascular space. Such terminal red blood cell diapedesis is commonly encountered in rapidly evolving neovascular complexes and is associated with immature endothelial cell junctions.[20] Although the new capillaries are perfused with blood or a plasma-like fluid, no fibrin deposition or intravascular clotting occurs, suggesting that the newly formed endothelium possesses some as yet undetermined cellular mechanism to counter fibrinogenesis and clotting.

Immature pericytes, which probably derive from normal pericytes in the damaged choroidal vasculature, are present in significant numbers at the advancing tip of the primitive endothelial tube. These active cells produce clouds of basement membrane which surround the endothelial cells and possibly prepare a rudimentary scaffolding along which endothelial cells can advance. At the leading edge of the vascular sprouts, the pericytes are separated from the endothelial cells by basement membrane and cell-to-cell contacts are not a feature; however, as the capillaries mature, the pericytes become incorporated into the perivascular basement membrane and achieve focal contact with endothelial cells. Such contacts pose the interesting possibility that the cells may communicate with each other via gap junctions at such points of apposition. These junctions play a major role in cell-to-cell communication in the smooth muscle coats of larger blood vessels and are known to connect endothelial cells and pericytes in retinal capillaries.[10] Such junctions, if present, would provide a convenient pathway for alterations in electrical potentials and exchange of low-molecular-weight solutes and ions. Direct contact of this type would also enhance the instruction of one cell by the other and possibly serve to regulate cell proliferation downstream to the growing front.[3]

Crocker, Murad, and Geer[3] suggested that such contacts between pericyte and endothelium activate a sequence of intracellular events leading to inhibition of macromolecular synthesis, thereby stopping endothelial proliferation, that is, "contact-inhibition."

The well-established capillaries proximal to the growing fronts of the neovascular fronds have a close resemblance to normal choriocapillaris. The endothelial cells become attentuated and fenestrated and show only a few scattered cytoplasmic organelles. As in normal choriocapillaris, pericytes were infrequent, quiescent, and exhibited only a low complement of intracellular inclusions. It was in these vessels that endothelial multitubular lattices first became prominent. These unusual endothelial bodies are an occasional constituent of normal choriocapillaris and have been found in mature retinal vessels of the rhesus monkey.[21] Peripheral sections of these tubular systems indicate that they are intimately associated with the smooth endoplasmic reticulum and may repre-

sent an extension of the reticulum as a storage facility for specific endothelial products. Extensive neovascular fronds often developed major feeding vessels that had the appearance of small choroidal arteries and venules, the arteries being distinguished by a well-developed muscularis. It is likely that the smooth muscle cells composing the muscularis developed from pericytes.[12] Large bundles of intermediate-sized filaments were particularly prominent in the endothelium of the arteries and probably corresponded to those filaments believed to contain the protein vimentin.[22]

Some established neovascular networks showed ultrastructural evidence of regression and atrophy. The cells of these vessels showed a variety of degenerative changes, particularly the intracytoplasmic accumulation of autophagic debris. Why proliferation should cease in some neovascular fronds and continue in others is not clear; however, vascular regression was particularly noticeable in choroidovitreal fronds and it may be that the vitreous body exerted some inhibitory effect on vasoproliferation. Brem and associates[23] have shown that vitreous extracts inhibit corneal vasoproliferation, whereas Chen and Chen[24] maintained that normal vitreous had angiogenic activity. Further investigation is required to clarify the nature and origin of angiogenic factors and the mechanism of choroidal neovascularization.

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A HISTOPATHOLOGIC STUDY OF THE POSITION OF THE SHEARING INTRAOCULAR LENS IN THE POSTERIOR CHAMBER

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Three human eyes that had successfully undergone Shearing posterior-chamber intraocular lens implants were examined post mortem. Supporting loops of these lenses were located deep within the ciliary body adjacent to a major ciliary artery (the superior loop in two cases), within the ciliary sulcus (the superior loop in one case), within the lens capsule bag (the inferior loop in two cases), and embedded within the iris stroma (the inferior loop in one case).

The position of the two supporting loops of the Shearing posterior-chamber intraocular lens has been studied experimentally in eyes from dogs,[1,2] from human cadavers,[3] and from monkeys.[4] Some surgeons try to place the loops in the bag of residual lens capsule and peripheral cortex; some prefer to place it in the region of the ciliary body sulcus.[5] The location of Shearing intraocular lenses already implanted in human eyes has not yet been documented by histologic techniques.

MATERIAL AND METHODS

Three human eyes which had had Shearing intraocular lenses successfully implanted in the posterior chamber nine months, 19 months, and 28 months before the patients died were obtained post mortem. They were fixed in formalin and examined by means of standard histologic sections and by serial sections through the critical areas of the supporting lens loops. Two of these three intraocular lenses had been implanted by the surgeon who developed the lens.

RESULTS

The superior loop of the lens in Case 1 extended through the pigment epithelium of the pars plicata of the ciliary body in the ciliary sulcus and was embedded in the ciliary body close to a major artery

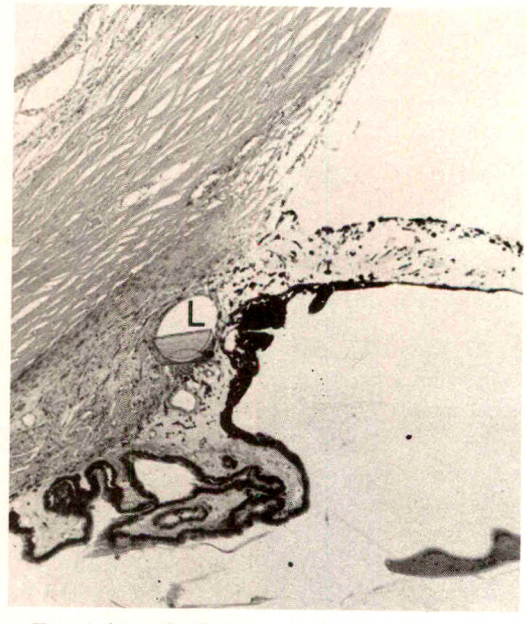


Fig. 1 (Crawford). Case 1. Superior loop (L) of Shearing intraocular lens embedded in ciliary body (hematoxylin and eosin, × 10).

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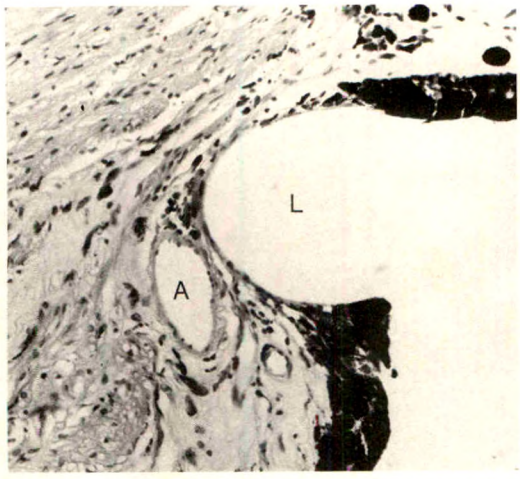


Fig. 2 (Crawford). Case 1. Superior loop (L) of Shearing intraocular lens extending through pigment epithelium of ciliary body and lying adjacent to artery (A) (hematoxylin and eosin, ×64).

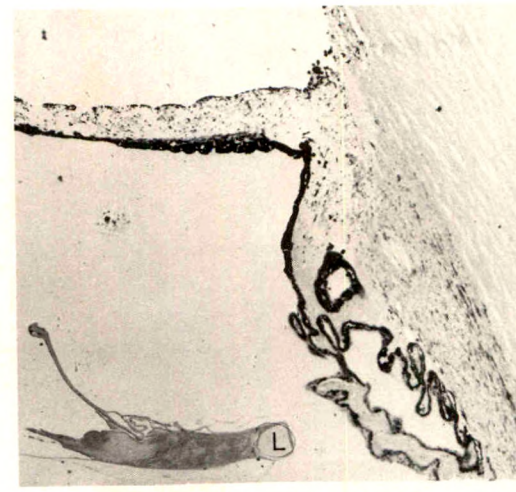


Fig. 3 (Crawford). Case 1. Inferior loop (L) of Shearing intraocular lens in residual lens capsule bag (hematoxylin and eosin, ×10).

(Figs. 1 and 2). The inferior loop of the lens was firmly fixed between the anterior and posterior lens capsule in the residual capsular sac (Fig. 3). In Case 2, the superior loop was located in the ciliary body sulcus and firmly held by adhesions between the posterior surface of the iris and the anterior face of the ciliary body (Fig. 4). The inferior loop was located within the peripheral iris (Fig. 5). As in Case 1, the superior loop of the lens in Case 3 was embedded deep

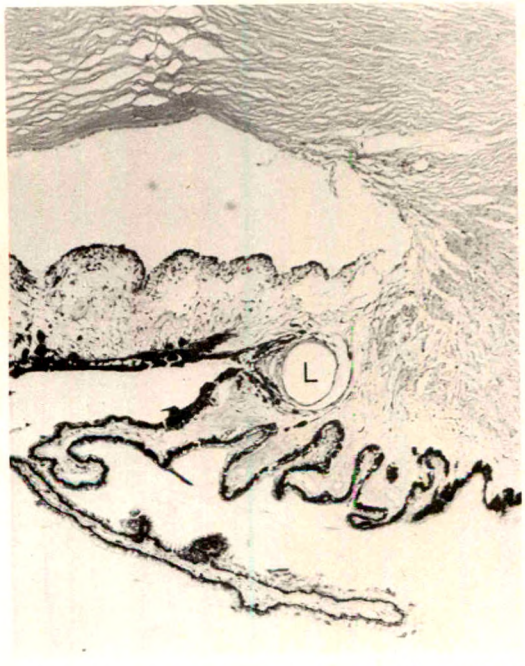


Fig. 4 (Crawford). Case 2. Superior loop (L) of Shearing intraocular lens in ciliary sulcus (hematoxylin and eosin, ×17).

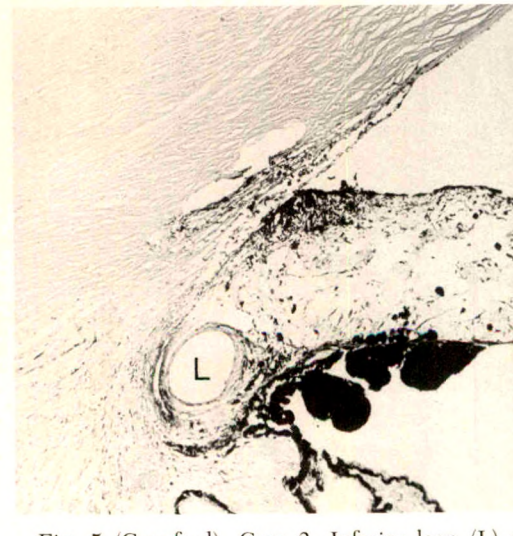


Fig. 5 (Crawford). Case 2. Inferior loop (L) of Shearing intraocular lens in peripheral iris (hematoxylin and eosin, ×17).

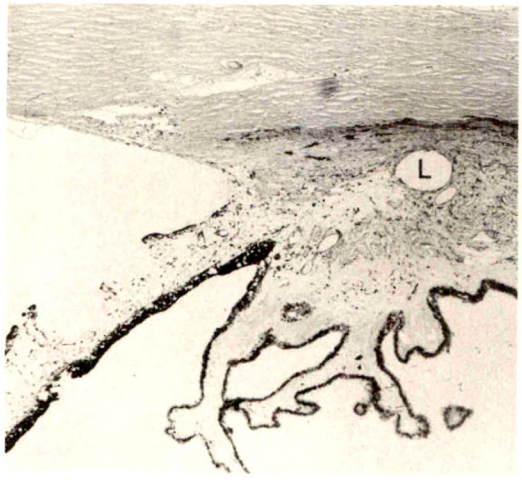


Fig. 6 (Crawford). Case 3. Superior loop (L) of Shearing intraocular lens in ciliary body (hematoxylin and eosin, ×10).

within the pars plicata of the ciliary body close to a major artery, and the inferior loop was fixed in the residual bag of lens capsule and peripheral cortex (Figs. 6 and 7). In Cases 1 and 3, a few epithelioid cells and giant cells surrounded the polypropylene loop embedded in the ciliary body (Figs. 7 and 8). None of the other supporting loops had provoked an inflammatory response.

Discussion

The Shearing posterior-chamber intraocular lens has been successfully implanted in many human eyes and is one of the more commonly used intraocular lenses.[6,7] Some surgeons prefer to insert the supporting loops in the lens capsule bag; others prefer to place them in the ciliary sulcus; still others believe that either of these locations provides good fixation. This study shows exactly where the supporting loops were located in three human eyes that had had Shearing intraocular lenses successfully implanted. The superior loops of two of the lenses were deeply embedded in the ciliary body, close to a major artery, and surrounded

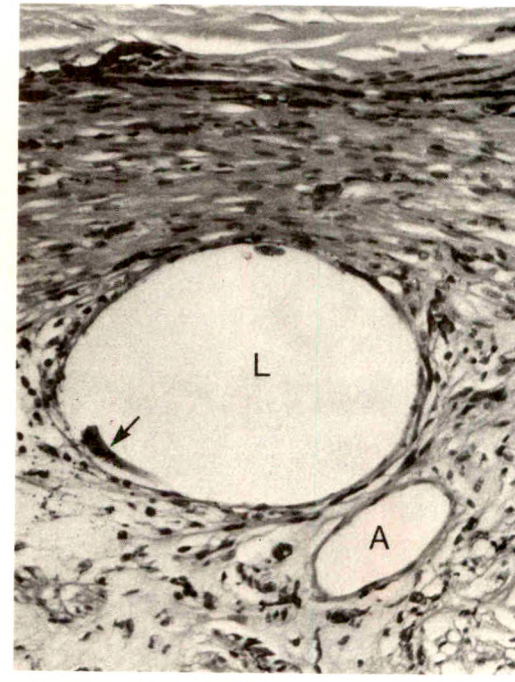


Fig. 7 (Crawford). Case 3. Giant cell (arrow) around superior polypropylene loop (L) embedded in ciliary body adjacent to an artery (A) (hematoxylin and eosin, ×64).

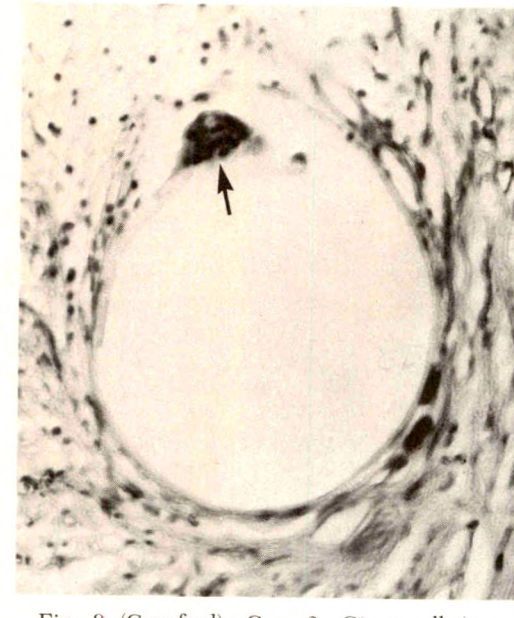


Fig. 8 (Crawford). Case 3. Giant cell (arrow) around superior loop of intraocular lens (hematoxylin and eosin, ×64).

by a slight foreign-body inflammatory response. The superior loop of the other lens was firmly fixed in the ciliary sulcus by adhesions between the posterior surface of the iris and the anterior face of the ciliary processes. The inferior loops of two of the lenses were located in the lens capsule bag, firmly fixed between residual anterior and posterior lens capsule and cortex. The inferior loop of the other lens was embedded within the iris stroma. In all three cases, the lens was well centered and firmly fixed in the posterior chamber of the eye. Olson, Morgan, and Kolodner[1-3] were concerned about possible bleeding caused by the migration of the polypropylene loop into the ciliary body. Despite the close proximity of the superior loop to a major artery in the ciliary body in Cases 1 and 3, neither of these eyes suffered from intraocular bleeding. Only the superior loops in Cases 1 and 3 provoked an inflammatory response; in both these cases the inflammatory response was minimal and had not destroyed a significant portion of the loop material. Olson and his associates[1-3] and Irvine[4] mentioned the potential difficulty in removing these lenses because of the firm fixation in the ciliary body of some of the supporting loops. Irvine suggested that if such a lens had to be removed, it might be prudent to cut the lens from its supporting loop and leave the loop in place.

ACKNOWLEDGEMENT

Steven Shearing, M.D., and Loren Little, M.D., donated these eyes to the University of California San Francisco Eye Pathology Laboratory.

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INTRAOCULAR LENS POWER CHECK

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AND

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We have developed a simple procedure that uses a standard keratometer to determine the power of an intraocular lens that is still in its sterile package. The procedure is accurate to within ±0.50 diopter and may be used in conjunction with any intraocular lens packaging system in which the convex side of a plano-convex intraocular lens is clearly visible through a clear, flat surface.

A recent study by McReynolds and Snider[1] reported that only 35% of some 300 intraocular lenses tested were the power indicated on their packages. Of the 300 lenses, 87% were within 2 diopters of the indicated power, but 13% were 2 to 9 diopters off. Although intraocular lens manufacturers are continually upgrading their quality control to minimize these errors, the ophthalmologist should be able to check the power of an intraocular lens sent to him by a manufacturer. Ideally, this power check could be made days or weeks before surgery without making the lens nonsterile. This power check would be particularly helpful for lenses with special powers.

Because the power of the intraocular lens primarily depends on surface curvature, the keratometer is an ideal device for measuring intraocular lens power. The keratometer, however, is internally calibrated for the combined indices of refraction of the cornea and tears, 1.3375, while the index of refraction of the plastic intraocular lens is 1.4917.[2] Thus, the dioptric power, or curvature, of the intraocular lens cannot be read directly by the keratometer. A conversion factor, K_1, must be used to account for this difference in indices of refraction. This factor is the ratio of the index of refraction of the intraocular lens minus that of air, over the index of refraction of the cornea minus that of air. If we assume that the index of refraction of the intraocular lens is 1.4917, that of air is 1.000, that of the cornea is 1.3375, and that of the aqueous humor is 1.336, then $K_1 = 1.457$.

When converting the power of the intraocular lens in air to that of the lens in aqueous humor, the power more familiar to clinicians, conversion factor K_2 must be used to account for these differences in indices of refraction. Thus, K_2 is the ratio of the index of refraction of the intraocular lens minus that of the aqueous over the index of refraction of the intraocular lens minus that of air: thus, $K_2 = 0.317$. The power of the intraocular lens in aqueous humor, as measured by a keratometer, may be summarized as follows:

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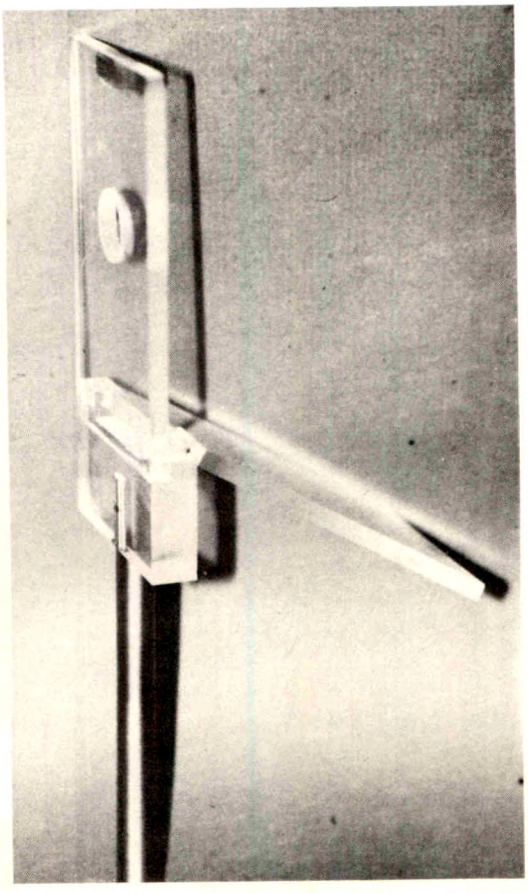


Fig. 1 (Miller and associates). Plastic holder for intraocular lens package. The hole is used to stretch and flatten the transparent package cover.

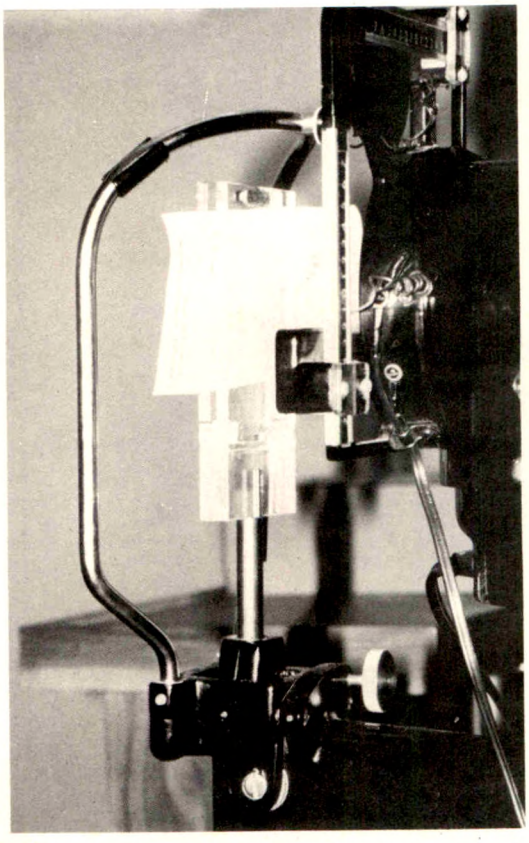


Fig. 2 (Miller and associates). Plastic holder with intraocular lens package, mounted facing mires of the keratometer.

$$^{F}IOL_{(aqueous)} = K_1K_2\ ^{F}IOL\ (keratometer)$$
$$^{F}IOL_{(aqueous)} = 0.46\ ^{F}IOL\ (keratometer).$$

The method used to derive this equation makes three important assumptions: (1) the intraocular lens is plano-convex; (2) the plano surface is a perfect plano surface; and (3) the power caused by lens thickness is negligible. Because this process requires the convex surface of the lens to be visible, the intraocular lens package must be made of clear, tightly stretched plastic on one side (Fig. 1).

The conventional keratometer is designed to measure the curvature of the cornea with the patient's head in an upright position. Therefore, we designed a holder for the lens package (Fig. 2). The holder must keep the intraocular lens in a vertical position with respect to the keratometer and keep the plastic covering directly in front of the intraocular lens flat to minimize distracting reflections.

Material and Methods

To test the method, 30 intraocular lenses between 10 and 25 diopters (in aqueous) were precisely measured with an optical bench. The lenses were then packaged as recommended (that is, with a tightly drawn, flat transparent window over the convex surface) and measured

TABLE

KERATOMETER READING TO DIOPTER CONVERSION

Lens Power (diopters)		
Optical Bench	Keratometer	T Lens*
25.0	25.4	+1.25
24.5	24.9	+1.25
24.0	24.5	+1.25
23.5	24.0	+1.25
23.0	23.0	0
22.5	22.5	0
22.0	22.0	0
21.5	21.5	0
21.0	20.9	0
20.5	20.4	0
20.0	20.0	0
19.5	19.5	0
19.0	19.0	0
18.5	18.5	0
18.0	17.9	0
17.5	17.4	0
17.0	17.0	0
16.5	16.8	−1.00
16.0	16.4	−1.00
15.5	15.7	−1.00
15.0	15.2	−3.00
14.5	14.4	−3.00
14.0	14.2	−3.00
13.5	13.7	−3.00
13.0	13.2	−3.00
12.5	12.7	−3.00
12.0	12.2	−3.00
11.5	11.7	−3.00
11.0	11.2	−3.00
10.5	10.9	−3.00

*T lens indicates that an auxiliary lens (specific power noted in column) was added to the keratometer to extend its range.

with the keratometer using the conversion factor of 0.46. The keratometric measurements were done by an individual unaware of the optical bench measurements.

RESULTS

We compared the optical power of each lens as determined by the optical bench method and by the keratometer method (Table). Both methods gave similar results, within 0.50 diopter or less, for each lens. For lenses above 23 diopters or below 17 diopters, auxiliary lenses must be used to extend the range of the keratometer.[3]

We have used this principle to confirm the power of an intraocular lens in a patient's eye. In the case in question, an extended-range attachment to the keratometer was used to prove that a mislabeled 19-diopter lens was really a 30-diopter lens.

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CONE PIGMENTS IN ACUTE POSTERIOR MULTIFOCAL PLACOID PIGMENT EPITHELIOPATHY

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We measured cone pigment density and kinetics with a reflection retinal densitometer during the evolution and resolution of posterior pole lesions in a 17-year-old patient with acute posterior multifocal placoid pigment epitheliopathy. Three weeks after onset, density was markedly reduced and regeneration complex and nonexponential. Pigment density increased, and kinetics returned to normal as the disease resolved.

On the basis of psychophysical dark-adaptation and color-matching studies, Smith and associates[1] predicted abnormal visual pigment density and kinetics in acute posterior multifocal placoid pigment epitheliopathy. Our measurements of cone visual pigments with a Rushton reflection retinal densitometer[2] confirmed these predictions. We made serial measurements of cone pigment density and regeneration during the evolution and resolution of bilateral posterior pole lesions in a 17-year-old girl with clinical and angiographic findings consistent with a diagnosis of acute posterior multifocal placoid pigment epitheliopathy.

MATERIAL AND METHODS

The reflection densitometer, which is based on the principles of the ophthalmoscope, directs a mixture of deep red ($\lambda > 685$ nm) control light and green ($\lambda = 559$ nm) measuring light into the eye. Some of this mixture is reflected from the fundus into a photomultiplier. The green light is absorbed by the photosensitive visual pigments and photostable structures in the eye. The deep red light is absorbed by photostable structures in the eye but not appreciably by the photosensitive visual pigments. Thus, changes in the mixture of red and green light reflected from the fundus into the photomultiplier are caused mainly by variations in the amount of visual pigment present. To measure changes in the amount of green light reflected from the fundus, a neutral density wedge in the red control beam is adjusted to equalize the amounts of red and green light reaching the photomultiplier. Wedge transmissivity—that is, the proportion of incident light that passes through the neutral density wedge—is used to estimate the fraction of cone pigment present, p, and the optical density of the pigment, $D(\lambda)$.

Previous investigators[3,4] have shown that the wedge transmissivity of the dark-adapted fovea, T_D, and fully bleached fovea, T_0, are related to pigment density, $D(\lambda)$, as follows:

$$(T_0 - T_D) / T_0 = (1 - s)\,[1 - 10^{-2D(\lambda)}]$$

where s represents the fraction of stray light (light that has not passed through

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the visual pigment) emerging from the eye. For the normal eye, if stray light is assumed to be zero and constant from session to session, this equation gives the lower limit of pigment density.[3] However, because of changes in photoreceptor orientation[1] and in the configuration of the light yellow fundus lesions, stray light may be abnormal and variable during the course of acute posterior multifocal placoid pigment epitheliopathy.

Wedge transmissivity, T, at any time is related to proportion of pigment present, p, and to pigment density, $D(\lambda)$, as follows[3,4]:

$$(T_o - T)/(T_o - T_D) = [1-10^{-2D(\lambda)p}]/[1-10^{-2D(\lambda)}].$$

If $D(\lambda)$ is assumed to be low, the higher-order terms of the expansion of $1-10^{-2D(\lambda)}$ may be ignored, and the equation simplified to:

$$(T_o - T)/(T_o - T_D) = p.$$

The patient was instructed to fixate coarse cross-hairs in the beam of light directed into the eye. Fixation was usually stable for the seven to ten seconds needed to adjust the wedge. Poor fixation was easily detected because it was associated with large, transient changes in reflection. We discarded these readings and repeated the measurements a few seconds later when the eye was stable. Cone pigments in a 2-degree patch of affected retina centered on the fovea were measured after exposure to light (6.3 log photopic trolands for two minutes) that bleached over 99% of the cone pigments in a concentric 5-degree patch of retina. As in the normal eye, bleaching increased the amount of green light reflected from the fundus.

After the bleaching light was extinguished, wedge settings, T, were determined repeatedly as pigment regeneration took place, and wedge transmissivity decreased to the dark-adapted value, T_D. We measured the wedge transmissivity for the fully bleached fovea, T_o, and the completely dark-adapted fovea, T_D, on each visit. Assuming pigment density is low, the proportion $(T_o - T)/(T_o - T_D)$ gives p, the fraction of pigment present (equations 2 and 3).

The regeneration of normal cone pigments after a 99% bleach is approximated by a simple exponential function with a time constant of 120 seconds.[5] If the logarithm of $(1-p)$ is plotted as a function of time, the exponential function is transformed into a straight line; the time constant, t_o, of the exponential can be obtained from the slope of the straight line. This transformation facilitates detection of abnormal kinetics because regeneration that does not follow a simple exponential appears as a deviation from a straight line, and a simple exponential with an abnormal time constant appears as a straight line with abnormal slope.

Results

Three weeks after the onset of symptoms, when parafoveal lesions were evolving, the course of pigment regeneration was slower and more complex than normal (Figure). One week later, regeneration was exponential, but the time constant was 210 seconds. Eight weeks after onset, when visual acuity was 6/12 (20/40) and the placoid lesions were resolving, cone pigment regeneration was normal. With further resolution of the placoid lesions and improvement of visual acuity 13 and 21 weeks after onset, the time course of pigment regeneration remained normal.

Three weeks after onset, $(T_o - T_D)/T_o$ was low, but thereafter it increased steadily to reach the lower limit of our normal range 13 weeks after onset (Table).

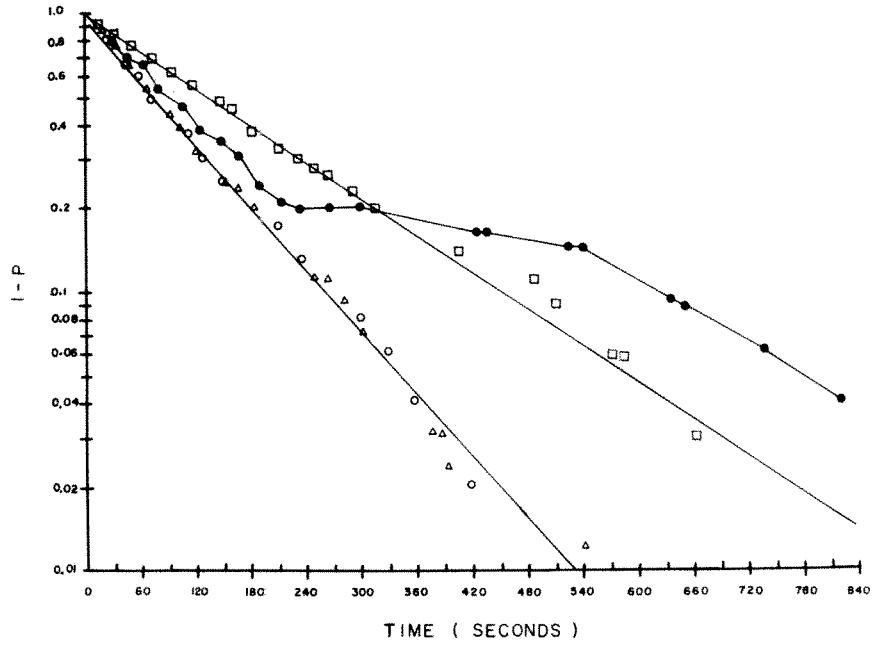


Figure (Hansen and Fulton). Cone pigment regeneration in acute posterior multifocal placoid pigment epitheliopathy. The logarithm of (1-p), the fraction of bleached pigment, is plotted as a function of time in seconds. Measurements were made three weeks (solid circles), four weeks (squares), eight weeks (open circles), and 13 weeks (triangles) after the onset of symptoms. Straight lines represent exponential functions with time constants, t_o, shown in the Table.

Discussion

Previous investigators have shown that $(T_o - T_D)/T_o$ depends not only on pigment density but also on the amount of stray light emerging from the eye.[3,4] As noted above, stray light may be abnormal and variable in this condition; consequently, changes of $(T_o - T_D)/T_o$ in our patient may have been caused by either variations in pigment density or stray light, or both. The pale yellow lesions could have increased fundus reflectance and caused $(T_o - T_D)/T_o$ to be low. How-

TABLE

CONE PIGMENTS IN ACUTE POSTERIOR MULTIFOCAL PLACOID PIGMENT EPITHELIOPATHY

Weeks After Onset	Visual Acuity	$(T_o-T_D) / T_o$ at 559 nm*	t_o (sec)
3	6/60 (20/200)	0.257	—†
4	6/60 (20/200)	0.292	210
8	6/12 (20/40)	0.366	120
13	6/7.5 (20/25)	0.369	120
21	6/6 (20/20)	0.383	120

*The average normal value of pigment density, given by $(T_o - T_D)/T_o$, is 0.419 (range, 0.369 to 0.543; No. = 9).

†Pigment regeneration was complex, and not a simple exponential. See Figure.

ever, $(T_o - T_D)/T_o$ was higher at four weeks than at three weeks even though the lesions were more extensive. Smith and associates[1] documented photoreceptor malorientation that recovered during a seven-month period in another patient with acute posterior multifocal placoid pigment epitheliopathy. Receptor misalignment may alter light reflected from the fundus, but even considering that the amount of time needed for recovery varies from patient to patient, it seems unlikely that changes in receptor orientation could account for the marked increase in $(T_o - T_D)/T_o$ that we observed during the first eight weeks after the onset of symptoms. Thus, we concluded that increases in $(T_o - T_D)/T_o$ were caused by increases in pigment density.

Cone visual photopigment density and kinetics became normal as visual acuity and fundus appearance improved. The improvement in pigment density was more gradual than was the return to normal kinetics. The psychophysical data of Smith and associates[1] suggested that cone kinetics become normal before density does; cone dark-adaptation function appeared normal seven months after the acute phase, but abnormal color matches, which indicated reduced pigment density, persisted as long as 13 months after the acute phase. The marked discrepancy between the time needed for healing in this patient and that described by Smith and associates[1] may have been caused by the variable effect of this disease on visual function. Alternatively, densitometric and psychophysical methods may give different assessments of cone pigment densities and kinetics. Nevertheless, it is clear that this infrequently seen disease, which primarily involves the pigment epithelium or choroid,[6] also affects cone visual photopigment density and regeneration.

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LIPOIDAL DEGENERATION OF THE RETINAL PIGMENT EPITHELIUM

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Vacuolation of the retinal pigment epithelium in the foveomacular region of aging rhesus monkey eyes was re-examined with the eyes initially fixed in osmium tetroxide to retain the alcohol-soluble lipid. All vacuoles were found to be completely occupied by lipid material. Similar cells were positive to oil red O. The vacuolation of the pigment epithelial cell is considered to be a form of lipoidal degeneration.

In a previous report,[1] a form of presumed lipoidal degeneration of the retinal pigment epithelium in the aging rhesus monkey eye was described. Although the membrane-bound vacuoles were mostly empty, we assumed that lipoidal material had been lost during processing of the tissue because of initial fixation in glutaraldehyde and subsequent dehydration in ascending concentrations of ethanol. To test this idea, three additional monkey eyes from our series, selected because they possessed the myriad characteristic foveomacular white dots, were initially fixed in osmium tetroxide and subsequently prepared for electron microscopic examination. Additionally, the foveomacular region of another similarly selected eye was frozen after glutaraldehyde fixation, and the frozen sections were treated with oil red O. With these exceptions, the methods used in this study were the same as in the previous report.[1]

From the Department of Ophthalmic Pathology, Armed Forces Institute of Pathology, Washington, D.C. This investigation was supported by research grant EY 03060 from the National Eye Institute.

The opinions or assertions contained herein are the private views of the author and are not to be construed as official or as reflecting the views of the Department of the Army or the Department of Defense.

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RESULTS

Retinal pigment epithelial cells located sporadically in the region of the foveomacula stained positive with oil red O.

By electron microscopy, cells containing many vacuoles showed complete filling of all vacuoles when the tissue was initially fixed in osmium tetroxide. By light microscopy, the affected cells fixed in osmium tetroxide showed a heavy content of dense granules (Fig. 1, inset), in striking contrast to their pale appearance after fixation with glutaraldehyde and dehydration by alcohols (Fig. 2, inset).

The lipoidal globules clearly formed two groups, one of large globules and another of smaller ones. The group of larger globules contained dark (electron-dense) and light (less dense), homogeneous, membrane-bound globules. Many globules contained a small "cap" of more dense material. Occasionally bubbles of less dense lipid were seen within the caps (Fig. 1). The density of the bubbles resembled that of the content of adjacent smaller globules, which were of the dark variety only. Staining, nonstaining (Fig. 3), and partial staining of the thin sections for electron microscopy did not significantly alter the density relationships of the lipoidal globules.

All the lipoidal globules differed markedly from other cell inclusions such as

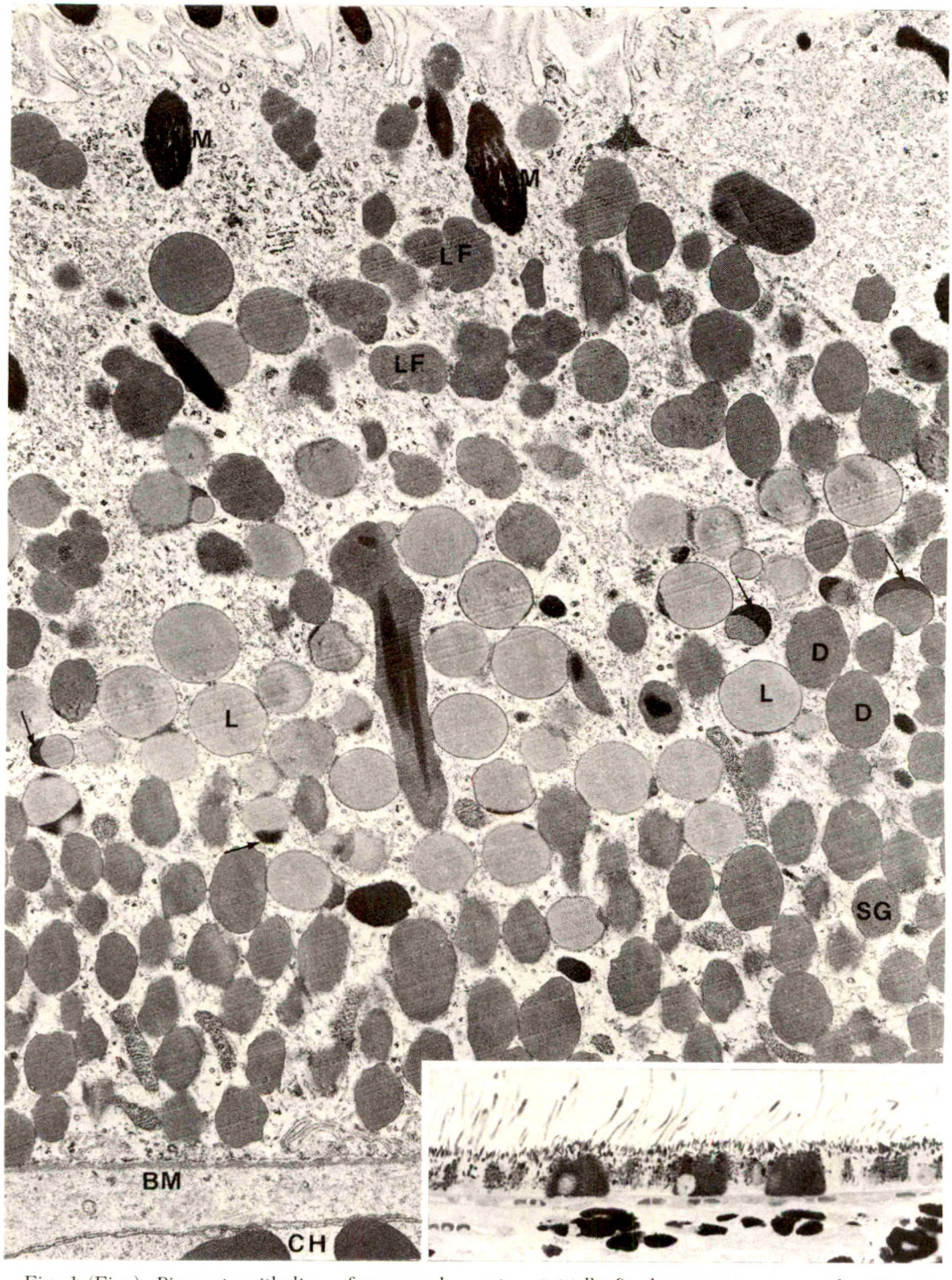


Fig. 1 (Fine). Pigment epithelium, foveomacular region, initially fixed in osmium textroxide. Pigment epithelial cell is filled with membrane-bound globules of various sizes. The most spherical globules are filled with homogeneous, less dense material (L). Similar-sized, slightly more ovoid globules contain dense material (D). Similar dense material is present in small globules (SG). Small caps (arrows) of denser material are present on many of the globules. Other large, homogeneous granules are even more dense and generally nonspherical in shape with some granular content (lipofuscin, LF) (see Fig. 4). Melanin granules (M) are exceedingly dense, generally ovoid to spherical in shape, and easily distinguished from the other types of cell inclusions, especially when their rodlet composition is visible. Bruch's membrane (BM) and the adjacent choriocapillaris (CH) are normal (× 10,000). Inset, Distribution of densely stained pigment epithelial cells with lipoidal degeneration in the region of the fovea (paraphenylenediamine, × 400).

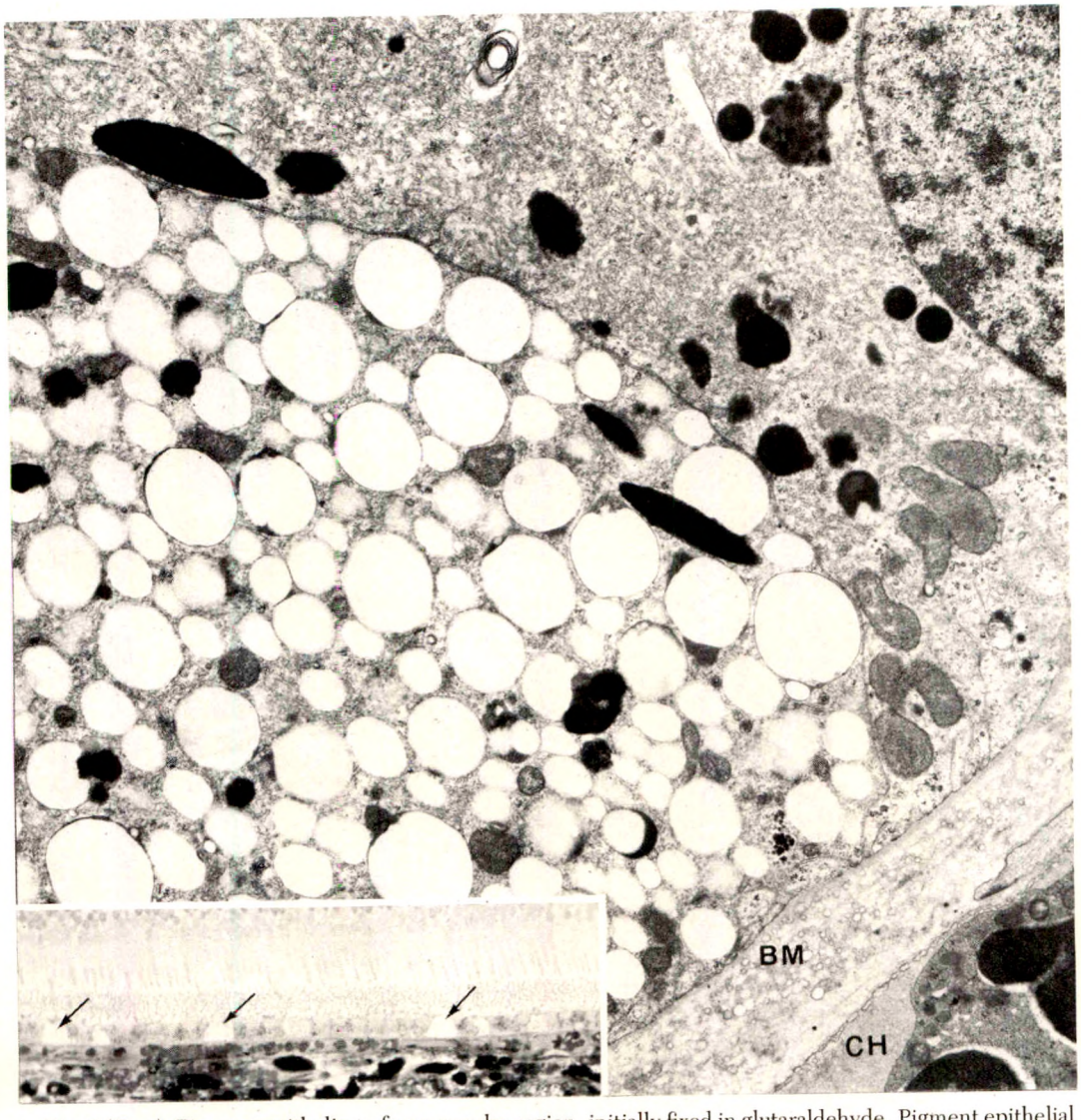


Fig. 2 (Fine). Pigment epithelium, foveomacular region, initially fixed in glutaraldehyde. Pigment epithelial cell at left is grossly vacuolated (lipoidal degeneration). Only a few of the membrane-bound vacuoles show contents suggestive of lipoidal material. Bruch's membrane (BM) and the adjacent choriocapillaris (CH) are normal (×10,400). Inset, The affected retinal pigment epithelial cells (arrows) are lighter than the adjacent cells when treated with glutaraldehyde and alcohol (toluidine blue, ×165).

lipofuscin (more dense and more irregular in shape) and melanin granules (extremely dense and ovoid to spherical in shape) and from phagosomes (containing much membranous material) (Fig. 4).

Discussion

Although it seemed reasonable to conclude, as we did previously, that the material removed from the cytoplasmic globules of the retinal pigment epithelium consisted of an ethanol-soluble fat or lipid, it was necessary to re-evaluate the observations by means of initial fixation with osmium tetroxide, which would prevent reactive (unsaturated) lipid from being extracted by alcohol. The contents of the membrane-bound globules were

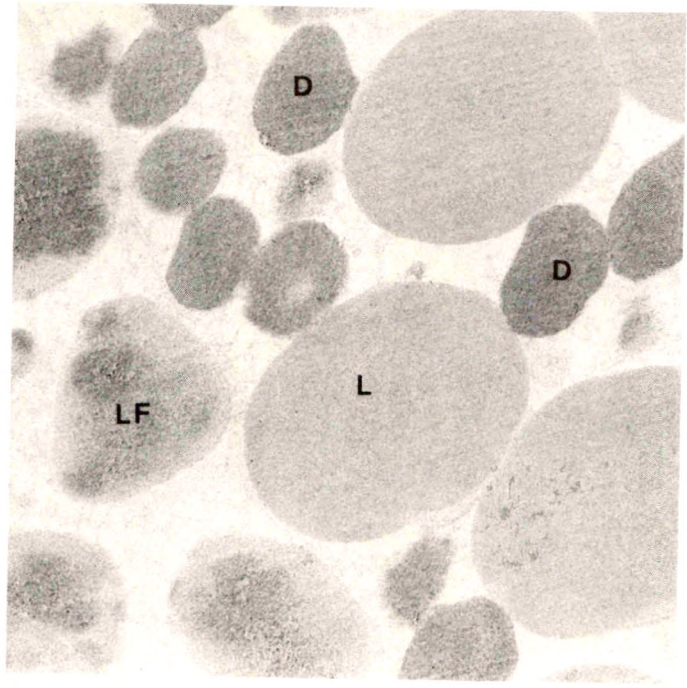


Fig. 3 (Fine). Retinal pigment epithelial cell inclusions fixed in osmium tetroxide with no counterstain (without uranyl acetate or lead citrate). Lipoidal globules of both the light (L) and dark (D) varieties are present. The more irregularly shaped lipofuscin granules (LF) are more easily identified by the greater difference in density between their central and peripheral areas (See Fig. 4) (×26,000).

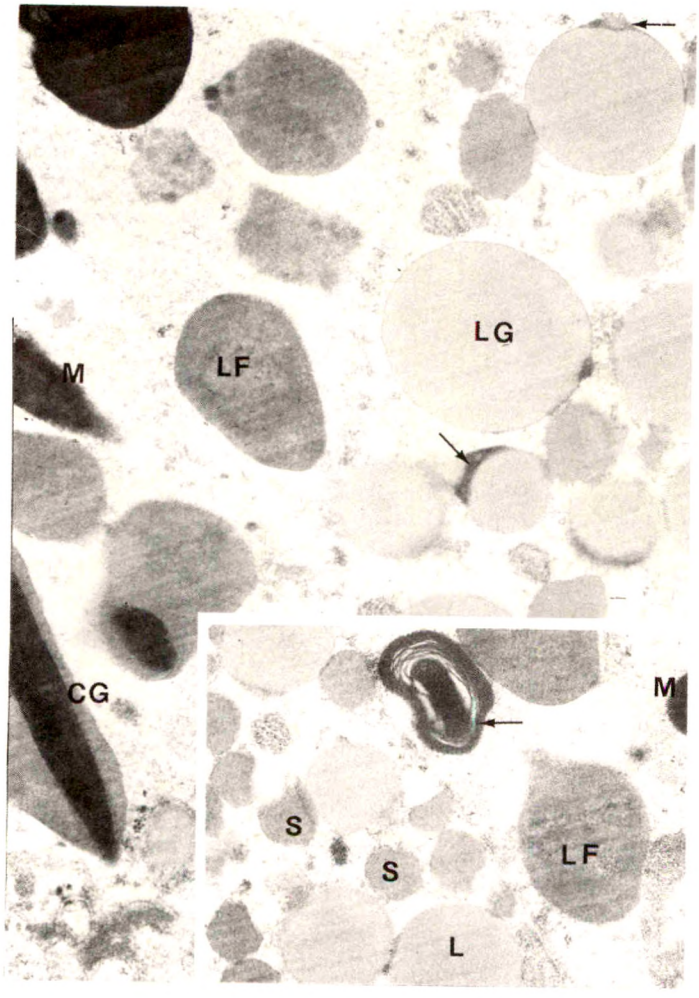


Fig. 4 (Fine). Morphologic differences between the more dense, irregularly shaped globules of lipofuscin (LF) and the less dense, spherical globules of lipid (LG) can be seen. Melanin granules (M) are extremely dense and easily identified. Melanin encased in lipofuscin forms a compound granule (CG). Note the difference in density between large globules (LG) and small globules. Dense caps are present (arrows) on many globules (×17,500). Inset, Phagosomes (arrows) are present in retinal pigment epithelial cells in addition to melanin (M), lipofuscin (LF), and large (L) and small (S) lipoidal globules (×17,000).

preserved. The globules were present in two distinct forms, electron-dense (dark) and less dense (light). The darker granules were present in both large and small sizes. The smaller globules, however, were always dense or dark. There is probably a transition from small dense globules to large dense globules. The caps of the large, light globules resembled the more dense globules. The observations suggested the presence of accumulating lipid in two slightly different forms, both of which, however, are soluble in ethanol. In some of the lighter globules, the lipid may accumulate first as small dense globules, with the less dense lipid later appearing and attenuating the denser material until the latter appears to be a cap on the periphery of the globule (Fig. 1).

As previously noted,[1] the lack of evidence for any exudative phenomena in the vicinity of the cell strongly suggests that degeneration takes place within the cell, causing the lipoidal materials to accumulate in the characteristic globular form. Similar lipoidal degeneration of the retinal pigment epithelium can also occur after mild trauma.[2] It appears analogous to the lipoidal degeneration that occurs in some cells, often referred to as "balloon cells," of choroidal melanomas.[3-5] Although the latter are also positive to oil red O,[4,5] no electron microscopic study has been done with these tumors initially fixed in osmium tetroxide. Similar changes have also been recently reported as "lipidization" in respiratory epithelium and underlying stromal fibroblasts and mast cells in sino-orbital polyposis.[6]

A retinal pigment epithelial cell, therefore, may contain lipoidal granules in addition to the more usual melanin and lipofuscin granules and phagosomes and their end products, residual bodies.

Summary

Vacuolation of the retinal pigment epithelium in the foveomacular region of aging rhesus monkey eyes was reexamined with the eyes initially fixed in osmium tetroxide to retain the alcohol-soluble lipid. All vacuoles were found to be completely occupied by lipid material. Similar cells were positive to oil red O. The vacuolation of the pigment epithelial cell is considered to be a form of lipoidal degeneration.

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SURGICAL MANAGEMENT OF RETINAL DETACHMENT WITHOUT CRYOPEXY

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A clinical study of eyes with rhegmatogenous retinal detachment compared the reattachment rate in two groups of eyes: in the control group, cryopexy was applied over the retinal breaks during surgery; in the test group, cryopexy was not applied.

There was a similar rate of retinal reattachment in the two groups in the immediate postoperative period. However, during the long follow-up period (36 months) there was a considerable difference in the two groups. Eyes that had undergone cryopexy had a lower rate of redetachment than eyes that had not. The rate of operative and postoperative complications was low in both groups, except for peri-retinal proliferation, which was more frequent in the test group. These results suggest that cryopexy may lower the incidence of redetachment after retinal detachment surgery.

Surgical management of retinal detachment often involves three procedures: scleral buckling, cryopexy, and puncture for release of subretinal fluid. These steps counteract vitreous traction and strengthen chorioretinal adhesion forces.[1] Because none of these procedures is free of complications,[2-5] surgeons try to avoid them whenever possible.[6,7]

Zauberman and associates[8-12] introduced a new approach to rhegmatogenous retinal detachment surgery. They operated on 37 eyes with retinal detachment but did not induce chorioretinal lesions during the surgery. The authors suggested that cryopexy during surgery was superfluous in most types of retinal detachment, when vitreous traction was successfully counteracted by a scleral buckling procedure.

We carried out a prospective study to evaluate the efficacy of this surgical approach.

MATERIAL AND METHODS

We included 70 eyes with rhegmatogenous retinal detachment which underwent retinal surgery in the study (Table 1). Various staff members and residents operated on the eyes between January 1975 and May 1976. Table 1 shows type, duration, and extent of the retinal detachments. The test group (41 eyes) underwent scleral buckling procedures, some with and some without puncture for release of subretinal fluid, but did not undergo cryopexy during the surgery. This group was composed of 30 phakic and 11 aphakic eyes. In ten eyes the retinal detachment had recurred after previous surgery.

The control group, 29 eyes with similar rhegmatogenous retinal detachments, underwent scleral buckling procedures, some with and some without release of subretinal fluid, but also underwent cryopexy applied to the retinal breaks during

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TABLE 1

RETINAL DETACHMENT

Detachment	Test Group (N = 41)	Control Group (N = 29)
Type		
Phakic	30	22
Aphakic	11	7
Redetachment	10	5
Duration		
< 1 wk	19	13
1 wk to 1 mo	6	6
> 1 mo	13	6
Unknown	3	4
Extent		
Group I	9	6
Group II	22	20
Group III	10	3
Detached macula	31	20
Attached macula	10	9

surgery. This group contained 22 phakic and seven aphakic eyes. Five of the eyes in this group had recurrent retinal detachment.

The first 11 eyes operated upon did not receive cryopexy; the rest of the eyes were allocated randomly to the two groups. This made the test group larger than the control group.

The preoperative refractive errors are shown in Table 2 and the surgical methods in Table 3. We classified the retinal detachments according to their extent, as Ashrafzadeh and Schepens[13] suggested: Group I included retinal detachments involving no more than one quadrant of the fundus, Group II consisted of retinal detachments involving more than one but less than four quadrants of the fundus, and Group III was composed of eyes with total retinal detachments.

The surgical procedures included intrascleral and episcleral local or circular buckling. We removed subretinal fluid by puncture unless the fluid was so minimal that the procedure was not justified. In some cases we injected saline or air into the vitreous cavity.

TABLE 2

REFRACTIVE ERRORS OF THE INVOLVED EYES

Refractive Errors	Test Group	Control Group
Hypermetropia	—	1
Emmetropia	11	9
Myopia	17	10
Severe myopia	13	9

We evaluated the postoperative results for each eye at three different periods: at one week, at eight months, and at 36 months after the operation. Three patients from the test group and four patients from the control group failed to appear for the last examination.

RESULTS

In the test group, where the operation was performed without cryopexy, we found reattachment of the retinas one week postoperatively in 37 of 41 cases (90%) (Table 4). After eight months, 28 of 41 eyes (68%) had attached retinas. After 36 months only 19 of 38 patients (50%) still had attached retinas.

In the control group, where cryopexy was applied, we found reattachment of the retinas one week postoperatively in 27 of 29 eyes (93%). After eight months, 24 of 29 eyes (83%) had attached retinas. After 36 months, 18 of 25 patients (72%) still had attached retinas.

Table 4 shows the different anatomic

TABLE 3

SURGICAL METHODS

Surgical Procedure	Test Group	Control Group
Local buckle	3	2
Encircling buckle	38	25
Scleral resection	—	2
Release of subretinal fluid	35	22
Intravitreal injection	9	7

TABLE 4

ANATOMIC RESULTS

Time After Surgery	Test Group			Control Group		
	Phakic	Aphakic	Total	Phakic	Aphakic	Total
1 week						
No.	30	11	41	22	7	29
Attached	28	9	37	20	7	27
Detached	2	2	4	2	0	2
8 months						
No.	30	11	41	22	7	29
Attached	21	7	28	19	5	24
Detached	9	4	13	3	2	5
36 months						
No.	27	11	38	18	7	25
Attached	15	4	19	13	5	18
Detached	12	7	19	5	2	7
% attached	56	36	50	67	70	72
% detached	44	64	50	33	30	28

results in phakic and aphakic eyes with retinal detachment. In the test group, 28 of 30 phakic retinal detachments (93%) were reattached after one week. After eight months, 21 of 30 (70%) of the retinas remained attached. After 36 months, only 15 of 27 eyes (56%) had attached retinas. In the aphakic group, nine of 11 eyes (82%) had attached retinas one week postoperatively; seven of 11 retinas (64%) remained attached after eight months; and only four of 11 eyes (36%) had attached retinas after 36 months. In the control group, reattachment of the retinas was observed one week postoperatively in 20 of 22 phakic eyes (90%). After eight months, 19 of 22 of the eyes (86%) had attached retinas; after 36 months, 13 of 18 of these phakic eyes (67%) had attached retinas. In the aphakic group, seven of seven (100%) had flat retinas one week postoperatively, five of seven retinas (70%) were still attached after eight months, and five of seven eyes (70%) still had attached retinas after 36 months.

Table 5 shows the operative and postoperative complications for both groups and Table 6 the causes of failure, primarily preretinal fibrosis and seepage of subretinal fluid through open retinal tears.

DISCUSSION

The nature of the chorioretinal adhesive forces in man, both natural forces and those induced at chorioretinal scars by diathermy, photocoagulation, and cryopexy, is still not sufficiently clear. Zauberman and Rosell[12] based their new

TABLE 5

POSTOPERATIVE COMPLICATIONS

Complications	Test Group	Control Group
Immediate postoperative		
Choroidal detachment	7/41	2/29
Choroidal and retinal hemorrhage	5/41	1/29
Vitreous hemorrhage	2/41	2/29
Late postoperative		
Preretinal fibrosis	14/38	5/25
Infected buckle	1/38	1/25
Maculopathy*	4/38	3/25
Vitreous opacities	4/38	3/25

*Maculopathy included macular edema, macular hole, and pigmentary changes.

TABLE 6

CAUSES OF FAILURE

Causes	Test Group	Control Group
No. of failures	19	7
Preretinal fibrosis	13	5
Buckle malposition	2	–
Seepage of subretinal fluid through open original retinal break	3	2
New or missed break	1	–

approach to retinal detachment surgery on experimental studies that measured the adhesive forces between the retina and the pigment epithelium.[8-10] However, the results of experimental studies carried out by other investigators[14-18] did not always support their observations, and Zauberman himself demonstrated in a later work[11] that dense chorioretinal scars resist penetration of subretinal fluid better.

Chignel[19] reported a series of 29 selected cases of retinal detachment treated with local scleral implants without cryopexy. Reattachment of the retina was achieved in 25 patients; one patient underwent further photocoagulation and three initial failures attributed to buckle malpositioning were successfully treated in second operations. The follow-up period was shorter than in our series (six to 24 months), and the selection of retinal detachment cases in which the buckle could be located with reasonable certainty and which required local implant alone, might have contributed to the good surgical results.

Fetkenhour and Hauch[20] recently reported a series of 175 consecutive, unselected patients with retinal detachments that were treated with a scleral buckling procedure and without an induced thermal adhesion. Initial reattachment was obtained in 143 (82%) cases and final success was achieved in 158 (90%) of the patients. These surgical results were maintained during a follow-up period of six months.

The immediate postoperative results in our test group confirm previous observations that retinal detachment surgery without cryopexy application is effective in reattaching 90% of the cases.[12,19,20] However, after eight months, the rate of success in the test group was lower than in the control group (68% vs 83%), and after three years, the percentage of eyes in which the retinas remained attached was much lower in the test group (50%) than in the control group (72%), even though vitreous traction was similarly counteracted in both groups by the buckling procedures during surgery.

Although most of the late redetachments in the test group resulted from preretinal fibrosis, three of them were caused by leakage of subretinal fluid through untreated open retinal breaks. Two other cases of late failure in the control group were also caused by seepage of fluid through original retinal breaks that were not entirely sealed by the cryotherapy. Those five cases were treated by argon-laser photocoagulation applied around the redetaching retinal break. The treatment invariably resulted in flattening or arrest of the retinal detachment. The postoperative course of these five cases suggested that the adhesive forces of the retina surrounding the open breaks may not be sufficient to keep the retina attached, and that the scar of a cryopexy-treated retinal tear adheres better to the pigment epithelium.

Most of the failures in the test group (13 of 19) were attributed to proliferation of periretinal membranes. Although this group included more cases of long-standing retinal detachments and recurrent detachments, a further analysis of the cases disclosed that this type of complica-

tion was equally distributed in the new and the recurrent detachments.

Fetkenhour and Hauch[20] observed periretinal proliferation in 10% of the patients in their large series. Zauberman and Rosell[12] did not specify the cause of redetachment, and Chignell[19] did not mention periretinal proliferation as a cause of surgical failure. In our test group, 13 of 38 (34%) patients developed redetachment of the retina caused by periretinal proliferation, whereas only five of 25 (20%) control patients developed periretinal proliferation that caused redetachment. In many cases the redetachment caused by traction of proliferating membranes occurred several months or years postoperatively.

The causes and mechanism of periretinal proliferation are still unknown. Machemer and associates[21-23] investigated proliferation of pigment epithelial and glial cells in areas where the pigment epithelium was in direct contact with the vitreous. Foos[24] has shown that breaks in the internal limiting membrane of the retina allow retinal glial cells to migrate and form cellular membranes on the retinal surface.

We agree with Fetkenhour and Hauch[20] that thermal lesions do not increase the risk of massive periretinal proliferation. Moreover, although biased by a larger number of long-standing cases, the test group exhibited an unusually high percentage of periretinal proliferation.

It is interesting to speculate on the role an open retinal break, through which pigment epithelial and glial cells may migrate and proliferate, plays in causing periretinal proliferation. Sealing of the breaks and destroying the bare pigment epithelium by cryopexy application may decrease migration and proliferation.

We concluded that retinal detachment surgery without cryopexy lesions results in a high rate of success in the immediate postoperative period, but is inferior to the traditional operation with respect to the rate of the late redetachments. Most redetachments are caused by periretinal proliferation, but some result from seepage of subretinal fluids through unsealed retinal breaks. Cryoapplication does not increase the incidence of postoperative complications but does seem to decrease the rate of periretinal proliferation.

Until more experimental and clinical data concerning this issue become available, it seems better to rely upon the long-term postoperative results that favor the use of cryopexy on retinal breaks during retinal detachment surgery.

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An anecdote illustrates his lifelong fondness for becoming acquainted with facts at their source. He had heard that in the Danube between Kritzendorf and Klosterneuburg there was a whirlpool which sucked even good swimmers under water. This was in his early college life. He persuaded friends to take him to the spot and jumped from his boat into the water. The whirlpool seized him and he was hauled into the boat half-conscious.

Professor K. Lindner, More About Ernest Fuchs
Am. J. Ophthalmol. 14:335, 1931

OCULAR RETICULUM CELL SARCOMA

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Three patients with ocular reticulum cell sarcoma underwent treatment with a combination of chemotherapy and irradiation. All three patients have survived an average of 36 months after treatment, significantly longer than would have been expected with radiation therapy alone. The chemotherapy appears to arrest the progression of central nervous system disease that eventually kills most patients with ocular reticulum cell sarcoma.

Ocular reticulum cell sarcoma is a rare lymphoma with an unusual predilection for the eye and central nervous system.[1] We have previously reported the favorable response to irradiation in ocular and central nervous system disease.[2] However, we noted that seven of nine patients eventually died of central nervous system disease progression. The average interval between diagnosis and death in these patients was approximately 12 months.[2] Chemotherapy has markedly improved the survival rate of patients with diffuse histiocytic lymphoma, the systemic tumor most similar to ocular reticulum cell sarcoma.[3] We treated three patients with ocular reticulum cell sarcoma with a combination of irradiation and chemotherapy; all three had improved vision and significantly prolonged survival.

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CASE REPORTS

Case 1—This 50-year-old man came to his primary ophthalmologist in September 1977, complaining of floaters. An examination disclosed bilateral vitreous cellular opacities. In the left eye there was a posterior uveitis characterized by yellow-white chorioretinal lesions. The ocular disease did not respond to high-dose systemic corticosteroids. A biopsy specimen of the left vitreous was consistent with, but did not prove, reticulum cell sarcoma. Results of a computed tomographic scan of the brain and a bone marrow biopsy were both within normal limits.

The patient underwent a six-week course of irradiation; he received 4,500 rads to the left eye and orbit in October 1977. The patient did well until January 1979, when the symptoms recurred and bilateral vitreous opacities were found. Treatment with a second course of systemic corticosteroids was ineffective.

When the patient was referred here in March 1979, his visual acuity was R.E.: 6/4.5 (20/15) and L.E.: 6/6 (20/20). Results of an anterior segment examination were within normal limits. Vitreous cells were present bilaterally, and the left fundus showed reticulum cell sarcoma lesions in a postradiation stage, characterized by scarring at the level of the retinal pigment epithelium.

The patient noted a gradual decrease in the visual acuity of his left eye and was admitted to the medical center here for examination and therapy in September 1979. On admission, his visual acuity was R.E.: 6/4.5 (20/15) and L.E.: 6/120 (20/400). The results of an anterior segment examination were within normal limits; the left vitreous showed 4+ cells and the right vitreous 2+ cells. There were no chorioretinal lesions in the right fundus. The view of the left fundus was obscured by dense vitreous cellular opacities. A vitreous biopsy and vitrectomy on the left eye resulted in a visual acuity of 6/9 (20/30).

Findings from a physical examination, bilateral bone marrow biopsies, serum chemistry studies, and



Fig. 1 (Char, Margolis, and Newman). Case 1. Brain computed tomographic scan with contrast demonstrating frontal lobe tumor (T).

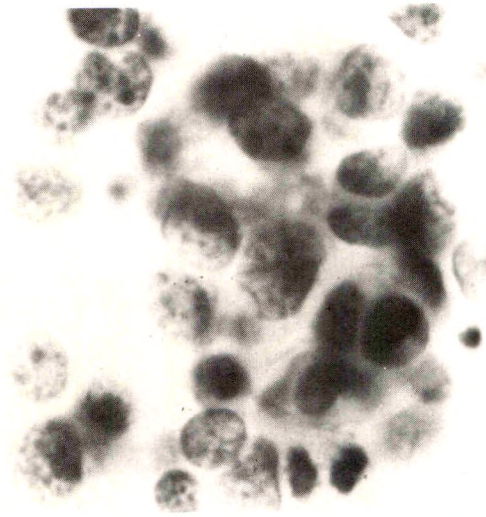


Fig. 2 (Char, Margolis, and Newman). Case 1. Cytologic preparation demonstrating large pleomorphic cells with eccentric nuclei.

a body computed tomographic scan were within normal limits. Computed tomography of the brain disclosed an area of tumor involvement (Fig. 1); cytologic evaluation of both the cerebrospinal fluid and the vitreous demonstrated malignant cells diagnostic of reticulum cell sarcoma (Fig. 2). Immunologic characterization of these demonstrated the absence of surface markers (null cells).[4]

The patient underwent an additional course of 4,000 rads of whole-brain and orbital irradiation, including the previously irradiated left eye. He also received six courses of intrathecally administered methotrexate and one consolidation course of intrathecally administered cytarabine. The patient is alive and well approximately one year after his second course of irradiation and shows no evidence of tumor.

Case 2—This 62-year-old man had a two-year history of decreased visual acuity and floaters with more severe involvement in the right eye. Systemic and topical corticosteroid treatment of his bilateral vitritis was unsuccessful. Approximately two months before he was admitted here, he had had an episode of disorientation, the cause of which was unknown.

An examination in June 1978, showed that the patient's visual acuity was R.E.: hand motions and L.E.: 6/9 (20/30). A bilateral diffuse uveitis was present. Significant anterior segment findings included bilateral fine cells. Ophthalmoloscopic examination showed 3+ vitreous cells and yellow-white chorioretinal lesions similar in appearance to those in Case 1.

A vitreous aspirate was performed on the right eye. Cytologic evaluation of both vitreous and cerebrospinal fluids was consistent with the diagnosis of ocular reticulum cell sarcoma.

Metastatic evaluation disclosed evidence of both central nervous system and systemic disease. A brain computed tomographic scan demonstrated a lesion in the left frontal lobe. A body computed tomographic scan gave normal results. However, a biopsy specimen from a mass on his left calf was consistent with a histiocytic lymphoma.

The patient underwent 4,000 rads of whole-brain irradiation, six courses of intrathecally administered methotrexate, and three courses of combination therapy with intravenously administered cyclophosphamide, vinblastine sulfate, prednisone, and procarbazine. The patient's visual acuity has been stabilized bilaterally. The patient is alive and well several months after therapy.

Case 3—This 38-year-old man, examined in 1973, had a five-year history of visual clouding. One year before examination here, he had noted the onset of neural hearing loss, episodic gait instability, and weakness. A thorough neurologic evaluation was undertaken. Results of a cytologic examination of the cerebrospinal fluid were consistent with reticulum cell sarcoma.

On ocular examination the patient's visual acuity was 6/22 (20/70) bilaterally. Results of an anterior segment examination were unremarkable. The patient had vitreous cells bilaterally; the right vitreous had 2+ cells and the left vitreous 4+ cells. There were some yellow-white lesions in the right fundus.

Because of the extensive central nervous system

and ocular disease, the patient received 5,000 rads of whole-brain radiation with 4,000 rads to the spine in January 1973. He also received three courses of intrathecally administered methotrexate. In 1980 the patient went to another institution because of a single episode of transient disorientation without localizing signs. A thorough medical evaluation was performed. Results of a cytologic examination of the cerebrospinal fluid, a general physical examination, and brain computed tomographic scans were unremarkable. Ocular findings included persistent 2+ vitreous cells in the left eye. Results of a cytologic examination of fluid obtained by an anterior chamber paracentesis were consistent with residual ocular reticulum cell sarcoma. At present the patient is alive and well; medication is limited to topical corticosteroids for his ocular disease.

Discussion

Ocular reticulum cell sarcoma is an unusual tumor that simulates diffuse uveitis; yellow-white chorioretinal infiltrates with vitreous cellular opacities are characteristic.[1,2] Approximately 50 cases have been reported. For patients aged 50 years or older who suffer from bilateral diffuse uveitis, ocular reticulum cell sarcoma should be considered in the differential diagnosis.[5-7]

Ocular involvement may initially be unilateral; eventually 80% of patients develop bilateral disease.[2] Usually the ocular lesions are either nonresponsive or exacerbated by the use of corticosteroids or cytotoxic agents.[8,9]

Approximately 75% of patients develop central nervous system involvement before or after the discovery of the ocular lesions.[1,2,5] In ten of 12 consecutive patients examined here, central nervous system involvement was documented (unpublished data). Many patients, including our Patients 2 and 3, have concomitant neurologic symptoms; some, including our Patient 1, have central nervous system disease without symptoms or positive findings on physical examination.

Recurrent or progressive central nervous system disease is the most common cause of death in ocular reticulum cell sarcoma. We previously reported that seven of nine patients died of central nervous system disease, and that the average interval between diagnosis and death was 12 months.[2] Only 25% of patients develop lymphoma outside the eye or central nervous system.[2] In six of eight patients we previously described, there was an improvement in visual acuity after treatment with 4,000 rads of whole-brain and eye irradiation.[2] At the present time we routinely irradiate both eyes of patients with reticulum cell sarcomas because there is a high incidence of bilaterality. Since most patients develop central nervous system disease, which is the ultimate cause of death, we treat the central nervous system with 4,500 to 5,000 rads of whole-brain irradiation followed by a 500- to 1,000-rad boost to the spinal cord.[2]

Radiation has been the standard treatment for both systemic and ocular histiocytic lymphoma (reticulum cell sarcoma).[10] Promising results have been obtained with combination drug chemotherapy in nonocular histiocytic lymphomas.[3,11] In central nervous system histiocytic lymphoma, the relative efficacy of chemotherapy, irradiation, or a combination of both is still unknown.[3] Possibly, the combination of intrathecal chemotherapy and irradiation improves the dismal prognosis which has been associated with ocular reticulum cell sarcoma. It is significant that the only long-term survivors we have treated during the last four years have received both chemotherapy and irradiation.

In these three cases, the combination of chemotherapy and irradiation significantly prolonged survival compared to patients treated previously with irradiation alone. These three patients are alive, on the average, 36 months after therapy and approximately 40 months after the initial diagnosis.

Our management of ocular reticulum cell sarcoma is changing. In older pa-

tients with diffuse uveitis, yellow chorioretinal infiltrates, and vitreous cells, we perform a vitreous biopsy with either a 20-gauge blunt needle or a vitrectomy instrument, using suction only. The biopsy material is examined with both standard cytologic and immunohistologic techniques; malignant cells are usually large, pleomorphic, with scant cytoplasm and eccentric nuclei; immunohistologic evaluation demonstrates monoclonality.[4,12] Each patient undergoes a complete central nervous system evaluation, including physical examination, computed tomography, and cytologic evaluation of the cerebrospinal fluid. Although systemic involvement is uncommon in patients with ocular reticulum cell sarcoma, we routinely perform a systemic evaluation, including bilateral bone marrow biopsies, lymphangiographic studies, and body computed tomographic scans.

We routinely administer bilateral ocular and whole-brain irradiation. If central nervous system disease is demonstrated, we add intrathecal chemotherapy. The best agent for treating ocular reticulum cell sarcoma is still undetermined. Although we did not observe significant side effects with chemotherapy, we would expect some when more patients have been treated.[3,13]

Similarly, the role of vitrectomy in the management of this condition is unclear. In Case 1 we performed a vitrectomy because the patient had failed to improve after an initial course of radiation, and we were uncertain whether an adequate dosage of chemotherapeutic agents would enter the vitreous.[14]

Ocular reticulum cell sarcoma is a rare tumor. Data from prospective randomized treatment trials are lacking. However, the results in these three patients, who were treated with a combination of chemotherapy and irradiation, suggested that combination therapy increases survival in this cancer.

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CONTACT LENSES IN THE MANAGEMENT OF MYOPIC ANISOMETROPIC AMBLYOPIA

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We treated 16 patients aged 1½ to 11 years with myopic anisometropic amblyopia with contact-lens correction of refractive error and occlusion. The degree of visual improvement compared favorably with that reported with the use of spectacles.

There were no complications from contact lenses, and only one patient required anesthesia for fitting. Contact lenses appear to be more satisfactory than spectacle lenses in the management of myopic anisometropic amblyopia in regard to cosmesis, comfort, and treatment compliance.

Patients with myelinated retinal nerve fibers and unilateral severe myopia had a poor visual prognosis. Treatment should not be abandoned in these children, but prolonged occlusion is not indicated.

The presence of strabismus at the start of treatment appeared to have little effect on the final visual results. Most patients with strabismus responded well to occlusion, and all such children should be considered candidates for treatment of their amblyopia.

Amblyopia secondary to unilateral severe myopia has had such a poor prognosis with both pleoptic therapy[1] and occlusion therapy[2] that many ophthalmologists have been discouraged from initiating treatment. Improvement in visual acuity to 6/12 (20/40) or better has been reported with pleoptic therapy in three of 21 patients[3] (14%) and in four of 16 patients (25%).[2] Results with spectacle correction of refractive errors and occlusion are only slightly more encouraging, with an improvement in visual acuity to 6/12 (20/40) or better shown in 16%,[4] 24%,[5] and 55% of patients.[2] The best result reported so far has been improvement in 12 of 15 patients (80%) who achieved final visual acuities of 6/12 (20/40) or better.[6] However, this study excluded patients with obvious abnormalities such as glaucoma, retinopathy of prematurity, degenerative myopia, and myelinated retinal nerve fibers.

These studies, for the most part, used spectacles to correct the refractive error, although contact lenses were used in a few unspecified patients. It is not possible to compare the results of treatment with contact lenses and spectacles in these studies, but it is interesting that none of the authors implied that one form of correction had an advantage over the other. The effectiveness of contact lenses with occlusion for anisometropic myopic amblyopia is unknown. Although Lebensohn[7] advocated contact lenses, others discouraged their use because of Knapp's rule that contact-lens correction induces a greater difference in image size than spectacle correction in anisometropia

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with axial myopia.[8] However, a study of aniseikonia in these patients with the phase-difference haploscope found that some individuals with anisometropic myopia had more aniseikonia with spectacle correction than with contact-lens correction.[9] These authors encouraged contact-lens correction when a reasonable stereo-acuity could not be obtained with spectacles.[9]

In theory, therefore, it appears that contact-lens correction of anisometropia produces a greater aniseikonia than spectacles in axial anisometropic myopia. Experiments, however, seem to indicate that spectacles themselves produce a greater degree of aniseikonia than contact lenses. However, this conflict, although influencing binocularity in these patients, has no bearing on the treatment of the amblyopia itself. We investigated contact-lens correction of refractive error with occlusion in the treatment of anisometropic myopic amblyopia.

Subjects and methods

We reviewed the records of patients examined during a three-year period and selected 16 who met the following criteria: (1) All patients had anisometropic myopia with amblyopia, with the amblyopic eye having a spherical equivalent of at least −5.00 diopters and a difference in the spherical equivalents between the eyes of 5.00 diopters. (2) All used contact-lens correction of the refractive error of the more myopic eye. (3) All were old enough at the completion of the study for a numerical visual acuity on Snellen alphabet letters or the isolated E chart to be measured. Patients who were too young at the beginning of the study for their numerical visual acuities to be measured were tested with Allen picture cards. To compare visual acuities, we mathematically converted the values obtained with the Allen picture cards to the Snellen fraction notation (Allen picture cards at 20 feet were considered to equal 6/9 [20/30]).

We determined refractive errors primarily by cyclogyl cycloplegia. Contact lenses were fitted while the patient was awake, in all except for Patient 7 who required insufflation anesthesia. Patient 8 used cellulose acetatebutyrate lenses; all others used polymethylmethacrylate lenses. The more myopic eye was fitted with a contact lens that overcorrected the myopia by −0.25 diopter. Black contact lenses were used exclusively for occlusion in seven patients and adhesive patches in the other nine patients. The contact lenses were worn all day in the amblyopic eye. Full-time occlusion was used for preschool patients. In school-aged patients, occlusion was used after school hours.

Results

The age of the patients at first examination ranged from 1½ to 11 years with a mean age of 5 years (Table). There were ten girls and six boys. The spherical equivalent of the more myopic eye ranged from −5.375 to −16.50 diopters with a mean of −10.00 diopters. The difference in the spherical equivalents between the two eyes ranged from 5.25 to 16.375 diopters with a mean of 10.25 diopters. Eight of the 16 patients had strabismus. Three had esotropia and five exotropia. The presence of strabismus did not significantly affect the visual results of the amblyopic treatment.

The 16 patients fall into three well-defined groups: Group 1, Patients 1 through 10 who achieved final visual acuities of 6/12 (20/40) or better; Group 2, Patients 11 to 13 who failed to achieve 6/12 (20/40) but improved two or more Snellen lines; and Group 3, Patients 14 through 16 who showed little if any improvement (Figure). Within each of these three groups, the improvement in visual acuity with occlusion was signifi-

TABLE

PRETREATMENT AND POSTTREATMENT FINDINGS IN 16 PATIENTS

Patient	Age (yrs)	Refraction	Spherical Equivalent: In Severely Myopic Eye	Spherical Equivalent: Difference Between Eyes
1	6	R.E.: − 7.25 +3.75 × 20 L.E.: − 1.00 +1.75 × 160	− 5.375	5.25
2	6	R.E.: −13.00 +2.50 × 20 L.E.: − 1.00 +1.00 × 20	−11.75	11.25
3	8	R.E.: −12.50 +3.00 × 160 L.E.: + 1.25	−11.00	12.25
4	5	R.E.: −10.00 +2.00 × 135 L.E.: + 1.00	− 9.00	10.00
5	4	R.E.: − 6.00 +0.50 × 180 L.E.: + 2.25 +0.25 × 180	− 5.75	8.125
6	5	R.E.: + 1.50 L.E.: −10.00	−10.00	11.50
7	2	R.E.: − 7.00 L.E.: Plano	− 7.00	7.00
8	6	R.E.: − 2.25 +1.00 × 50 L.E.: − 8.50 +1.25 × 100	− 7.875	6.125
9	2	R.E.: − 1.50 L.E.: −11.50	−11.50	10.00
10	2	R.E.: −11.00 +3.00 × 170 L.E.: − 4.00 +1.75 × 170	− 9.50	6.375
11	11	R.E.: − 0.50 +0.75 × 90 L.E.: −16.50	−16.50	16.375
12	6	R.E.: −13.50 L.E.: Plano	−13.50	13.50
13	6	R.E.: −13.75 +1.50 × 180 L.E.: − 3.50 +0.75 × 180	−13.00	9.875
14	3	R.E.: −10.50 +3.00 × 125 L.E.: + 1.00 +1.00 × 90	− 9.00	10.50
15	4	R.E.: + 1.75 L.E.: −12.50 +1.25 × 180	−11.875	13.625
16	3	R.E.: −11.00 +2.00 × 90 L.E.: + 1.50 +0.50 × 90	−10.00	11.75

*By isolated Snellen letters.

†By whole-line Snellen letters.

‡By isolated E.

§By Allen cards mathematically converted to Snellen notation (cards at 20 feet = 6/9 [20/30]).

cant only in Group 1 ($P = .008$ by Student's t-test). However, if the results for Groups 1 and 2 are totaled, the improvement in visual acuity is still significant ($P = .025$ by Student's t-test).

The three patients in Group 3 (Patients 14 to 16) differed significantly from the other 13 patients in that they had myelinated retinal nerve fibers. None of the other 13 patients had any observable retinal abnormalities that could account for a significant decrease in visual acuity. The myelination extended vertically from the optic disk superiorly or inferiorly, or both, for at least 4 disk diameters, but did not involve the macular area or the area of the papulomacular bundle. This was the only finding other than visual response that distinguished these patients from the others in the study.

TABLE (continued)

PRETREATMENT AND POSTTREATMENT FINDINGS IN 16 PATIENTS

Visual Acuity		Occlusion (mos)	Strabismus	Final Stereopsis (sec)
Initial	Final			
6/120 (20/400)*	6/7.5 (20/25)*	5	—	3,000
6/60 (20/200)*	6/7.5 (20/25)†	9	—	3,000
6/60 (20/200)*	6/9 (20/30)†	9	Intermittent exotropia	40
6/60 (20/200)*	6/9 (20/30)†	9	—	3,000
6/30 (20/100)‡	6/9 (20/30)*	11	Constant esotropia	0
6/24 (20/80)†	6/6 (20/20)†	17	—	100
6/24 (20/80)§	6/9 (20/30)*	10	Constant exotropia	0
6/22 (20/70)†	6/9 (20/30)†	4	Constant exotropia	0
6/18 (20/60)§	6/9 (20/30)*	5	—	140
6/18 (20/60)§	6/9 (20/30)‡	11	Constant esotropia	0
C.F. —	6/60 (20/200)*	6	Constant exotropia	0
6/120 (20/400)*	6/24 (20/80)†	8	—	0
6/240 (20/800)*	6/60 (20/200)*	8	Constant esotropia	0
C.F. —	6/60 (20/200)‡	4	Constant exotropia	0
6/60 (20/200)*	6/30 (20/100)†	12	—	0
6/30 (20/100)‡	6/30 (20/100)*	14	—	3,000

The three patients in Group 2 had the greatest myopia in the amblyopic eye, ranging from −13.00 to −16.50 diopters, and they began treatment with the poorest visual acuity. Patient 11, the oldest patient in the study, was 11 years old at the time of initial treatment. All of the other 15 patients in the study began treatment while less than 8 years old. Patient 12 had a posterior staphyloma, although the macula appeared to be normal; Patient 13 was lost to follow-up after eight months.

The ten patients in Group 1 were not otherwise distinguishable from the other six patients. The presence of strabismus in five of these patients apparently did not affect their final visual results.

The fitting and wearing of contact lenses in these children did not present a problem, and anesthesia was required for the initial fitting in only one patient. All 16 patients achieved six to eight hours of continuous wear within the first ten days after receiving their contact lenses. There were no significant complications with respect to wearing these contact lenses, and no patient left the study

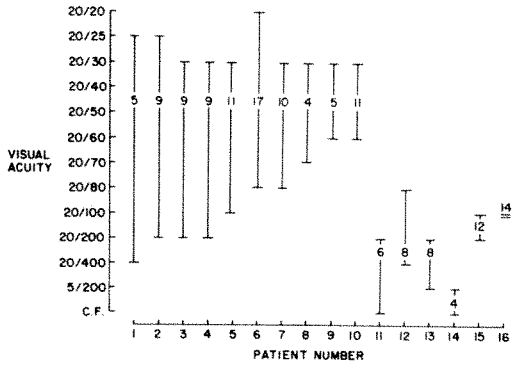


Figure (Mets and Price). Visual results of the 16 patients shown in order of decreasing response to occlusion. The lower bar indicates the preocclusion visual acuity and the upper bar indicates the final visual acuity. The months of occlusion necessary to achieve this final visual acuity are shown within each bar.

because of inability to wear contact lenses.

Discussion

The results of this study, which used contact lenses in the management of amblyopia secondary to unilateral severe myopia, compared favorably with those of previous studies, which primarily employed spectacle correction. Only one study[6] had results comparable to ours, and that study excluded all patients with obvious abnormalities, including myelinated retinal nerve fibers.

There are a number of possible explanations for these results. After successfully adapting to contact lenses, the wearer is generally unaware of their presence, whereas spectacles, particularly heavy high-minus spectacles, are constantly annoying because of their weight. The spectacles used in anisometropia also tilt annoyingly. For example, if Patient 12 had been fitted with standard 44-mm round plastic spectacle lenses, the −13.50 right lens would have weighed 12 g, whereas the plano left lens would have weighed only 5 g, a 7-g tilt factor.

Additionally, spectacles are easily removed and left behind, whereas children in the age group in this study are seldom able to remove their contact lenses. The optical correction of the severely myopic eye is, therefore, in place more of the time, as is the black contact lens used for occlusion.

Another advantage of high-minus contact lenses over spectacle lenses is the lack of peripheral distortion, resulting in more comfortable vision. The improvement in visual acuity in myopic anisometropic amblyopia is directly proportional to the time that occlusion and refractive correction are in effect. Contact lenses appear to accomplish this better than spectacle lenses do.

The three patients in this study with myelinated retinal nerve fibers were the only patients to show no visual improvement. A previous report of four cases of ipsilateral myopia, amblyopia, strabismus, and myelinated retinal nerve fibers,[8] suggested that the symptoms of these patients form a syndrome. One of the four patients mentioned in that report was treated with spectacle correction of refractive error and occlusion and failed to respond. The three patients in the present study with myelinated retinal nerve fibers had no other unfavorable findings when compared with the other 13 patients. They were among the youngest in the series treated, and two had initial visual acuities better than those in Group 2, but these three were the only patients who did not respond to therapy.

Eight patients in this study had strabismus in addition to anisometropia; three had esotropia; and five had exotropia. Two of the patients with esotropia and three with exotropia improved to 6/12 (20/40) or better and one with esotropia and one with exotropia improved at least two Snellen lines. Only the one patient, who had strabismus and myelinated retinal nerve fibers, failed to improve. It seems that strabismus had no unfavorable effect on visual improvement in these patients.

The use of contact lenses in anisometropic myopia has previously been discouraged because of the theoretical aniseikonia induced and its effect on binocularity.[8] In our Group 1, five of ten patients had orthotropia and one had intermittent exotropia, and yet all six achieved some degree of stereopsis after treatment. All wore at least one contact lens and those whose nonamblyopic eye required refractive correction wore two. Their stereopsis varied from 40 to 3,000 seconds on the Titmus stereotest. None of the four patients with constant strabismus achieved any binocularity. It would appear, therefore, that contact lenses in anisometropic myopia do not prevent binocularity.

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CYCLOPLEGIC REFRACTIONS OF PREMATURE INFANTS

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Cyclopentolate refractions of 146 premature infants disclosed higher incidences of myopia and anisometropia than those reported for full-term infants. Also, more than 70% of the premature infants had 1 diopter or more of astigmatism; 83% of the astigmatism was against-the-rule. Infants with shorter gestational periods had more severe myopia and astigmatism. Infants born very prematurely should undergo follow-up refractions to determine whether they retain refractive errors large enough to interfere with visual function.

Infants born prematurely are at risk for visual problems. These problems include the visual consequences of the conditions that caused prematurity (for example, congenital infections and developmental anomalies), retrolental fibroplasia caused by oxygen therapy, a higher-than-normal incidence of strabismus and poor visual acuity in later years,[1] and the myopia of prematurity that is present in early infancy[2-8] and that may or may not continue into childhood.[1,6-13] The degree to which premature infants are at risk for astigmatism[4,5,13] and anisometropia,[4,13] two treatable factors known to influence visual performance, has not been thoroughly investigated.

Our purpose was to examine the spherical and cylindrical components of refractive errors in a group of premature infants of known gestational ages, and to evaluate the relationship between gestational age and refractive error.

SUBJECTS AND METHODS

Infants at two Boston hospitals who are at risk for retrolental fibroplasia undergo thorough ophthalmic examinations, including cycloplegic refractions by a staff ophthalmologist. Between Oct. 18, 1978, and March 14, 1980, 233 such infants were examined. Of these, we included in

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this study 146 infants who met the following criteria: (1) gestational age at birth, as determined by the attending neonatologist, ≤ 36 weeks; (2) age at the time of first refraction ≤ 8 weeks; and (3) no ocular abnormalities found at a complete ophthalmic examination. At the time the infants underwent refraction, their mean age from onset of the mother's last menstrual period was 34.2 weeks (S.D. = 2.4 weeks) and their mean postnatal age was 2.8 weeks (S.D. = 2.9 weeks). The mean gestational age of the infants at birth was 31.4 weeks (S.D. = 2.9 weeks). We excluded 42 infants from the study group because their gestational ages were more than 36 weeks, five infants because they had not undergone refraction during the ocular examination, and 24 infants because ophthalmic examinations disclosed retrolental fibroplasia in 19 infants, extensive retinal or conjunctival hemorrhages in two infants, and coloboma of the macula, severe conjunctivitis, and hypertensive retinopathy in one infant each. Also, we excluded 16 infants who were older than 8 weeks at the time of first refraction from the data analysis because the ages of these infants ranged so widely (9 to 38 postnatal weeks) that the data were insufficient to allow a meaningful analysis of the results.

The cycloplegic agent we used for the refraction was cyclopentolate 1%; one drop was given 30 to 45 minutes before retinoscopy. Retinoscopies were performed with a Copeland streak retinoscope in a dimly lighted room. We evaluated retinoscopic reflexes while the infant was fixating the light of the retinoscope. One refractionist (R. A. P.) performed all but seven of the retinoscopies; previous retinoscopies performed by this refractionist differed on the average by less than 0.5 diopter from those of a second skilled refractionist.[14] All retinoscopies were done with the minus cylinder convention.

Computer-aided analyses were carried out on data coded for power of spherical and cylindrical components for each eye, axis of cylindrical component for each eye, gestational age at birth, and postnatal age at the time of retinoscopy. We calculated the spherical equivalent for each eye, and determined right-left eye differences in spherical equivalents for each infant. We classified the axis of any cylindrical component as with-the-rule if the minus cylinder axis was at 180 ± 15 degrees, against-the-rule for minus cylinder at 90 ± 15 degrees, or oblique (other than with-the-rule or against-the-rule).

RESULTS

Spherical equivalents—The mean spherical equivalents for the right and left eyes of the 146 premature infants undergoing refraction in this study were −0.69 diopter (S.D. = 2.8 diopters) and −0.41 diopter (S.D. = 2.8 diopters), respectively. These values were similar to those reported in previous studies of premature infants.[2,4-6,8,13,15]

The distribution of spherical equivalents for the right and left eyes were not different ($\chi^2 = 14.1$; df = 15; $P > .05$) (Fig. 1). However, the distribution of spherical refractive errors for 280 full-term infants (560 eyes) who underwent refraction during the first 24 hours after birth[6] was different ($\chi^2 = 325.9$; df = 18; $P < .001$) (Fig. 1). Premature infants undergoing refraction before completion of a normal 40-week gestational period tend to be more myopic than newborn full-term infants. The data for full-term infants was similar to the findings of other investigators for full-term newborns.[15]

A linear regression indicated that the gestational age of the infant at birth was significantly correlated with the infant's refractive error; infants who were born more prematurely tended to be more myopic than infants who were born less prematurely ($r = .30$; $P < .001$). This

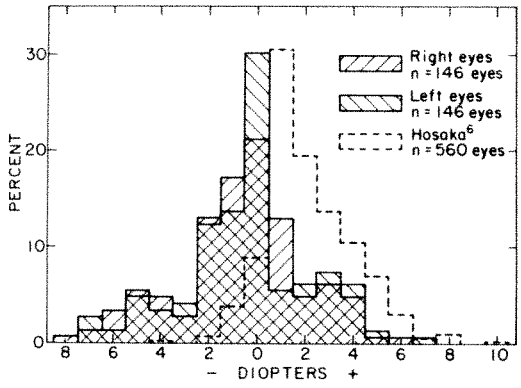


Fig. 1 (Dobson and associates). Distributions of spherical equivalents in diopters for 146 premature infants, plotted with the distribution of spherical refractive errors in diopters reported by Hosaka[6] for 280 full-term newborn infants. (The spherical equivalent equals the algebraic sum of the spherical power plus one half of the cylindrical power).

result cannot be attributed to the fact that infants in the present sample underwent refraction at different ages; we found the same effect when we performed linear regressions on the data for groups of infants undergoing refraction at the same number of weeks after the last maternal menstrual period (Table).

Figure 2 plots the differences in spherical equivalents between fellow eyes for the premature infants in the present study, along with previously published data for differences between vertical meridians of the fellow eyes of 150 premature infants[4] and our recently published data for differences in spherical equivalents between fellow eyes of 124 full-term infants undergoing refraction in the first postnatal year.[16] The distributions for the two groups of premature infants were quite similar ($\chi^2 = 2.39$; df = 5; $P > .05$). Conversely, the full-term infants, tested at somewhat older ages, showed significantly less anisometropia (≥ 1 diopter difference between eyes) than did the premature infants ($\chi^2 = 42.7$; df = 4; $P < .001$). Further, only 20.5% of the infants in the present study showed a difference of less than 0.25 diopter in spherical equivalents between eyes, whereas 91.9% of the full-term infants in the previous study showed a difference of less than 0.25 diopter between eyes.

Astigmatism—The mean cylindrical components of the right and left eye refractions were 1.9 diopters (S.D. = 1.8 diopters) and 1.4 diopters (S.D. = 1.3 diopters), respectively. These values were considerably larger than the values found in the three other studies[4,5,13] that reported astigmatic error in premature infants. Furthermore, 71.3% of right eyes and 65.8% of left eyes examined in the present study showed at least 1 diopter of astigmatism (Fig. 3), whereas Hosaka[5] found astigmatism of at least 1 diopter in

TABLE

CORRELATIONS BETWEEN REFRACTIVE ERROR AND GESTATIONAL AGE

Time Between Last Maternal Menstrual Period and Refraction (wk)*	No. of Infants	Spherical Equivalent (Pearson's r)	Cylinder (Pearson's r)
36.1 – 38	22	.56†	−.47‡
34.1 – 36	50	.44†	−.43†
32.1 – 34	37	.21	−.19
30.1 – 32	20	.19	−.03

*Only groups with ten or more infants are included.
†$P < .001$.
‡$P < .05$.

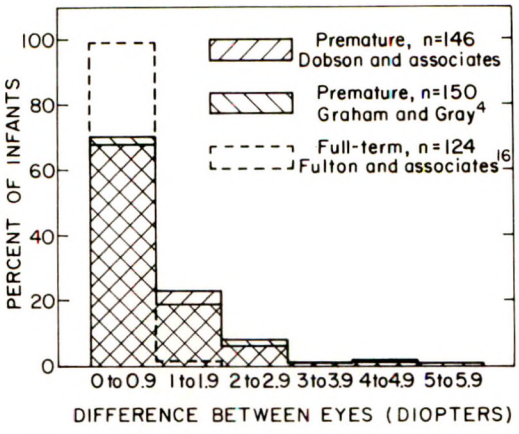


Fig. 2 (Dobson and associates). Differences in spherical equivalents in diopters between right and left eyes of the 146 premature infants in this study, plotted with the distribution of differences in diopters of the refractive errors of the vertical meridians of the right and left eyes reported by Graham and Gray[4] for 150 premature infants and the distribution of differences in spherical equivalents in diopters between eyes found by Fulton and associates[16] for 124 full-term infants between 0 and 50 weeks of age. The distributions from the two studies of premature infants are very similar and show a higher incidence of anisometropia than was found in the study of full-term infants.

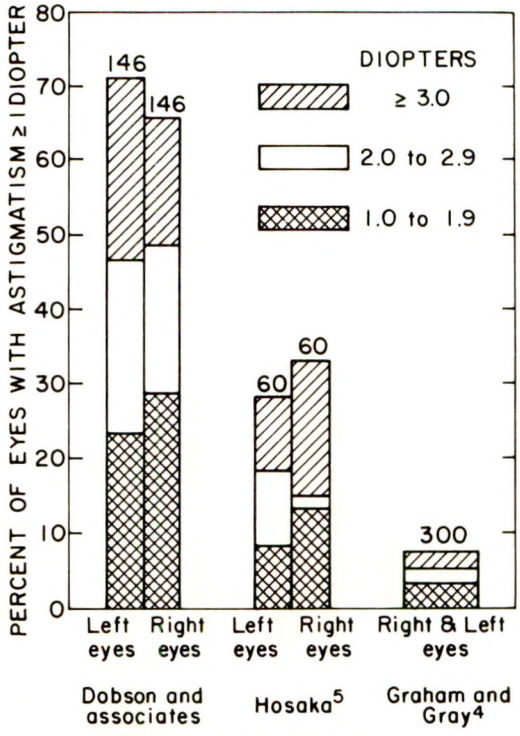


Fig. 3 (Dobson and associates). Percentage of premature infants who showed significant (≥1 diopter) astigmatism. Results of the present study and the two previous studies that examined astigmatism in premature infants are plotted. Total number of eyes refracted is indicated at the top of each bar.

31% of premature infants and Graham and Gray[4] reported astigmatism of at least 1 diopter in only 8% of their sample.

A linear regression indicated that the cylindrical component of an infant's refractive error was correlated with the infant's gestational age at birth; infants born after the shortest gestational periods tended to have more astigmatism than infants born after somewhat longer gestational periods ($r = .24$; $P < .01$). We obtained the same result when analyzing data for groups of infants undergoing refraction at the same number of weeks after the last maternal menstrual period (Table).

Figure 4 shows the axis of cylindrical error for the 101 infants who had 1 diopter or more of astigmatism in the right eye. These results indicated that 83% of the astigmatic infants had against-

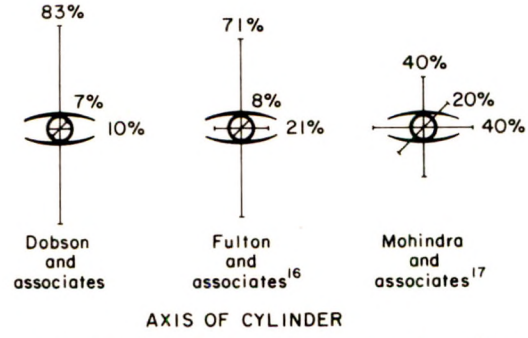


Fig. 4 (Dobson and associates). Axis of significant (≥1 diopter) cylindrical error in minus cylinder convention for the 101 astigmatic infants in this study, 63 full-term astigmatic infants and children in the study by Fulton and associates,[16] and 62 11- to 20-week-old astigmatic full-term infants studied by Mohindra and associates.[17] Data shown are for right eyes only. In this study, 83% of infants with significant astigmatism had against-the-rule, 10% had with-the-rule, and 7% had oblique astigmatism.

the-rule astigmatism. Only 10% had with-the-rule and 7% had oblique astigmatism. Figure 4 also shows the results of two studies of full-term infants who underwent refraction in the first postnatal year. The cycloplegic retinoscopies of Fulton and associates[16] showed that there was also a high incidence (70%) of against-the-rule astigmatism among full-term infants and young children who had at least 1 diopter of astigmatism, whereas the result of noncycloplegic refractions by Mohindra and associates[17] showed a more equal distribution of with-the-rule and against-the-rule astigmatism.

Discussion

Our cycloplegic refractions of 146 premature infants showed that these infants had a higher incidence of myopia and anisometropia (a difference of at least 1 diopter in spherical equivalents between eyes) than has been reported for newborn full-term infants. Additionally, the premature infants showed a high incidence of astigmatism of 1 diopter or more, similar to that reported in two recent studies[17,18] of full-term infants who underwent refraction during the first postnatal year. Finally, analysis of refractive error as a function of the infants' gestational ages at birth indicated that infants born more prematurely tended to be more myopic and more astigmatic than infants born less prematurely.

The distribution of spherical equivalents for infants in this study was remarkably similar to the results of other studies of premature infants undergoing refraction during the first two postnatal months. The results indicated that very young premature infants have more myopia than do full-term newborn infants. Whether this tendency of premature infants to be myopic remains past early infancy has not yet been resolved. Although a number of studies have found a higher incidence of myopia in children who were born prematurely than in children who were born at term,[1,10,12] other studies[7,13] have shown that by the time the infants are 6 months old, the mean refractive errors for premature and full-term infants do not differ. Unfortunately, most of the infants we studied were lost to further follow-up, because they were from homes located quite far from Boston.

The high incidence (71% for right eyes and 66% for left eyes) and the severity of astigmatism (1.9 diopters for right eyes and 1.4 diopters for left eyes) seen in the present sample of premature infants were greater than those seen in previous studies.[4,5,13] Differences in the samples of premature infants included in each study or in techniques of refraction may explain these discrepant results. However, a surprisingly high incidence of astigmatism has also been found in full-term infants.[16-19] The cause of the high incidence of astigmatism in infants has yet to be discovered. Banks[15] suggested that it is the result of the disparity between the optical and visual axes of infants' eyes, a hypothesis that predicts against-the-rule astigmatism. Eighty-three percent of the astigmatic infants in our sample showed against-the-rule astigmatism. Only follow-up refractions and visual acuity tests can determine the effects of such severe astigmatism on the visual development of premature infants.

The incidence of anisometropia found in our sample of premature infants (32% showed a difference of at least 1 diopter in spherical equivalents between eyes) was similar to that previously reported for a group of premature infants,[4] and was much greater than that reported for full-term infants undergoing refraction during the first postnatal year.[16] This suggested that premature infants may be at risk for the development of amblyopia. Only follow-up refractions can determine whether the anisometropia remains and

interferes with the infants' visual function.

Our study was the first to examine the correlation between refractive error and gestational age at birth. The results indicated that premature infants with lower gestational ages tend to have more myopia and more severe astigmatism than do premature infants born after longer gestational periods. These results were consistent with previous findings that infants with many signs of prematurity tend to show a high incidence of myopia.[5]

In conclusion, the results indicated that premature infants who do not have retrolental fibroplasia are nevertheless at risk for severe refractive errors. These infants have higher incidences of myopia and anisometropia than those reported for full-term infants. Additionally, the premature infants have a high incidence and a severe degree of astigmatism. Previous studies suggested that myopia in premature infants and astigmatism in full-term infants decrease with age.[6-9,11,13,16-19] However, age-related changes in anisometropia and severe astigmatism have yet to be established for premature infants. If the astigmatism and anisometropia found in this sample persist into childhood, visual function will be impaired. Therefore, premature infants should have ophthalmic follow-up examinations.

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PRIMARY HERPES SIMPLEX SUBEPITHELIAL DENDRITIC KERATITIS

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A 37-year-old man developed an acute follicular conjunctivitis with preauricular lymphadenopathy believed to be epidemic keratoconjunctivitis. On the eighth day of his disease, subepithelial dendritic opacities developed in the cornea which were not typical of either epidemic keratoconjunctivitis or herpetic keratitis. A diagnosis of primary herpes simplex virus infection was established by positive viral culture and a rise in serum antibody titer to herpes simplex virus. Subepithelial dendritic keratitis as a manifestation of herpes simplex infection of the cornea has not been previously described. The lesions seen in this patient were not reproducible in rabbits and we believe they represent an unusual host response to the virus. This form of herpetic keratoconjunctivitis is extremely difficult to differentiate from epidemic keratoconjunctivitis. Corticosteroids should be used with caution in cases that are not completely typical of epidemic keratoconjunctivitis.

Acute follicular conjunctivitis can be caused by a number of organisms, including adenoviruses, herpes simplex virus, Newcastle disease virus, and *Chlamydia trachomatis*. The most important differentiation to be made among these diseases is between epidemic keratoconjunctivitis, caused by adenovirus types 8 and 19,[1] and primary herpes simplex keratoconjunctivitis. This distinction can usually be made clinically on the basis of the characteristic corneal lesions that occur in these syndromes.

We studied a patient, believed to have epidemic keratoconjunctivitis, who developed an acute follicular conjunctivitis with preauricular lymphadenopathy. Eight days after the onset of symptoms, subepithelial infiltrates developed which were large and dendritic in shape and not characteristic of either epidemic keratoconjunctivitis or herpes simplex keratitis. Viral cultures and a rise in antibody titer to herpes simplex virus established a diagnosis of primary herpes simplex virus infection. Subepithelial dendritic lesions are an unusual manifestation of herpes simplex virus infection and, to our knowledge, have not been previously described.

CASE REPORT

A 37-year-old man had a two-day history of redness, irritation, and a watery discharge in the right eye. There was no recent exposure to eye infections, and no history of gingivostomatitis or other manifestation of herpetic infection.

An ocular examination disclosed a best corrected visual acuity of 6/6 (20/20) in both eyes. A tender preauricular lymph node was present on the right side. The eyelids on the right were mildly swollen,

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but there were no vesicular lesions. Severe follicular conjunctivitis and a mild punctate epithelial keratitis were present on the right side. The left eye was normal. A diagnosis of probable epidemic keratoconjunctivitis was made and the patient was treated prophylactically with topical 10% sulfacetamide solution. He was reexamined at three-day intervals.

Six days later (day 8 of his disease) he returned because of an increasing foreign-body sensation and photophobia in the right eye. At that time, several subepithelial infiltrates were seen centrally in the right eye. However, these lesions were atypical for epidemic keratoconjunctivitis in that they were large in size and assumed ameboid and branching, serpiginous shapes, most easily observed by retroillumination (Figs. 1 and 2). There were no epithelial erosions or dendritic epithelial ulcers at this, or any other, time. The lesions did not stain with fluorescein. A vesicular lesion also developed at the margin of the right upper eyelid (Fig. 1). The left eye remained normal. Because herpes simplex virus infection was suspected, specimens were collected from the right eye for viral culture and serum was collected for viral titers. The patient was treated with topically applied 1% trifluridine solution every four hours. Within one week, the conjunctivitis and subepithelial lesions resolved completely.

One month later, serum was again collected for convalescent viral titers. The right eye showed no sign of recurrence.

Material and methods

Viral cultures—Specimens taken from the right eye were inoculated onto a monolayer culture of primary human foreskin cells grown in RPMI 1640 medium supplemented with 10% fetal calf serum, 1% glutamine, gentamicin, 50 μg/ml, and amphotericin B, 5 μg/ml. The cultures were examined daily for cytopathic effect. After a cytopathic effect was observed, the virus was harvested and identified by neutralization with virus-specific antibody produced in rabbits.

Serum titers—We determined neutralizing antibody titers by plaque reduction assay of acute and convalescent sera against the patient's isolate.

Animal studies—We inoculated both eyes of two 3-kg white New Zealand rabbits by instilling 0.05 ml of the patient's virus containing 1×10^6 plaque-forming units onto the corneas. The eyelids were then closed and the eyes massaged for 20 seconds. We examined the animals at two-day intervals with a slit-lamp biomicroscope and fluorescein staining and observed them for conjunctivitis, epithelial and stromal keratitis, corneal vascularization, and iritis.

Results

Viral cultures—Within 48 hours, we observed the cytopathic effect characteristic of herpes simplex virus. The virus was identified as herpes simplex virus type 1.

Serum titers—We found that the acute serum had a neutralizing antibody titer against type 1 herpes simplex virus of 1:8. The convalescent serum showed a four-

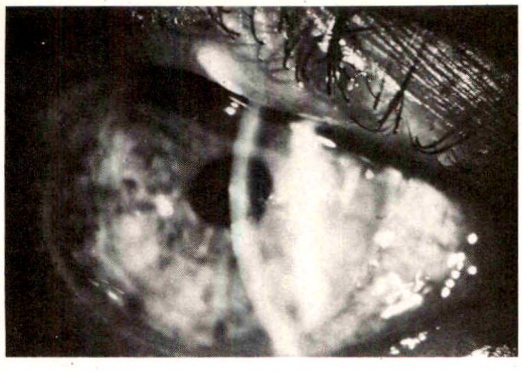


Fig. 1 (Stern, Zam, and Gutgesell). Right eye, day 8. Slit-lamp photograph shows the subepithelial location of corneal opacities. A vesicular lesion is present at the upper eyelid margin.



Fig. 2 (Stern, Zam, and Gutgesell). Right eye, day 8. Retroillumination photograph of right cornea shows the ameboid and dendritic shape of the lesions.

fold rise in titer to 1:32, consistent with a primary herpes simplex infection.

Animal studies—Three days after inoculation, all eyes had developed a conjunctivitis and either punctate epithelial erosions or dendritic ulcers. During the next week the corneal lesions enlarged to form geographic ulcers in all eyes. Two eyes developed stromal keratitis and iritis, leading to corneal vascularization and scarring. The other two eyes cleared with minimal vascularization. One rabbit developed encephalitis. These findings were typical of herpes simplex type 1 infection in the rabbit.

Discussion

In the absence of characteristic corneal lesions, the clinical differentiation of epidemic keratoconjunctivitis and primary herpes simplex virus infection can be difficult. Knopf and Hierholzer,[2] in a study in which the cause of viral keratoconjunctivitis was established by viral cultures and serum titers, indicated that some cases believed clinically to be epidemic keratoconjunctivitis were caused by herpes simplex virus and that some cases believed to be herpes simplex keratoconjunctivitis were caused by adenovirus.

Our patient developed corneal lesions atypical of both syndromes. In epidemic keratoconjunctivitis, the subepithelial infiltrates develop later in the course of the disease than did those that were observed in our patient,[3] and assume a more uniform, round shape than the pleomorphic, serpiginous lesions seen in this case. Additionally, the subepithelial lesions in epidemic keratoconjunctivitis are not responsive to antiviral agents[4] and disappear slowly during the course of several months. In our patient, these lesions resolved completely in one week after being treated with trifluridine.

In primary herpes simplex keratoconjunctivitis, the characteristic corneal lesion is dendritic epithelial ulceration. These lesions usually occur seven to ten days after the onset of the conjunctivitis, which coincided with the appearance of the corneal lesions in this case.[5] Also, numerous small dendrites are often seen; this corresponded with the pattern seen in our patient. In our patient, however, the lesions were subepithelial opacities rather than epithelial ulcerations. We examined the patient at frequent intervals and at no time did we see any epithelial defects by fluorescein staining. Subepithelial ghost opacities similar to those seen in our patient may occur as a residual effect of herpetic keratitis.[6] However, these opacities only occur in areas of previous dendritic ulceration and may be accentuated by antiviral drugs,[6] in contrast to the response seen in our patient.

That herpes simplex virus type 1 was the causative agent of this infection was established by a positive culture for the virus. The fourfold rise in serum titer to herpes simplex virus is consistent with a primary infection. To our knowledge, subepithelial dendritic keratitis as a manifestation of either primary or recurrent herpes simplex infection in the cornea has not been previously described.

Recent studies[7] have shown that different strains of herpes simplex virus cause different types of keratitis in laboratory animals, leading to speculation that different forms of herpetic keratitis may be the result of a virus-specific effect rather than a variable host response to the organism. We were not able to reproduce the subepithelial lesions in laboratory animals inoculated with the strain of herpes simplex virus isolated from our patient. The disease seen in our laboratory studies was typical of the infection caused by other virulent herpes simplex type 1 strains. We must conclude, there-

fore, that the findings in our patient represented an unusual host response to the virus.

The recognition of this new form of herpes simplex keratitis and its differentiation from epidemic keratoconjunctivitis is important. The corneal lesions of epidemic keratoconjunctivitis do not respond to antiviral drugs, but may respond to topical corticosteroids.[8] Conversely, in herpes simplex keratitis, antiviral therapy is effective and corticosteroids are contraindicated. Ophthalmologists should exercise caution in using corticosteroids for the treatment of viral keratoconjunctivitis because herpes simplex infection may be difficult to differentiate from other viral infections.

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CORNEAL TOXICITY WITH SYSTEMIC CYTARABINE

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Three patients with leukemia developed corneal toxicity while receiving high doses (3 g/m^2) of systemic cytarabine. Symptoms began five to seven days after initiation of treatment with high doses of systemic cytarabine and consisted of ocular pain, tearing, foreign-body sensation, photophobia, and blurred vision. All three patients developed bilateral conjunctival hyperemia and fine corneal epithelial opacities and refractile microcysts that were more numerous in the central than in the peripheral cornea. The symptoms disappeared without treatment in approximately one week. The corneal changes we observed with high doses of systemic cytarabine resembled descriptions of corneal toxicity from topical cytarabine and were probably secondary to inhibition of DNA synthesis in the corneal epithelium.

Topically applied cytarabine has caused corneal toxicity[1,2] and, therefore, it cannot be used in treating herpes simplex keratitis. Cytarabine in high doses (3 g/m^2), however, has been used systemically to treat refractory adult acute nonlymphocytic leukemia.[3,4]

To the best of our knowledge, there have been no reports of corneal toxicity secondary to systemic cytarabine. We recently treated three leukemic patients who developed corneal toxicity while receiving high doses of systemic cytarabine.

CASE REPORTS

Case 1—A 54-year-old woman with acute promyelocytic leukemia underwent various courses of chemotherapy with daunomycin, thioguanine, and cytarabine in standard doses from November 1979 to August 1980.

Although she had no symptoms, the patient was admitted for re-induction chemotherapy in August 1980 because blast cells were found on a bone marrow aspirate. At physical examination, the patient's pupils, conjunctivas, and fundi were normal. The lungs were clear to auscultation, and a 2/6 systolic murmur was heard at the left sternal border that radiated to the apex. Results of abdominal and neurologic examinations were normal. Laboratory tests gave the following values: white blood cell count, 4,200/mm^3 with 66% neutrophils, 5% band cells, 24% lymphocytes, and 5% monocytes; hemoglobin, 11.5 g/100 ml; hematocrit, 33.8%; platelet count, 293,000/mm^3; and reticulocyte count, 2%. Prothrombin time and partial thromboplastin times were within normal limits. Results of chest X-ray film and electrocardiogram were normal.

The patient was given cytarabine (3 g/m^2) intravenously every 12 hours for five days. Two days after admission, a routine ophthalmologic consultation was requested. At that time there were no ocular complaints except for floaters in the left eye. On examination, her corrected visual acuity was 6/7.5 (20/25) in both eyes. Her pupils reacted normally to light and accommodation, and her extraocular motility was full. The conjunctivas, corneas, and anterior chambers were normal in both eyes. Tension by applanation tonometry was 12 mm Hg in each eye. Ophthalmoscopy disclosed normal optic disks and tortuous retinal blood vessels but no hemorrhages or exudates.

After five days of cytarabine therapy, the patient requested another ophthalmologic consultation. She complained of foreign-body sensation, photophobia, tearing, and blurred vision in both eyes that had been present for two days. On examination her corrected visual acuity was 6/60 (20/200) in both eyes.

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Tension by applanation tonometry was 10 mm Hg in each eye. Her pupils reacted normally to light and accommodation, and her extraocular motility was full. Preauricular and submandibular lymph nodes were not palpable. The eyelids and eyelashes were normal. Bilateral intense conjunctival hyperemia was present without prominent follicles or papillae. Both corneas showed fine epithelial opacities and refractile microcysts that were more numerous centrally than peripherally. The corneal epithelium showed mild superficial punctate staining with fluorescein. Folds in Descemet's membrane and mild stromal edema were also noted. The anterior chamber was clear in both eyes. The second ophthalmoscopic examination gave the same results as the first one. Cultures of the eyelids and conjunctivas were taken on blood agar and mannitol plates with a cotton-tipped applicator moistened with trypticase soy broth. Mannitol-negative staphylococci were recovered from the left eyelid. Viral cultures of the conjunctiva for herpes simplex and adenovirus were negative. Giemsa stain of conjunctival scrapings showed many polymorphonuclear leukocytes and moderate amounts of necrotic and degenerating epithelial cells. A biopsy of the corneal epithelium of the left eye was done.

Two days later, her symptoms began to improve. Her corrected visual acuity was R.E.: 6/18 (20/60) and L.E.: 6/30 (20/100). Mild conjunctival hyperemia was still present, and there were fewer corneal epithelial opacities and microcysts. Five days later, the symptoms of foreign-body sensation, tearing, and redness disappeared and her visual acuity improved to 6/12 (20/40) in both eyes. The conjunctival hyperemia resolved, and only a rare epithelial opacity was noted in either cornea.

We fixed the stripped corneal epithelium obtained from this patient in Karnovsky's solution. The tissue was then minced, postfixed in 1% osmium tetroxide, dehydrated in graded alcohols, and embedded in Epon. We cut 1-μ sections for light microscopic study and stained them with toluidine blue. We examined ultrathin sections stained with uranyl acetate and lead citrate with an electron microscope.

Examination by light microscopy showed that the stripped stratified squamous corneal epithelium averaged six to seven cells in depth. The basal layer lacked an orderly arrangement of columnar cells and had been replaced by one to two layers of irregularly shaped degenerating squamous epithelial cells (Fig. 1). These cells, which were present throughout the deep aspect of the specimen, showed loss of cohesiveness, contained pyknotic nuclei, and were intermixed with granular cytoplasmic debris. Isolated degenerated cells were present within the wing cell layer, and there were many small microcysts containing cellular debris.

Electron microscopy disclosed that the basal aspect of the epithelium contained several layers of irregularly shaped, flattened squamous cells (Fig. 2). These had an electron-dense cytoplasm with abundant tonofibrils. There were numerous vacuoles and distended mitochondria with disrupted cristae. The nuclei were shrunken with a convoluted, folded configuration and contained highly condensed heterochromatin and dense granular euchromatin. Cytoplasmic granular debris containing organelles coated the deepest cells. In the areas sampled, the wing and superficial cells were unremarkable. No virions were identified.

Case 2—A 64-year-old man with acute myeloblas-

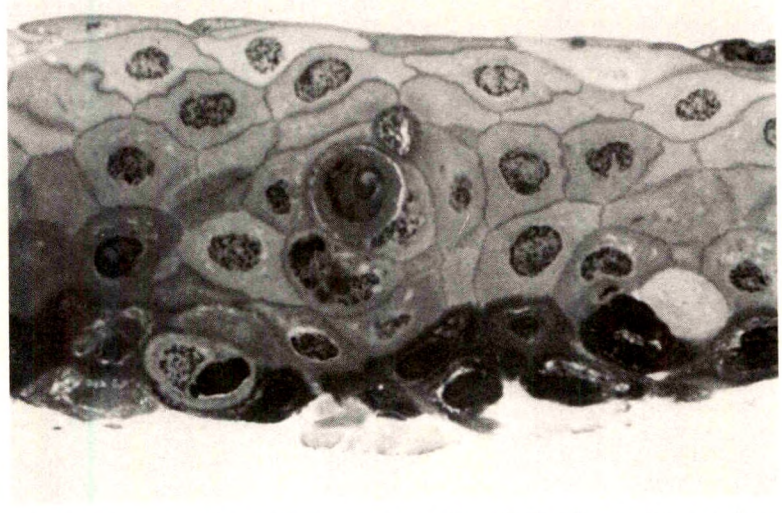


Fig. 1 (Hopen and associates). Case 1. Cells with deeply staining cytoplasm and pyknotic nuclei present throughout the basal layer of the corneal epithelium. An early stage of a microcyst represented by an isolated degenerating cell is present within the mid-epithelium centrally (toluidine blue, ×800).



Fig. 2 (Hopen and associates). Case 1. The markedly altered corneal basal epithelium contains several layers of flattened cells with electron-dense cytoplasm and shrunken nuclei (×6,720).

tic leukemia underwent various courses of chemotherapy with daunomycin, thioguanine, and cytarabine in standard doses from November 1979 to August 1980.

The patient was hospitalized for evaluation of progressive lethargy and angina after exertion in August 1980. Physical examination at admission disclosed normal extraocular motility, conjunctivas, and fundi. The oral mucosa showed scattered petechiae. Chest examination showed an increased anterposterior diameter with normal breath sounds. A grade 2/6 systolic murmur was heard over the aortic area. Results of abdominal and neurologic examinations were normal. Laboratory tests gave the following values: white blood cell count, 1,100/mm^3 with 8% neutrophils, 3% bands, 64% lymphocytes, 4% monocytes, and 15% blast cells; hemoglobin, 8.1 g/100 ml; hematocrit, 23.4%; and platelet count, 27,000/mm^3. Prothrombin time and partial thromboplastin times were normal. A chest X-ray film gave normal results, and an electrocardiogram showed no acute changes. A bone marrow aspirate on admission showed blast cells with Auer rods.

The patient was given intravenous cytarabine (3 g/m^2) every 12 hours for four days. One day after completing his course of chemotherapy, the patient complained of tearing, burning, blurred visual acuity, foreign-body sensation, and redness in both eyes. The patient had used no topical ocular medications except artificial tears. On examination, his visual acuity was R.E.: 6/60 (20/200) and L.E.: counting fingers. There was an iridectomy in the right eye from a cataract extraction in March 1977. He was wearing a hard contact lens in his right eye. Both pupils reacted to light, and his extraocular motility was full. Preauricular and submandibular nodes were not palpable. The eyelids and eyelashes were normal. Bilateral conjunctival hyperemia was present without prominent papillae or follicles. Fine corneal epithelial opacities and refractile microcysts were seen in both eyes; the lesions in the left cornea were denser and more numerous than those in the right. There was mild superficial punctate staining with fluorescein bilaterally. The anterior chamber was clear in both eyes. Results of an ophthalmoscopic examination were normal in the right eye, but the left fundus was poorly visualized because of a dense cataract. Cultures of the eyelids and conjunctivas on blood agar and mannitol plates showed coagulase-positive, mannitol-negative, beta-hemolytic staphylococci. Viral cultures of the conjunctiva for adenovirus and herpes simplex were negative.

Approximately one week later, his symptoms had cleared up without treatment. The conjunctival hyperemia resolved, and the corneal lesions disappeared.

Case 3—A 55-year-old man, examined in May 1977, had a four-month history of fever, malaise, petechiae, and an axillary mass on the left side. A diagnosis of acute myelomonocytic leukemia was finally made in August 1978. Treatment consisted of various courses of doxorubicin HCl, daunomycin, thioguanine, and cytarabine in standard doses during the next 20 months.

From April until August 1980, the patient was an outpatient undergoing chemotherapy with thioguanine, 160 mg daily every other week.

In September 1980, a bone marrow biopsy showed blast cells, and the patient was admitted for another course of chemotherapy. On physical examination, his pupils, conjunctivas, extraocular motility, and fundi were normal. Results of examinations of the chest, heart, lungs, abdomen, and extremities were normal. Laboratory tests gave the following values: white blood cell count, 2,800/mm^3 with 51% neutrophils, 36% lymphocytes, 8% monocytes, 1% eosinophils, 1% atypical lymphocytes, and 3% blast cells; hemoglobin, 15.7 g/100 ml; hematocrit, 41.1%; and platelet count, 115,000/mm^3. Results of a chest X-ray film and electrocardiogram were within normal limits.

The patient received two doses of daunomycin (45 mg/m^2), and cytarabine (3 g/m^2) was started intravenously every 12 hours for four days. Five days after completing chemotherapy, he requested ophthalmologic consultation because he had had ocular burning, tearing, foreign-body sensation, and photophobia in both eyes for two days. The symptoms began in the left eye and were noted about 12 hours later in the right eye. He was not using ocular

medications. The patient had a history of amblyopia in his right eye.

On examination his corrected visual acuity was R.E.: 6/60 (20/200) and L.E.: 6/12 (20/40). The pupils reacted normally to light and accommodation, and his extraocular motility was full. Preauricular and submandibular lymph nodes were not palpable. Tension by applanation tonometry was 18 mm Hg in both eyes. The eyelids and eyelashes were normal. There was bilateral conjunctival hyperemia without prominent papillae or follicles. Both corneas showed fine epithelial opacities and refractile microcysts that were more numerous in the central than in the peripheral cornea (Fig. 3). Mild superficial punctate staining with fluorescein was present bilaterally. There were folds in Descemet's membrane and mild stromal edema. The anterior chamber was clear in both eyes. Results of an ophthalmoscopic examination were normal in both eyes. Cultures of the eyelids and conjunctivas on blood agar and mannitol plates were negative for potential pathogens. Viral cultures of the conjunctiva for adenovirus and herpes simplex were negative.

A soft contact lens was placed in the right eye. On the following day there was no change in his symptoms. His corrected visual acuity was R.E.: 6/120 (20/400) and L.E. 6/15 (20/50). When the soft contact lens was removed from the right eye, the corneal epithelium showed punctate and geographic areas of staining with fluorescein. There were mild stromal edema and folds in Descemet's membrane in both eyes. The right eye was patched for 24 hours; 2% cyclogyl and sodium sulfacetamide drops were then instilled four times each day. On the following day, the patient stated that his symptoms were improving in the left eye. Slit-lamp examination showed a decrease in the number of fine epithelial opacities, microcysts, and punctate erosions in the left eye. The right eye showed an inferior epithelial defect approximately 1.5 mm in diameter. Five days later the patient had no symptoms in either eye, and the appearance of his corneal epithelium returned to normal by slit-lamp examination.

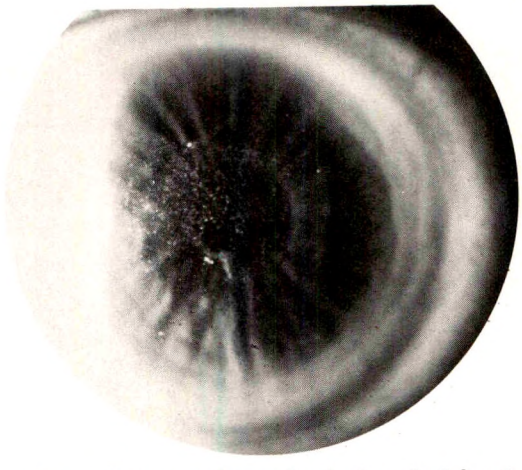


Fig. 3 (Hopen and associates). Case 3. Sclerotic scatter used to demonstrate numerous central epithelial opacities.

Discussion

High doses of systemic cytarabine have been used in an attempt to increase the effectiveness of the drug in the treatment of acute nonlymphocytic leukemia. Momparler[4] proposed a model for high doses and treated one patient with 1.7 g/m^2 of cytarabine. Rudnick and associates[3] treated 13 patients with 1.0 to 7.5 g/m^2 cytarabine. The reported adverse effects included nausea and vomiting, fever, somnolence, one episode of inappropriate vasopressin secretion, and two cases of spontaneously resolving conjunctivitis without corneal abnormalities.

Cytarabine inhibits DNA synthesis but not DNA repair.[4,5] It exerts its action at the DNA polymerase level. Accordingly, adverse effects should be greatest on rapidly dividing cells.[4,6] It is not surprising, therefore, to see involvement of the corneal epithelium. Corneal toxicity is a well known complication of topical administration of cytarabine. Elliott and Schut[2] described a fine speckling of the corneal epithelium with refractile bodies in rabbits, monkeys, and humans that reached a peak after six to eight days of topical cytarabine. Histopathologic studies showed thinning of the corneal epithelium with degeneration of individual epithelial cells.

Kaufman and associates[1] described corneal toxicity from topical cytarabine as glittering opacities in the lower layers of the epithelium. The epithelium did not stain with fluorescein initially, but a punctate staining pattern developed as the toxicity became more severe. They demonstrated that cytarabine inhibits DNA synthesis and enzymes of the glyco-

lytic pathway in the cornea. The corneal toxicity did not occur when the eyes were treated concurrently with deoxycytidine, which reverses the inhibition of DNA synthesis by cytarabine.

The corneal changes we observed after systemic administration of cytarabine resembled descriptions of those seen after topical administration of cytarabine, and are probably secondary to inhibition of DNA synthesis in the corneal epithelium. In addition to microcyst formation, histopathologic examination of the corneal epithelium in Case 1 showed a zonal topographic distribution of degenerating cells diffusely involving the rapidly reproducing cells of the basal layer. Corneal toxicity secondary to systemic cytarabine has not been previously described. Cytarabine in high doses probably overloads the deaminase system that inactivates cytarabine to arabinosyl uridine in dividing cells and results in increased toxicity.[4-6] Cytarabine is known to penetrate the blood-brain barrier after intravenous injection[4,6] and may also penetrate the blood-aqueous barrier and be found in the aqueous humor. Cytarabine may enter the cornea not only through the aqueous humor but also through the tears.

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ELECTROPHYSIOLOGIC CHANGES IN CHRONIC PARS PLANITIS

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We studied 13 patients, six males and seven females, ranging in age from 14 to 53 years, with chronic pars planitis and a form of secondary retinal degeneration. All had experienced visual blurring but none had significant pain. Nine of the 13 had some disturbance in night vision. Visual acuity had decreased in three patients (in one because of cataract, in one because of cystoid macular degeneration, and for unknown reasons in the third), increased in two patients (because of improvement in cystoid macular edema), and remained stable in eight.

There were electrophysiologic abnormalities, consisting of delayed B-wave implicit time, abnormal response to 30-Hz flicker, and reduced B-wave oscillations, in almost every patient.

Our finding suggested that pars planitis is a vitreoretinal disorder rather than an inflammatory disease of the uveal tract.

Many of our patients with chronic pars planitis complain of night blindness and dimness of vision. Some of them have peripheral pigmentary changes and peripheral vascular attenuation. These findings may reflect a form of secondary retinal degeneration that is the result of chronically active disease. Although loss of central vision for this reason would be unusual in patients with pars planitis, secondary retinal degeneration may account for some of their visual complaints. We studied the results of electrophysiologic testing in 13 patients with chronic pars planitis. Our findings support the view that pars planitis is a vitreoretinal disorder rather than an inflammatory disease of the uveal tract.

SUBJECTS AND METHODS

We reviewed the charts of all patients with pars planitis seen here during the past five years. We included only those patients with a history of unilateral or bilateral visual blurring with minimal anterior chamber reaction and the typical vitreous cellular findings with pars plana exudates or fibrosis in this study. We excluded all patients with signs of significant anterior chamber reaction, posterior synechiae, or evidence of chorioretinal scarring. We also excluded those patients with systemic findings of sarcoidosis, syphilis, Behçet's disease, or Reiters' syndrome. No patient fit the clinical description of birdshot choroidopathy.[1] No patient had a family history of night vision disorders.

Only those patients who had had symptoms for at least two years were considered to have chronically active disease. We re-examined 13 patients who met

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these criteria. All patients were specifically questioned about night vision abnormalities. We performed a complete visual examination, including best corrected visual acuity, slit-lamp examination of the anterior chamber and vitreous, and ophthalmoscopic examination by indirect ophthalmoscopy with scleral depression. Goldmann visual fields and photopic and scotopic electroretinography were performed on all patients. Additionally, most patients underwent fundus photography and fluorescein angiography.

Electroretinography—We performed electroretinography, as previously described,[2] after pupils were dilated and light adapted at more than 5 ft L. We used a bipolar contact lens with a photostimulator placed 12 inches above the patient's head in an electrically shielded room. The stimulus measured approximately 30 degrees of visual field. Photopic recordings were made with a series of balanced white, red, and blue lights. We used a 30-Hz flicker to determine the phase responses to white light at maximum intensity. The patients' eyes were then dark adapted for 20 minutes and scotopic responses to white light flashes were recorded, starting with the threshold intensity necessary to evoke a measurable scotopic B-wave. We then increased the intensity in increments of 0.5 log units or less in order to define the maximum B-wave. We made at least two identical recordings at each stimulus intensity to insure reproducibility of responses. Each eye was tested separately.

We recorded B-wave amplitude and implicit time (100 μV/division amplitude and 10 msec/division sweep speed) by photographing the oscilloscope tracings. We measured B-wave amplitude from the A-wave trough to the B-wave peak. We measured B-wave implicit time from the stimulus artifact to the B-wave peak, using the most intense light stimulus. We recorded the presence or absence of B-wave oscillations.

Response amplitudes and latencies have been standardized in normal volunteers in our laboratory and are reproducible. For the purposes of our study, we considered photopic B-wave amplitudes of less than 80 μV and scotopic amplitudes of less than 250 μV subnormal. We considered photopic B-wave amplitudes of more than 120 μV and scotopic amplitudes of more than 500 μV supranormal. Photopic B-wave implicit times of less than 36 msec were considered normal as were scotopic implicit times of less than 50 msec. We considered phase relations to 30-Hz flicker delayed if the peak response appeared after the following stimulus artifact (33 msec).

Results

Thirteen patients fulfilled the criteria for inclusion in this study (Table 1). There were six males and seven females. The average age was 31 years, with a range from 14 to 53 years. The average duration of symptoms was longer than six years, with a range from two to 11 years. The average follow-up period was 3.8 years. Five patients had unilateral involvement (38%) and eight had bilateral involvement (62%).

All 13 patients complained of visual blurring at some time during the course of their disease. No patient complained of significant pain. In three patients (23%), episodes of disease activity were separated by intervals during which they remained asymptomatic. However, most of the patients showed a slowly progressive course without exacerbations or remissions (77%). Nine patients (69%) gave a definite history of night vision disturbance, but the remaining four (31%) specifically denied any problems of night vision. There was no relationship between the night vision complaints and the duration of disease. In all cases the

TABLE 1

CLINICAL FINDINGS IN 13 PATIENTS

Patient, Sex, Age (yrs)	Involvement	Duration of Symptoms (yrs)	Night Blindness	Clinical Course	Visual Fields	Pigmentary Changes
1, F, 27	Unilateral	6½	Yes	Progressive	Blunting	Focal
2, F, 51	Unilateral	4	Yes	Progressive	Normal	Focal
3, F, 25	Unilateral	9	Yes	Intermittent	Constriction	None
4, F, 40	Unilateral	6	Yes	Progressive	Normal	None
5, F, 28	Unilateral	4	Yes	Progressive	Blunting	Diffuse
6, M, 26	Bilateral	2	No	Intermittent	Normal	None
7, M, 18	Bilateral	5	No	Progressive	Blunting	None
8, M, 40	Bilateral	9	Yes	Progressive	Constriction	Diffuse
9, M, 14	Bilateral	4½	No	Progressive	Normal	None
10, M, 36	Bilateral	11	Yes	Progressive	Normal	Focal
11, M, 53	Bilateral	2	Yes	Progressive	Constriction	Diffuse
12, F, 22	Bilateral	8½	Yes	Intermittent	Normal	Perivascular
13, F, 25	Bilateral	10	No	Progressive	Normal	None

degree of night vision impairment was mild compared to that usually seen in patients with hereditary retinal degenerations.

Eleven patients were intermittently treated with systemic or periocular corticosteroids. The indication for treatment was either an increase in the vitreous cellular reaction or clinically significant cystoid macular edema. No patient was treated with immunosuppressive agents or cryotherapy. Two patients developed tractional retinal tears during the course of their disease. These were focally treated with either laser or cryopexy. One patient developed a retinal detachment that could not be repaired successfully.

Some degree of vitreous cellular reaction with accumulation of debris over the pars plana inferiorly was present in all patients. Fine cellular dusting of the anterior vitreous was the most common finding. Two patients showed marked syneresis with vitreous membrane formation and collapse. White, fluffy debris bridging the ora serrata in the area of the vitreous base was seen most frequently. Some patients also had yellow nodular opacities floating freely over the peripheral retina. Six patients showed extensive pars plana fibrosis. These peripheral changes had a white, fibrous texture and appeared to be adherent to the underlying retina and pars plana. Vascularization of this fibrous membrane was noted in one case.

We found retinal pigmentary changes in seven patients. Three patients had a diffuse granularity and stippling of the pigment epithelium in the periphery. Three patients had focal areas of peripheral pigment clumping. One patient showed localized perivascular pigment epithelial atrophy with some clumping along the vessel wall. We did not find typical bone-corpuscular pigmentary changes in any patient. There was a relationship between the presence of pigmentary changes and night vision complaints. Generalized vascular attenuation or optic nerve atrophy were not noted in any patients; however, vascular attenuation in the extreme periphery was a common finding. Cystoid macular edema and optic disk edema were intermittently present in all patients.

Visual acuity remained surprisingly stable throughout the course of the observation period. Only three patients showed a significant decline in visual

acuity. In one patient decreased visual acuity was caused by a cataract; in the second it was caused by cystoid macular degeneration. The cause in the third patient was obscure. Significant visual improvement was noted in two patients, both caused by improvement in cystoid macular edema. In eight patients visual acuity remained stable.

For the purposes of our study, enlargement of the blind spot and the presence of a relative central scotoma were not considered visual field abnormalities. In seven patients the visual fields were normal. Three patients showed depression of the visual field in one meridian or quadrant; the depression could be explained by the retinal abnormalities. One patient showed constriction to within 30 degrees of fixation using the I_4e test object. A ring scotoma was not detected in any of the patients. The degree of visual field abnormality could not be correlated with other clinical findings, such as the duration of symptoms and the presence of pigmentary changes or night vision abnormalities, except in one case with severe involvement.

Electrophysiologic testing showed a wide variation in B-wave amplitude and reduced B-wave amplitudes were found in three patients (Table 2). Two of these three patients had evidence of severe involvement, indicated by night blindness and widespread pigmentary changes. The third patient had unilateral involvement, but reduced response amplitudes in both eyes with an otherwise normal record in the uninvolved eye. Supranormal responses were recorded in four

TABLE 2

ELECTROPHYSIOLOGIC FINDINGS

		Photopic			Scotopic		
Patient	Eye	B-Amplitude	B-Implicit Time	Phase Relations	B-Amplitude	B-Implicit Time	B-Oscillations
1	R.E.	120	39	Delayed	320	57	Absent
	L.E.	80	34	Normal	300	50	Normal
2	R.E.	100	39	Delayed	500	55	Absent
	L.E.	100	34	Normal	400	48	Normal
3	R.E.	60	40	Delayed	250	58	Reduced
	L.E.	50	34	Normal	250	48	Normal
4	R.E.	80	34	Normal	500	45	Normal
	L.E.	100	38	Delayed	400	55	Absent
5	R.E.	110	40	Delayed	250	52	Absent
	L.E.	100	34	Normal	300	46	Normal
6	R.E.	160	38	Delayed	650	55	Reduced
	L.E.	160	37	Delayed	650	54	Reduced
7	R.E.	140	34	Normal	550	48	Normal
	L.E.	140	35	Normal	600	50	Normal
8	R.E.	60	40+	Absent	160	60	Absent
	L.E.	40	40+	Absent	160	60	Absent
9	R.E.	160	38	Delayed	500	52	Absent
	L.E.	120	38	Delayed	500	52	Absent
10	R.E.	100	40	Delayed	350	55	Absent
	L.E.*	—	—	—	—	—	—
11	R.E.	80	40+	Delayed	180	60	Absent
	L.E.	80	40+	Delayed	180	60	Absent
12	R.E.	160	38	Delayed	500	53	Absent
	L.E.	160	38	Delayed	500	55	Reduced
13	R.E.	120	33	Normal	300	50	Reduced
	L.E.	120	33	Normal	350	48	Normal

*Vision lost because of retinal detachment.

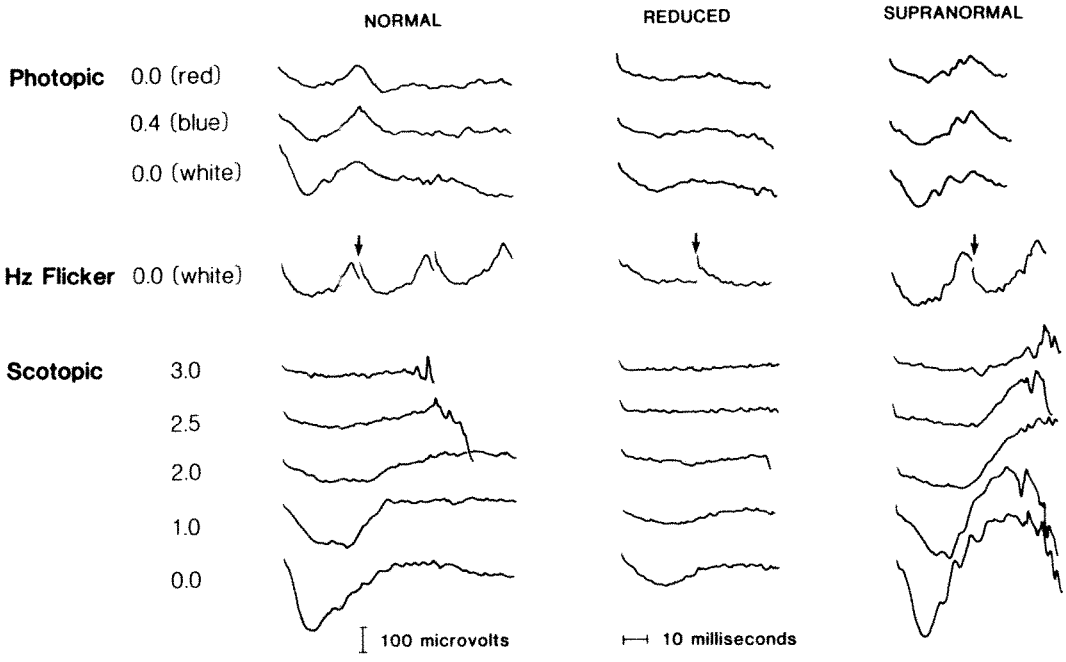


Fig. 1 (Cantrill and associates). Electroretinograms from a normal subject and from two patients with pars planitis demonstrating reduced and supranormal responses. Photopic responses to balanced red, blue, and white light (top), phase response to 30-Hz flicker (middle), and scotopic response to white light of increasing intensity (bottom) are shown. The position of the stimulus artifact is indicated by the vertical arrow.

patients. Only one of these four patients had night blindness and pigmentary changes. Response amplitude did not correlate with the duration of the disease. Normal, reduced, and supranormal records from three of our patients are shown in Figure 1.

Prolonged B-wave implicit time, abnormal phase relations to 30-Hz flicker, and the absence of scotopic B-wave oscillations were more sensitive indicators of disease activity. Photopic and scotopic B-wave implicit times were normal in only two patients. Neither patient complained of night blindness or had pigmentary changes. The five patients with unilateral involvement showed significant prolongation of the implicit time only in the involved eye. These differences were most obvious under photopic conditions and were most easily measured in the response to 30-Hz flicker. In the five patients with unilateral involvement, there was no difference in B-wave amplitude between the involved and the uninvolved eye. Figure 2 compares the ERGs of the involved and fellow eye of one of our patients with unilateral disease. Reduction or absence of scotopic B-wave oscillations also provided a sensitive indicator of involvement. Twelve patients (92%) showed such changes. Again, the responses were consistent in the five patients with unilateral disease. There was a relationship between these abnormal temporal aspects of the electroretinogram and clinical findings such as night blindness, pigmentary changes, and overall severity of the disease. Response latency did not correlate with duration of disease.

Discussion

Pars planitis is a poorly understood clinical entity which was first described by Schepens[3] in 1950. Since that time, the disorder has been called peripheral uveitis, chronic cyclitis, uveoretinitis,

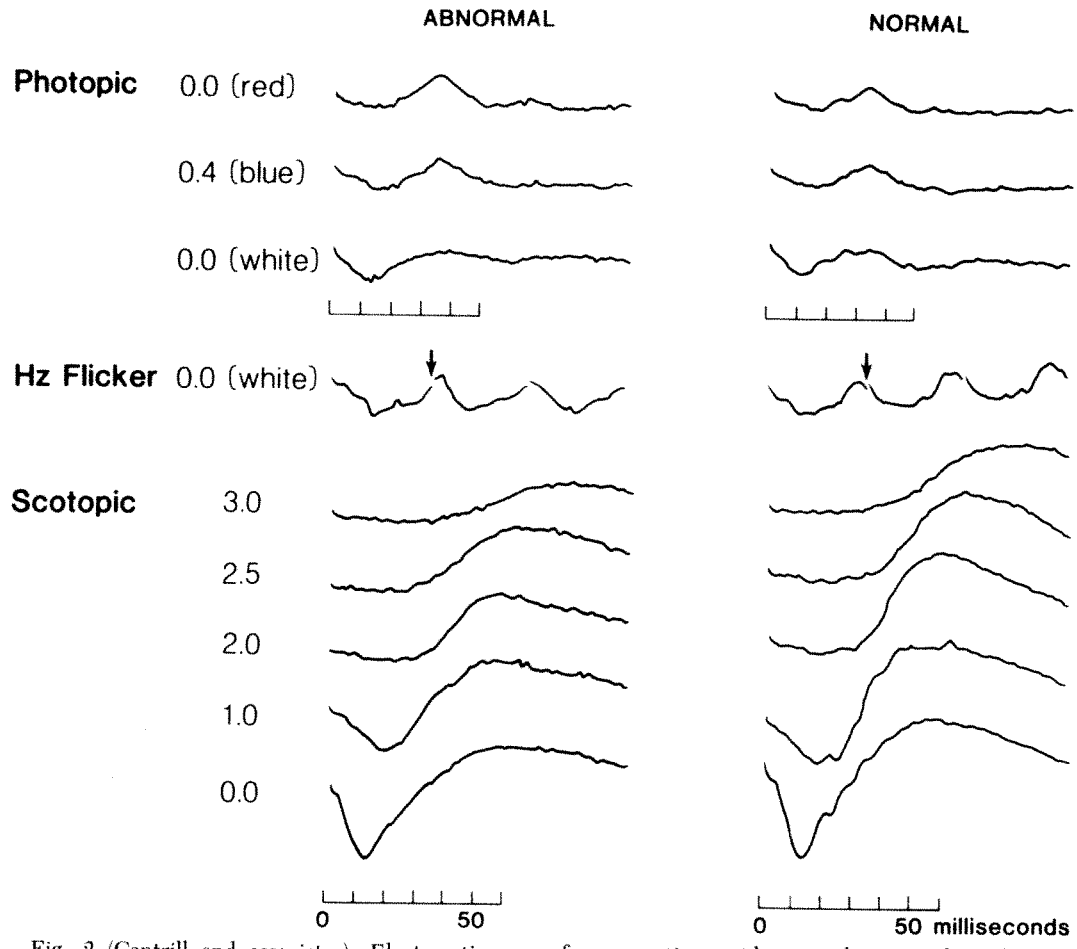


Fig. 2 (Cantrill and associates). Electroretinogram from a patient with pars planitis with unilateral involvement. Photopic B-wave implicit time is prolonged in the involved eye (top). The stimulus artifact (vertical arrow) precedes the cone B-wave in the involved eye and follows it in the uninvolved eye (center). Scotopic B-wave implicit time is prolonged in the involved eye (bottom).

angioretinitis, and vitritis—terms referring to the clinical manifestations of the disease rather than its cause. The term pars planitis was first used by Welch, Maumenee, and Whalen[4] in 1960. Pars planitis was originally thought to be granulomatous uveitis with an inflammatory focus in the ciliary body, peripheral choroid, or peripheral retinal vessels.[5] Some eyes studied pathologically have shown inflammatory cells in the choroid and surrounding peripheral retinal vessels, but these eyes had been removed for complications of diseases such as secondary glaucoma or retinal detachment.[5,6] The pathologic findings in the acute form of the disease are unknown. Pederson and associates,[7] in a review of seven cases studied pathologically, drew attention to the lack of inflammation in the uveal tract, and postulated that the disease sequela may be a result of vitreoretinal inflammation. They also showed that the vitreous "snowbank," previously thought to be composed of chronic inflammatory cells, was composed of condensed vitre-

ous debris and glial cells derived from the retina, again suggesting a vitreoretinal source. Finally, Pruett, Brockhurst, and Letts[8] drew attention to the vascular component of the disease by noting perivascular leakage on fluorescein angiography in all their cases. This vascular hyperpermeability is directly related to the major vision-threatening complication of the disease, cystoid macular edema. Our findings of electrophysiologic abnormalities in chronic pars planitis also support a vitreoretinal pathogenesis.

Electroretinography has been used in the evaluation of retinal degenerations, toxic retinopathies, and widespread retinal vascular disease. In most retinal degenerations, the electrophysiologic changes are quite advanced early in the course of the disease. In some forms of retinal degeneration, however, such as dominantly inherited retinitis pigmentosa with reduced penetrance and X-chromosome-linked retinitis pigmentosa, there can be more subtle abnormalities of amplitude and latency.[9,10] Some of these patients can be detected only on the basis of prolonged B-wave implicit time.[11] Similarly, patients with involutional proliferative diabetic retinopathy can have subnormal responses and delayed implicit times which progress with time.[2] Our findings in patients with chronic pars planitis are similar to these. We found abnormalities of response latency in 85% of the patients tested, whereas reduction in amplitude was present in only 15%. The degree of electrophysiologic abnormality did relate to the clinical severity of night blindness, pigmentary changes, and field loss. Visual acuity did not correspond to these electrophysiologic changes because visual acuity depends upon localized retinal function and on the presence of cystoid macular edema in particular.

The temporal aspects and the amplitude of the ERG can vary independently. Focal chorioretinal scarring reduces the amplitude of the ERG without affecting the timing of the response.[9] Even panretinal photocoagulation for proliferative retinopathy has little effect on the timing of the ERG.[12] The timing of the ERG response, however, is influenced by such factors as uniformity of retinal stimulation, stimulus strength, and the state of retinal adaptation. Focal stimulation may produce faster responses or a heterogeneous group of responses, making it difficult to interpret the response timing. Implicit time is inversely related to stimulus strength. Finally, implicit time decreases with dark adaptation. None of these factors can explain the abnormal electrophysiologic findings in our patients, particularly because of the normal findings in the fellow eyes of unilaterally involved patients.

Electrophysiologic abnormalities are unusual even in severe forms of anterior and posterior uveitis. Algverre[13] found reduced response amplitudes only in patients with extensive chorioretinal scarring. Supranormal responses were noted in some cases. There was a slight prolongation of the B-wave in some patients with chronic uveitis, but no significant differences. More severe forms of uveitis, such as Behçet's disease, typically show profound electrophysiologic changes. These are not unexpected because of the severe retinal vasculitis seen in this disease. Electrophysiologic abnormalities have also been produced in experimental forms of uveitis corresponding to the extent of clinical and histopathologic changes in the retina.[14] An increase in ERG amplitude, presumably caused by retinal irritation, has been demonstrated in mild cases.

Peripheral pigmentary changes have been reported in some patients with pars planitis. In a follow-up study of 100

patients with pars planitis, Smith, Godfrey, and Kimura[15] had one patient who developed extensive pigmentary changes during the course of the illness. Results of electrophysiologic testing were not reported. Chester, Blach, and Cleary[16] reviewed 51 cases of pars planitis diagnosed on the basis of the typical pars plana exudates. Six of these cases were associated with retinitis pigmentosa. Four patients had contributory family histories and all six had unrecordable ERGs.

These patients probably did not have retinal degeneration secondary to pars planitis. Characteristic changes in the vitreous can be seen in patients with retinitis pigmentosa.[17] Dust-like opacities similar to the typical response seen in pars planitis are characteristic. In some patients with retinitis pigmentosa, we have seen peripheral globular opacities and pars plana exudates not unlike those seen in patients with pars planitis. These changes in the vitreous are related to the retinal degenerative process. It is interesting to speculate whether a similar vitreoretinal degenerative process accounts for the clinical findings in the vitreous of patients with pars planitis and those with retinitis pigmentosa. Additionally, both groups share the frequent complication of cystoid macular edema, the origin of which remains unclear.

Our findings suggest that ERG abnormalities are common in patients with chronic pars planitis and correlate with clinical findings of night blindness and pigmentary changes. They suggest that a mild secondary retinal degeneration can occur in chronic pars planitis and account for some visual complaints. Our findings are in keeping with a pathogenesis based on a vitreoretinal inflammatory process rather than inflammation within the uveal tract. A prospective study will be necessary to document the development of these clinical and electrophysiologic changes during the course of the disease.

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COMBINED JONES TUBE-CANALICULAR INTUBATION AND CONJUNCTIVAL DACRYOCYSTORHINOSTOMY

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Six patients underwent combined Jones tube-canalicular intubation and conjunctival dacryocystorhinostomy. These patients had epiphora secondary to partial obstruction of the upper and lower canaliculi, complete obstruction of the common canaliculus or a single canaliculus, or paresis of the orbicularis oculi muscle. In these patients we could have performed a dacryocystorhinostomy with silicone elastic intubation, risking recurrent obstruction, or a conjunctival dacryocystorhinostomy with placement of a Jones tube, risking failure of the procedure and complete obstruction of patent canaliculi by placement of the tube.

The combined Jones tube-canalicular silicone elastic intubation and conjunctival dacryocystorhinostomy ensured successful treatment of epiphora for this group of patients. Intubating the canaliculi with Bowman probes during placement of the Jones tube avoids injury and subsequent postoperative canalicular obstruction. After several months, the silicone elastic tubing is removed and the Jones tube is plugged. If the patient is asymptomatic and the canaliculi are patent and can be irrigated freely to the nasal cavity, the Jones tube can be removed. However, if epiphora occurs, the plug can be removed and the tube left in place to drain the tears without additional surgery.

A dacryocystorhinostomy is a preferred method of relieving epiphora and discharge secondary to partial or complete acquired nasolacrimal duct obstruction, lacrimal dacryoliths, and lacrimal diverticulum. A conjunctival dacryocystorhinostomy with placement of a Jones tube is performed for epiphora secondary to upper and lower canalicular obstruction.[1]

In patients with epiphora secondary to lacrimal pump dysfunction, obstruction of only one canaliculus, or common canalicular obstruction, whether to perform a dacryocystorhinostomy or a conjunctival dacryocystorhinostomy becomes a more difficult decision. A dacryocystorhinostomy with excision of the obstructing canalicular tissue and temporary intubation of the lacrimal passages with silicone elastic tubing relieves epiphora in some of these patients.[2] However, recurrent epiphora secondary to repeated stenosis of the lacrimal passages frequently occurs and later requires a conjunctival dacryocystorhinostomy with placement of a Jones tube. If the Jones tube is used in the initial procedure, there will be some failures because the tube may shift or because patients are unwilling to care for the tube postoperatively or unable to tolerate the occasional need for tube replacement or adjustment. Because a conjunctival dacryocystorhinostomy fre-

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quently obstructs or destroys part of the distal or common canaliculi that was not previously obstructed, patients who cannot tolerate the Jones tube may suffer more epiphora postoperatively than preoperatively.

We have developed a combined Jones tube-conjunctival dacryocystorhinostomy with canalicular silicone elastic intubation that makes a second operation unnecessary and preserves the unobstructed canaliculi. The silicone elastic tubing is removed and the Jones tube is plugged postoperatively. If the patient remains asymptomatic and the canaliculi can be irrigated freely to the nasal cavity, the Jones tube can be removed. However, if the symptoms recur, the plug can be removed and the Jones tube left in place to relieve symptoms without performing additional surgery. The procedure has had good results in six patients.

Material and Methods

All patients undergo a complete evaluation of the lacrimal system, with particular attention given to the excretory system. The dye retention test is performed by instilling two drops of sodium fluorescein in the superior cul-de-sac. After two minutes the amount of dye retained at the eyelid-cornea junction is graded from 0 to 4. In the normal patient, no dye remains after two minutes.

The primary dye test is given immediately after the dye retention test. Saline solution is irrigated into the nasal cavity ipsilateral to the eye in which fluorescein was instilled. The patient is then instructed to bend the head forward, occlude the contralateral nostril, and blow into a piece of white tissue paper. The presence of fluorescein suggests a normal physiologic lacrimal excretory system. If no dye appears on the paper, the secondary dye test is performed.

A lacrimal needle is placed in the lacrimal sac and saline solution is irrigated through the upper or lower canaliculus. Several milliliters of solution are injected while the patient bends the head forward and holds a piece of white tissue paper beneath the nostrils. The presence of fluorescein suggests a normal physiologic lacrimal excretory system. If no dye appears on the paper, the secondary dye test is performed.

A lacrimal needle is placed in the lacrimal sac and saline solution is irrigated through the upper or lower canaliculus. Several milliliters of solution are injected while the patient bends the head forward and holds a piece of white tissue paper beneath the nostrils. The presence of fluorescein on the paper suggests that the nasolacrimal duct is partially obstructed. Clear saline on the paper indicates a partial obstruction of the punctum or canaliculus. If no saline or dye reaches the nose there may be a complete nasolacrimal obstruction. The ability to irrigate solution from the lower canaliculus through the lacrimal sac and out via the upper canalicular system is common with a complete obstruction of the nasolacrimal duct.

The procedure—A dacryocystorhinostomy is performed according to standard techniques, whereby the posterior nasal mucosal flap is sutured to the posterior lacrimal sac (Fig. 1, A).[2,3] The anterior tip of the middle turbinate is resected with a rongeur if it rises above the closed posterior flaps.

A punctum dilator is used to dilate the upper and lower lacrimal puncta, and a No. 0 Bowman probe is placed through the upper punctum and canaliculus into the common canaliculus and over the sutured posterior lacrimal sac-nasal mucosal flaps until it meets the nasal septum. A No. 00 or 1 Bowman probe is then placed through the lower punctum and canaliculus in a similar manner. An eyelid speculum is inserted between the eyelids to expose the caruncle. The Bow-

man probes are bent proximal to the puncta, and the ends are taped to the sterile head drapes (Fig. 1, B).

The nasal half of the caruncle is excised with Westcott scissors. A 19-gauge needle is bent slightly and is inserted 2 mm posterior to the skin-mucosal junction at the medial canthus and passed over the sutured posterior nasal mucosal-lacrimal sac flaps (Fig. 1, C). The needle is directed to avoid the intubated canaliculi by passing it anterior or posterior to the Bowman probes. A Von Graefe knife is passed along the side of the needle, and the needle is withdrawn (Fig. 1, D). The knife then slices in a superior and inferior direction to enlarge the track. A No. 1 or 2 Bowman probe is passed along the shaft of the knife until it meets the nasal septum. The Bowman probe is grasped with a hemostat where it enters the caruncle and is withdrawn. The distance from the probe end to the hemostat is measured, and a Jones Pyrex tube 3 to 4 mm shorter than this length is chosen. A tube with a 3- to 4-mm collar width is desirable. The selected Jones tube is slid onto the probe and the probe is passed again along the Von Graefe knife shaft; the knife is withdrawn. The Jones tube is slid through the conjunctival dacryocystorhinostomy ostium until its collar rests at the medial canthus; the distal end is within the nasal cavity but at least 3 mm from the nasal septum (Fig. 1, E). The Bowman probe is removed. The Jones tube is anchored by passing a 6-0 black silk double-armed suture several times around the tube just adjacent to its collar. Each end of the suture is passed through the skin of the medial canthus and tied to prevent the tube from sliding through the large ostium (Fig. 2, F). A 6-0 black silk suture passed through and around the tube is another way of securing the tube.[4]

A Guibor silicone elastic tube with a Bowman probe cemented to each end is used to intubate the canaliculi. One end of the tube is passed through the upper punctum and canaliculus and over the closed posterior lacrimal sac-nasal mucosal flaps (Fig. 2, G). The probe is withdrawn from the wound. A sterile ointment placed over the site where the silicone elastic tube meets the Bowman probe facilitates passage of the tube and prevents it from slipping off the metal probe. In a similar manner, the other probe end is passed through the lower punctum and canaliculus. The metal probes are removed from the silicone elastic tubing.

Nasal forceps are passed through the nasal cavity until the tips are visible over the closed posterior lacrimal sac-nasal mucosal flaps. The tips grasp each end of the silicone elastic tube and pull them into the nasal cavity and out the external nostrils (Fig. 2, H). A 4-0 polypropylene suture is passed around the tubes at the site where the tubes enter the nasal cavity from the lacrimal sac and where the tubes pass out of the external nostrils (Fig. 2, I). These sutures prevent loss or migration of the tubes postoperatively. The excessive ends of the tubes are then cut so that they are flush with the external nostrils.

The anterior lacrimal sac flap is then sutured to the anterior nasal mucosal flap with several 4-0 chromic catgut sutures. The periosteum is then sutured to periosteum with 4-0 polyglactin 910 sutures, subcutaneous tissue is sutured with 5-0 sutures, and the skin sutured with a continuous 6-0 black silk suture (Fig. 2, J).

Six days postoperatively, the skin sutures are removed. Three weeks postoperatively, the suture anchoring the Jones tube is removed. After three months, the silicone elastic tubing is severed between the upper and lower puncta and slid out of the nose.

The patient begins using dexamethasone phosphate ophthalmic solution four times a day for 1.5 weeks and then twice a

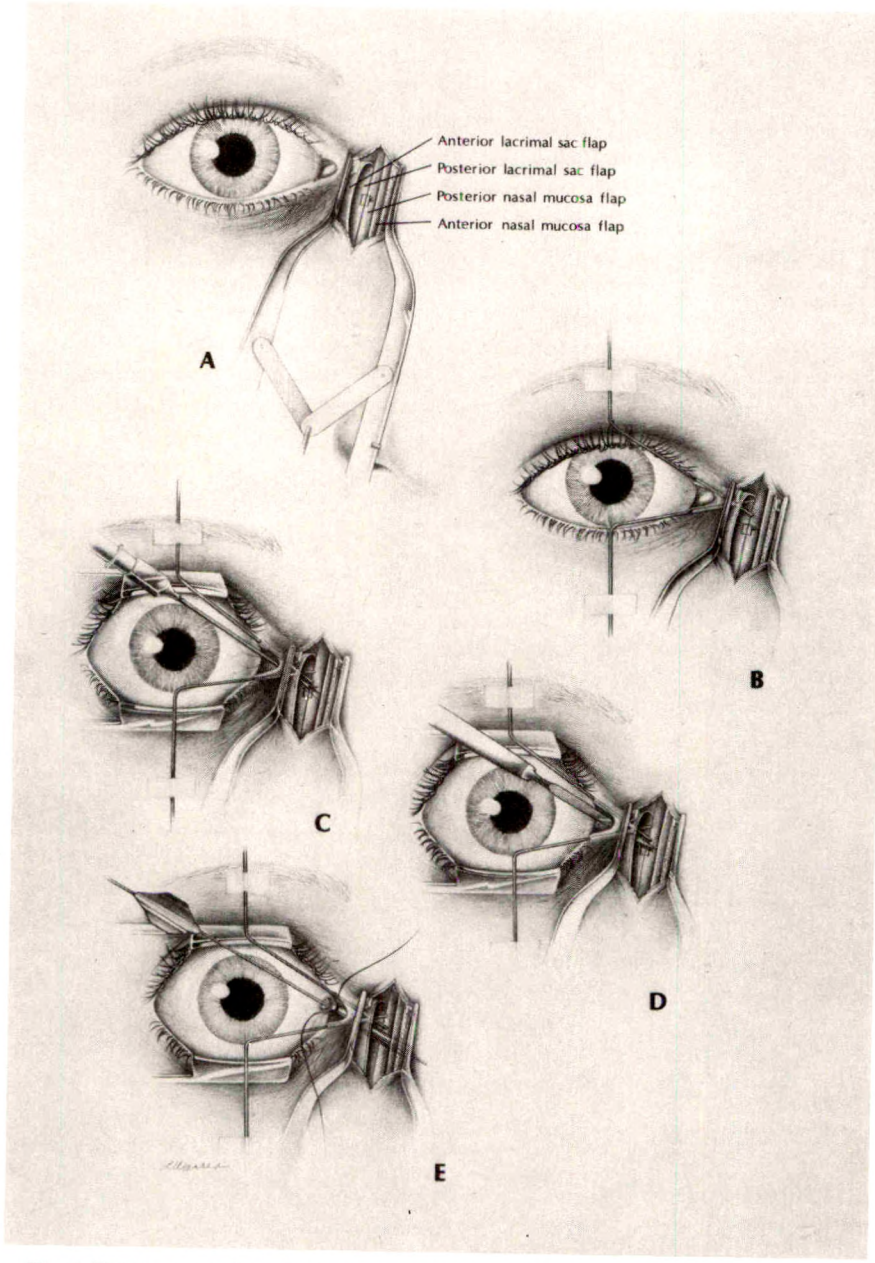


Fig. 1 (Putterman and Epstein). A, Posterior flaps of nasal mucosa and lacrimal sac have been closed with a 4-0 chromic catgut suture. The anterior flaps remain open at this stage. B, Bowman probes are placed through the upper and lower canaliculi into the common canaliculus and enter the new ostium between anterior and posterior flaps. The Bowman probes are taped to the surgical drape to ensure stability. C, A 19-gauge needle is passed 2 mm posterior to the skin-mucosal junction at the medial canthus, while the Bowman probes protect the common canaliculus. The needle may pass anterior or posterior to the probes. D, A von Graefe knife is passed through the pervious tunnel and then is rotated interiroly and superiorly to enlarge the tract. E, A Jones tube placed on a Bowman probe is slid through the tract. A 6-0 black silk double-armed suture is wrapped around the Jones tube several times.

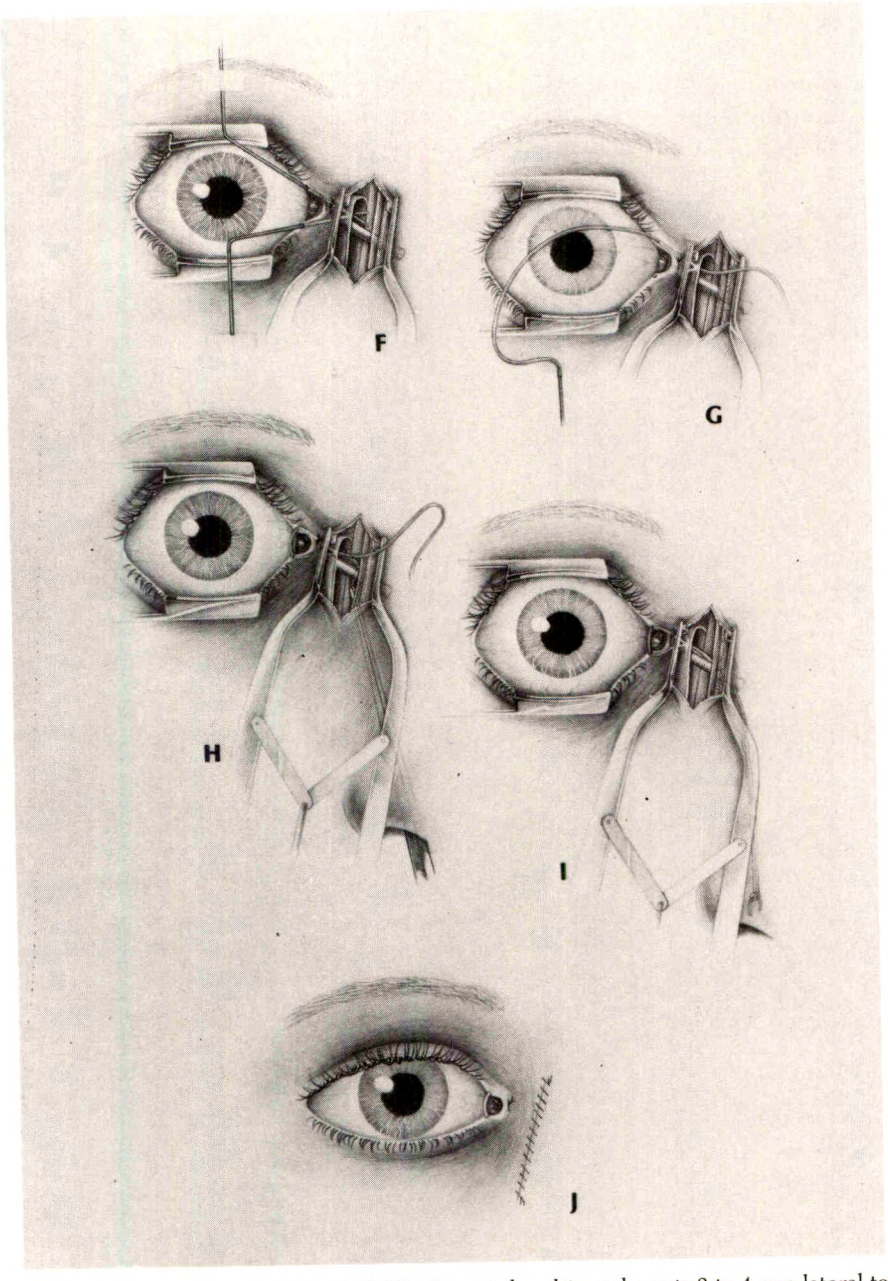


Fig. 2 (Putterman and Epstein) F, The Jones tube ultimately rests 3 to 4 mm lateral to the nasal septum. Each arm of the 6-0 black silk suture is passed on through the medial canthal skin to anchor the Jones tube and is then tied. G, A silicone elastic tube is passed through the upper canalicular system and out the lacrimal sac wound. The lower canalicular system is intubated with the other end of the metal probe. H, With the metal probes removed, each arm of the tubing is brought into the nasal cavity and the nostril by grasping with nasal forceps. I, A 4-0 polyester fiber suture conjoins the tubes in the ostium and in the distal nasal cavity to prevent migration. J, Final appearance of combined conjunctival dacryocystorhinostomy with silicone elastic intubation.

day for another 1.5 weeks. Next, the canaliculi are intubated with a lacrimal needle and are irrigated with saline solution. If the saline passes easily to the nasal cavity and no significant canalicular obstruction is present, the Jones tube is plugged with a special polyethylene stent (Fig. 3). If the patient remains free of tearing with the Jones tube plugged, removal of the Jones tube is possible. However, if canalicular obstruction or epiphora recurs, the plug is removed and the tube is left in place to drain the tears. (If the patient is asymptomatic and the Jones tube is in good position, we usually leave the tube in place, but are unconcerned if the tube eventually extrudes, migrates, or is lost.)

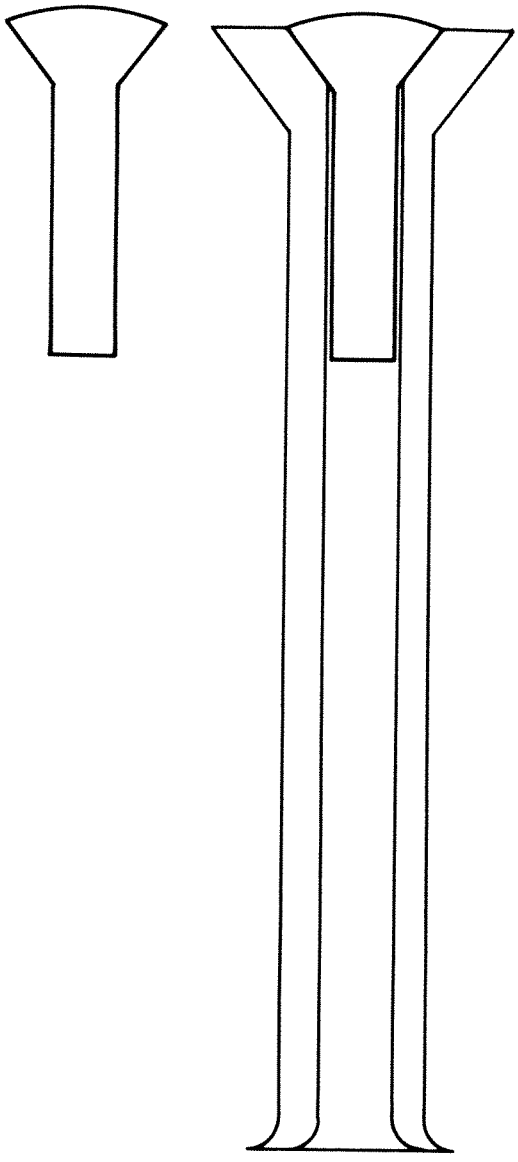


Fig. 3 (Putterman and Epstein). Polyethylene Jones tube plug.

CASE REPORTS

Case 1—A 69-year old man complained of tearing of his left eye of six months' duration. He reported that a military injury during World War II involving the left side of his face resulted in a partial left-sided facial paralysis.

Ocular examination disclosed 3+ dye retention with pooling of tears on the left side. The primary dye test failed to demonstrate fluorescein in the left nasal cavity. The secondary dye test, however, did show the presence of dye in the left nasal cavity, suggesting a partial obstruction of the nasolacrimal duct. The nasal cavity was free of disease, and no tears or discharge could be expressed from the left lacrimal sac with massage. A mild left-sided facial paresis was noted, but there was no evidence of ectropion, lagophthalmos, or significant weakness of the orbicularis oculi muscle. Results of the remainder of the ocular examination were unremarkable (Fig. 4).

A left-sided dacryocystogram showed slight retention of contrast material in the lacrimal sac on the 30-minute film. This was suggestive of a partial nasolacrimal duct obstruction. Because of the tear pooling and the partial facial paresis, it was thought that a weak tear pump mechanism was partly responsible for the patient's condition. It was our opinion that the presence of a poor tear pump mechanism would mean that a standard dacryocystorhinostomy would fail.

The patient underwent a left-sided conjunctival dacryocystorhinostomy. A 4 × 17 mm Jones Pyrex tube was inserted and silicone elastic tubing placed in the left lacrimal system. One month postoperatively, the left caruncle was excised further because it was blocking the Jones tube. Two months postoperatively, we removed the silicone elastic tubing. The upper and lower canalicular system could be irrigated freely to the nose. At the same time, we plugged the Jones tube. One week later, the patient complained of recurrent epiphora, although the canaliculi were patent. This confirmed our preoperative impression that a weak lacrimal pump mechanism was responsible for the recurrent epiphora. The plug was removed and the patient has remained asymptomatic for the past seven months (Fig. 4).

Case 2—A 48-year-old woman complained of tearing of her left eye of three months' duration. She

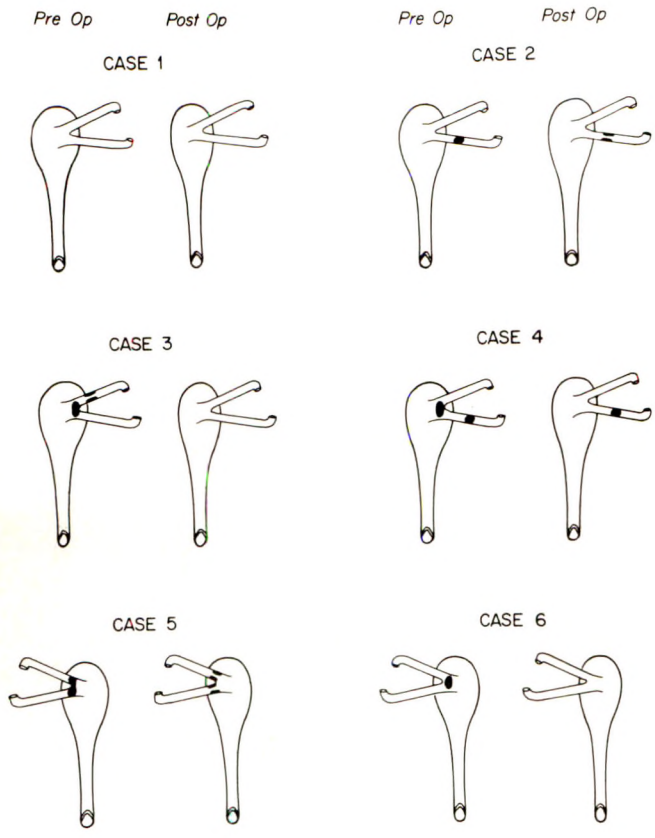


Fig. 4 (Putterman and Epstein). Preoperative and postoperative lacrimal excretory systems in cases 1 to 6. Each diagram demonstrates upper and lower puncta and canaliculi, common canaliculus, lacrimal sac, and nasolacrimal duct. Black areas within ducts demonstrate sites of obstructions.

described having a *Pseudomonas* infection of her left lacrimal sac two years previously that had been treated medically. She denied other medical problems.

The essential ocular findings included a positive result for the dye retention test of the left eye. The primary test showed no dye entering the nasal cavity. We noted a stricture 5 mm distal to the left lower punctum. The entire left upper canaliculus was partially obstructed but could be dilated. However, there was some resistance to irrigation, although dye reached the nasal cavity. No tears or discharge could be expressed by compressing the left lacrimal sac, and the nasal cavity failed to disclose any abnormalities. Results of the remainder of the ocular examination were unremarkable. A dacryocystogram disclosed an obstruction at the distal end of the left lacrimal sac (Fig. 4).

The patient underwent a combined left-sided conjunctival dacryocystorhinostomy with placement of silicone elastic tubing into the left lacrimal passages. Three months postoperatively, we removed the silicone elastic tubing. The upper canaliculus was found to be patent and irrigation through the upper system was easily accomplished. However, a persistent stricture in the lower canaliculus prohibited irrigation. Because of this, the Jones tube was retained and the patient has remained free of epiphora for the past six months (Fig. 4).

Case 3—A 48-year-old woman had had tearing of her left eye for three years. Two dacryocystorhinostomies within the previous 24 months had not relieved her epiphora.

Ocular examination disclosed a 3+ positive dye retention in the left eye; no dye entered the nose on the primary dye test. The left upper punctum and canaliculus were stenotic; however, it was possible to irrigate solution from upper to lower puncta as well as into the nasal cavity. The left lower canaliculus could be irrigated with some resistance into the nasal cavity and to a fistula located at the inferior aspect of the left nasocanthal scar from previous lacrimal surgery. Results of the remainder of the ocular examination were unremarkable (Fig. 4).

A left-sided dacryocystogram disclosed an enlarged upper lacrimal sac with two small diverticuli. The lower third of the sac appeared narrowed, but contrast material entered the nasal cavity. The 30-minute films demonstrated retention of the contrast material within the upper portion of the sac and within the diverticuli.

The patient underwent a left-sided conjunctival dacryocystorhinostomy with excision of the fistula and excision and revision of the nasocanthal scar.

Silicone elastic tubing was placed in the left lacrimal passages.

Postoperatively, the patient had several ear and nasal infections in which discharge traveled up the Jones tube and blocked its patency. Seven months postoperatively, we removed the silicone elastic tubing and easily irrigated through the upper and lower canaliculi to the nasal cavity. Two weeks later, we plugged the Jones tube. The patient was asymptomatic. We decided to leave the unplugged Jones tube in place. The canalicular system functions normally, and she has remained free of tearing for the past two years (Fig. 4).

Case 4—A 51-year-old woman reported a one-year history of recurrent tearing in her left eye. In 1966 she had had a *Streptothrix* infection of the left lower canaliculus, in which the concretions were easily expressed. In 1972 and 1976 her primary physician had probed the area, but had not relieved the epiphora.

Ocular examination disclosed a 2+ dye retention in the eye. No dye entered the nasal cavity on the primary dye test. The left lower canaliculus was obstructed at its midpoint. The upper canaliculus was stenotic distally near the common canaliculus, but irrigation through the upper system was free to the nasal cavity. No tears or discharge could be expressed with compression of the lacrimal sac, and the nasal cavity was normal. Results of the remainder of the ocular examination were unremarkable (Fig. 4).

The patient underwent a left-sided conjunctival dacryocystorhinostomy combined with silicone elastic intubation. Three months postoperatively, we removed the silicone elastic tubing. The lower canaliculus remained obstructed but the upper and common canaliculi were patent and could be irrigated freely. We decided to leave the Jones tube in place because the lower canaliculus was obstructed. The patient has remained asymptomatic for the past 14 months and has tolerated the Jones tube (Fig. 4).

Case 5—A 68-year-old woman had undergone a left-sided dacryocystectomy in 1963. In September 1977, an acutely inflamed right lacrimal sac was incised and drained. In November 1977, she underwent a right-sided dacryocystorhinostomy, but epiphora and discharge associated with a chronic right lacrimal sac infection persisted postoperatively.

We first examined the patient in December 1977. There was 2+ dye retention in the right eye and 1+ in the left. The primary dye test showed no fluorescein in the nasal cavity on either side. A right-sided common canalicular or possibly distal upper and lower canalicular obstruction was demonstrated when the canaliculi were probed. Irrigation from upper to lower canaliculi and into the nasal cavity was not possible. We noted a firm palpable mass in the right nasocanthal region in the area of the lacrimal sac. Results of the remainder of the ocular examination were unremarkable (Fig. 4).

A right-sided dacryocystogram disclosed a small amount of contrast material entering the lacrimal sac; most of the contrast material remained in the inferior cul-de-sac. The 30-minute films showed no emptying of contrast material from the lacrimal sac.

The patient underwent a right-sided conjunctival dacryocystorhinostomy with excision of a membrane obstructing the distal common canaliculus and silicone elastic intubation of the lacrimal passages. Seven months postoperatively, we removed the silicone elastic tubing. The upper and lower canaliculi had strictures at their distal ends, but irrigation without resistance was possible into the nasal cavity through each canaliculus. We plugged the Jones tube. The patient remained asymptomatic. We believed that the lacrimal passages were functioning properly. The Jones tube was unplugged and left in place. The patient has remained asymptomatic for the past 15 months and has tolerated the Jones tube well (Fig. 4).

Case 6—A 30-year-old woman complained of tearing and discharge from her right eye of eight years' duration. She had undergone two dacryocystorhinostomies. The second dacryocystorhinostomy had been performed because of recurrent symtpoms one year before our evaluation.

Ocular examination disclosed a 3+ dye retention in the right eye. No dye reached the nasal cavity on the primary dye test. Both right canaliculi were slit from the puncta to the medial canthus. By probing the canaliculi with a lacrimal needle, we found a complete obstruction at the common canaliculus. It was possible to irrigate saline solution from lower to upper canaliculi. No tears or discharge could be expressed from the canaliculi when pressure was applied against the right lacrimal sac. The right nasal cavity appeared normal. Results of the remainder of the ocular examination were unremarkable (Fig. 4).

The patient underwent a right-sided conjunctival dacryocystorhinostomy combined with silicone elastic intubation of the right lacrimal passages. Four months postoperatively, we removed the silicone elastic tubing and found that it was possible to irrigate saline through both upper and lower canaliculi into the nasal cavity. However, one week later symptoms of tearing and discharge from the right eye recurred. We found a large mucous plug obstructing the Jones tube. After the tube was carefully cleaned, it functioned normally, and irrigation through the tube to the nasal cavity was easily achieved. We decided to keep the Jones tube in place because symptoms recurred when the tube was plugged. We considered the slit canaliculi and poor lacrimal pump function to be the cause of the tearing when the Jones tube was plugged. The patient has remained asymptomatic for the past eight months. She has had no difficulty in tolerating the Jones tube (Fig. 4).

Discussion

Indications for a combined conjunctival dacryocystorhinostomy with intubation of the lacrimal passages include (1) a poor

tear pump mechanism, (2) obstruction of the upper or lower canaliculus, and (3) a common canalicular obstruction.

Quickert and Dryden advocated silicone elastic intubation of the lacrimal passages for the treatment of canalicular stenosis.[5] However, the results are unpredictable. In two of our cases (Patients 2 and 4) the intubation had not relieved the stenosis. Thus, we believed that the Jones tube accounted for the resolution of the epiphora. We avoided subsequent surgery in these cases combining a Jones tube procedure with intubation of the lacrimal system.

In the other two cases of canalicular stenosis (Patients 3 and 5), silicone elastic tubing aided in maintaining a functioning tributary. We think that an effort should be made to preserve the physiologic lacrimal system by intubation. However, a conjunctival dacryocystorhinostomy combined with the intubation enhances the success of the results.

We believed that a poor tear pump mechanism was responsible for recurrent epiphora in two of our cases (Patients 1 and 6). A conventional dacryocystorhinostomy would probably have failed in these cases. A poor tear pump mechanism should be suspected if there is disease, degeneration, or trauma to the facial nerve or orbicularis oculi muscle.

If the patient can tolerate the Jones tube, we believe that it should be left in place postoperatively, even if the normal lacrimal passages are preserved. If the Jones tube cannot be tolerated, it can be removed and the physiologic lacrimal system will function adequately. The Jones tube should be plugged mainly to determine whether the tube or the canalicular system is functioning primarily in the drainage of tears.

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REMOVAL OF HEPATITIS B SURFACE ANTIGEN FROM A CONTAMINATED APPLANATION TONOMETER

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Hepatitis B surface antigen was found in the conjunctival fluid of 42% of hepatitis B surface antigen carriers and could be detected on the tonometer tip after tonometry in one of four hepatitis B surface antigen carriers whose conjunctival fluid was positive for hepatitis B surface antigen.

The Goldmann applanation tonometer tip was contaminated by immersion into solutions containing high concentrations of hepatitis B surface antigen (average 15,000 cpm). Prompt rinsing of the contaminated tonometer in running tap water for ten seconds proved sufficient to remove all detectable hepatitis B surface antigen from the tonometer.

Darrell and Jacob[1] suggested that small breaks in the conjunctival mucous membranes may offer a means of entry for hepatitis B virus during contact-lens fitting or contact tonometry. The presence of hepatitis B surface antigen in human serum has been correlated with its presence in human tears.[1]

Abbott[2] suggested some methods of sterilizing office equipment in order to minimize the transmission of hepatitis B virus in the ophthalmologist's office. These include autoclaving, prolonged boiling, and the use of various chemicals. Corboy, Goucher, and Parnes[3] successfully removed from the applanating tip of a Goldmann tonometer a contaminating bacterial virus bacteriophage in 98% of their trials by flushing the tip with water and then drying it with a tissue. The purposes of this study were (1) to confirm the previously reported finding that tears collected from patients whose sera were positive for hepatitis B surface antigen do contain hepatitis B surface antigen; (2) to determine whether hepatitis B surface antigen can be detected on the tonometer tip after tonometry performed on carriers whose conjunctival fluid was positive for hepatitis B surface antigen; and (3) to determine whether washing the prism with running water is a practical way of removing detectable hepatitis B surface antigen from contaminated applanation tonometers.

MATERIAL AND METHODS

Detection of hepatitis B surface antigen in conjunctival fluid—We tested 31 carriers (originally found to be positive through Canadian Red Cross blood donor screenings and observed for several years) and ten normal laboratory staff members for hepatitis B surface antigen

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in serum and conjunctival fluid. We used Schirmer strips to collect the conjunctival fluid, one per eye both before and after ocular anesthesia. We allowed the conjunctival fluid to fill approximately three fourths of the strip. We placed two strips in vials containing 0.5 ml of physiologic saline and 5% human serum albumin and then tested them for blood and hepatitis B surface antigen. We used a radioimmune assay to detect hepatitis B surface antigen. This is a very sensitive test for hepatitis B surface antigen but requires a minimum of 10^9 hepatitis B surface antigen particles per milliliter of serum to be positive.[4]

Hepatitis B surface antigen on the tonometer tip in carriers with positive conjunctival fluid—We performed tonometry in four patients with positive conjunctival fluid. The tonometer tip was swabbed with a 2 × 2-cm piece of filter paper soaked in physiologic saline and then inserted into a tube containing 0.5 ml of physiologic saline. We then tested this solution for hepatitis B surface antigen.

In vitro contamination and decontamination of a tonometer—We dipped tonometers into solutions of 0.5% physiologic saline containing 5% human serum albumin and hepatitis B surface antigen to give concentrations of 15,000 cpm and 1,200 cpm. The tonometers were then rinsed for various amounts of time with physiologic saline or tap water and blotted on 2 × 2-cm pieces of blotting tissue. These papers were then immersed in 0.5 ml of physiologic saline and human serum albumin and assayed for hepatitis B surface antigen. We repeated this experiment seven times.

RESULTS

Detection of hepatitis B surface antigen in conjunctival fluid—Hepatitis B surface antigen was only detected in the conjunctival fluid of carriers who had higher titers of hepatitis B surface antigen in their sera (Table). Of these carriers, 44% were positive for hepatitis B surface antigen in conjunctival fluid before ocular anesthesia, whereas 33% were positive after ocular anesthesia. We detected no blood in any of the samples collected with Schirmer strips. We detected no hepatitis B surface antigen in the conjunctival fluid of the ten controls.

Hepatitis B surface antigen on the tonometer tip in carriers with positive

TABLE

DETECTION OF HEPATITIS B SURFACE ANTIGEN IN THE SERUM AND CONJUNCTIVAL FLUID

Hepatitis B	Carriers		Normal Staff Members
	High-Titer (>10,000 cpm)	Low-Titer (<10,000 cpm)	
No. tested	25	6	10
Serum (average cpm)	15,781	4,769	0
Conjunctival fluid*			
Without anesthetic			
No. positive	11	0	0
%	44	—	—
After anesthetic			
No. positive	8	0	0
%	33	—	—

*Levels of hepatitis B surface antigen averaged 1,005 cpm (range, 287 to 3,454 cpm).

conjunctival fluid—We detected hepatitis B surface antigen in the saline solutions into which the filter paper had been inserted after tonometry in one of four patients with positive conjunctival fluid.

In vitro contamination and decontamination of the tonometer—We detected no hepatitis B surface antigen in either rinses or blots after the tonometer was dipped in the solution containing low counts of hepatitis B surface antigen corresponding to the counts found in the conjunctival fluid. We performed the experiment seven times with the same results. However, we did detect hepatitis B surface antigen in 1-ml rinses and in the blot after two 1-ml rinses when the tonometer was dipped in the solution containing the high counts corresponding to serum counts.

Running tap water for ten seconds was sufficient to remove detectable hepatitis B surface antigen from the tonometer. We detected no hepatitis B surface antigen in the blot after the tonometer was dipped into the serum concentration and then rinsed for ten seconds. We also detected hepatitis B surface antigen in a 0.5-ml rinse (ten-minute soak) after the tonometer had been dipped into the serum concentration and allowed to stand at room temperature for 24 hours.

Discussion

We know that hepatitis virus can be transmitted by blood or blood products, even through small cuts or scratches.[5-6] Hepatitis B surface antigen has been found not only in serum but also in many other human secretions, including fluid, saliva, nasal washings, and tears.[7-10]

It is important to take all possible precautions to avoid transferring the virus from one patient to another. This study confirms the findings of others that hepatitis B surface antigen is present in the tears of a high percentage of patients with hepatitis B surface antigen in their sera. It is possible, therefore, to transmit the virus by office tonometry, particularly since we have demonstrated the presence of hepatitis B surface antigen on the tonometer after tonometry in one of four carriers with positive conjunctival fluid. Even if hepatitis B surface antigen had not been demonstrated on the tonometer, transmission might still have occurred. Gerety and associates[4] showed in chimpanzee inoculation experiments that the infecting dose (10^6 particles of hepatitis B surface antigen per milliliter of serum) may be smaller than the detectable dose as measured by radioimmune assay (10^9 particles per milliliter). Pretesting blood for transfusion for hepatitis B surface antigen by radioimmune assay reduces but does not eliminate post-transfusion hepatitis, indicating that the infecting dose may be below the detectable dose in humans as well. Our study showed that simply holding the tonometer prism under a stream of running water for ten seconds completely removed hepatitis B surface antigen even when the tonometer was highly contaminated.

Simply dipping the tonometer into a solution, or allowing it to dry overnight, may not be adequate. Removal of measurable hepatitis B surface antigen from the tonometer probably markedly reduces, if not eliminates, any danger of transmitting hepatitis B by tonometry since infectivity is related to the exposure dose.

Ophthalmologists should be aware of the possibility of transmitting hepatitis B by tears when doing contact tonometry. Particular care should be exercised if patients are known hepatitis B surface antigen carriers or are considered to be at high risk for this infection. Such high-risk patients include those undergoing renal dialysis, diabetics, drug addicts, and those who have received blood transfusions.

Because it is easy to eliminate or at least reduce the possibility of transmitting of this disease with a potentially infected applanation tonometer by holding the tonometer under running tap water for a period of ten seconds, this should become a routine procedure after every tonometric examination.

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Ophthalmic Miniature

The light was always afflictive to his eyes; he carried in his pocket something like a skeleton brass candlestick, in which, setting it on the dinner-table, between him and the most afflictive or nearest of the chief lights, he touched a little spring, and there flirted out, at the top of his brass implement, a small vertical green circle which prettily enough threw his eyes into shade, and screened him from that sorrow.

Thomas Carlyle, *Reminiscences* 1867,1881
(William Wordsworth in his Seventies)

TREATMENT OF CHRONIC OCULAR PAIN BY SELECTIVE THERMOCOAGULATION OF THE TRIGEMINAL GANGLION

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We used graded radiofrequency heating to destroy pain fibers in the ophthalmic division of the trigeminal ganglion while sparing most of the touch fibers in nine patients (seven men and two women, ranging in age from 51 to 74 years) with chronic ocular pain. All nine experienced immediate and complete pain relief. Although one patient required a second procedure after 57 months, the other eight have been pain-free for 44 to 59 months. There were no serious complications. The corneal reflex was preserved and there was no damage to the optic or extraocular muscles.

For treatment of chronic, severe ocular pain systemic analgesic drugs may be effective in some cases. However, many patients with chronic ocular pain do not obtain satisfactory relief even with narcotic agents. Retrobulbar alcohol injections may relieve pain and photophobia in potentially useful and blind eyes.[1-6] However, this relief is often temporary and the procedure is not without significant complications.[1,3,4,6] Ticho, Olshwang, and Magora[7] reported that subcutaneous electrical stimulation is useful for short-term treatment of postoperative ocular pain.

We have found that controlled thermocoagulation of the trigeminal ganglion and its rootlets provides long-term relief of chronic ocular pain.

SUBJECTS AND METHODS

Nine patients (seven men and two women, aged 51 to 74 years) with chronic ocular pain were referred to one of us (C. S. H.) for treatment. All of these patients had been treated previously with systemic analgesics but had not obtained adequate relief. Four of the patients had undergone at least one retrobulbar alcohol injection, but the relief was incomplete or temporary.

All of these patients underwent complete ophthalmologic examinations, including slit-lamp evaluation, tonometry, and ophthalmoscopy after pupillary dilatation. If opacities within the ocular media made a detailed accurate view of the fundus impossible, we completed the examination by ultrasonography. The ocular pain was attributed to glaucoma, penetrating injury, or previous retinal detachment surgery. Visual acuity in the involved eye ranged from 6/24 (20/80) to light perception.

After giving an informed consent, each patient underwent controlled thermocoagulation of the trigeminal ganglion and its rootlets ipsilateral to the painful eye.[8] This procedure was carried out in the radiographic room after intravenous injection of a fentanyl-droperidol mixture (Innovar). A hollow 20-gauge cannula-

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electrode was used to make the destructive lesion. The approach to the trigeminal ganglion was via the foramen ovale. The skin was penetrated 3 cm lateral to the labial commissure. The surgeon placed the index finger of one gloved hand in the patient's mouth, feeling the electrode as he advanced it just medial to the mandibular ramus. If a few minor changes in the electrode course did not bring it into the painful zone of decreased resistance indicative of the foramen ovale, a submentovertical radiograph was taken to aid the placement and to minimize the hazards of exploring by touch alone.

Electrical stimulation precisely located the ophthalmic division of the trigeminal ganglion. A 60-cycle square wave pulse 0.001 second in duration was used. At threshold, the sensation was not painful and, therefore, it could be located and described calmly by the alert patient. Intravenous methohexital, 20 to 50 mg, was administered before each lesion was coagulated. The first lesion was made at a setting between 47 and 65 C, administered for one minute. The heat was then increased by five- to ten-degree increments, depending on the sensory findings after each lesion. No further lesions were placed after the corneal reflex was diminished. The number of lesions required to accomplish this averaged 3.6.

The patients went home after the procedure. None required hospitalization. We examined each patient one, seven, 14, and 21 days after treatment.

Results

All nine patients experienced immediate and complete relief of their pain. One patient required a second treatment 57 months after the first because the pain recurred; he has been pain-free for nine months. The duration of pain relief in these patients ranged from 44 to 59 months (Table). No serious complications developed. One patient did develop anesthesia dolorosa that resolved spontaneously after a few months. Another patient had mild anesthesia of the face subserved by the second division of the trigeminal ganglion. The corneal reflex remained intact in all of these patients. No corneal ulcers or herpetic infections have developed.

Discussion

The treatment of prolonged, severe ocular pain can be trying for both the

TABLE

CLINICAL SUMMARY OF NINE PATIENTS TREATED WITH THERMOCOAGULATION OF TRIGEMINAL GANGLION

Patient	Age (yrs)	Cause of Pain	Visual Acuity	Length of Pain Relief (mos)	Comments
1	57	Glaucoma	6/120 (20/400)	59	Two previous retrobulbar alcohol injections
2	68	Glaucoma	L.P.	58	Developed anesthesia dolorosa
3	61	Retinal detachment	6/120 (20/400)	57	Had second treatment after 57 mos; pain-free 9 mos later
4	59	Penetrating injury	6/30 (20/100)	51	—
5	66	Glaucoma; bullous keratopathy	6/24 (20/80)	50	—
6	51	Glaucoma	6/120 (20/400)	48	—
7	59	Glaucoma	6/60 (20/200)	47	Developed mild facial anesthesia
8	74	Glaucoma	6/60 (20/200)	45	—
9	64	Glaucoma	L.P.	44	Three previous retrobulbar alcohol injections

patient and the ophthalmologist. Eyes with partial vision have been enucleated because the patients could not endure the pain they caused. Systemic analgesics are rarely successful for long-term treatment of ocular pain; additionally, these drugs are potentially addictive.

Retrobulbar alcohol injections have been used successfully in the treatment of some patients with chronic ocular pain.[1-6] Alcohol injections seem to relieve pain by interrupting transmission within the nerve fibers.[5] The proteins in the fibers are coagulated at the site of injection and the fibers degenerate peripherally from this point. If the nerve is not injected but the surrounding area is infiltrated with alcohol, the fibers may not be destroyed. The peripheral portion of the fibers usually regenerate in several months after treatment and pain may then recur.[6]

Some degree of blepharoptosis, proptosis, chemosis, and extraocular muscle palsy are common after retrobulbar alcohol injections. However, Maumanee[5] and Michels and Maumenee[6] emphasized that these findings are usually transient and do not constitute a contraindication to this form of therapy. Possible damage to the optic nerve and corneal reflex is a more serious concern.

Cordes[1] reported a case of optic atrophy that followed an attempt to inject alcohol into the sphenopalatine ganglion. This appears to be an unusual complication of retrobulbar alcohol injections. Most authors have stressed the lack of optic nerve damage with this therapy.[3,5,6] The optic nerve is apparently protected to a marked extent from the effects of alcohol by its sheath.[3] It appears that this treatment does not damage the optic nerve unless the needle is inserted into the nerve tissue.[5]

Corneal sensation is usually lost or markedly diminished after the injection of alcohol, but usually returns to normal within several weeks.[5] The loss of the corneal reflex may cause asymptomatic corneal ulcers to develop after treatment with retrobulbar alcohol. Moreover, Kornblueth[9] reported that the rate of mitosis in the epithelial and stromal cells of the cornea are reduced after retrobulbar alcohol injection.

We agree with Michels and Maumanee[6] that retrobulbar alcohol injections are relatively safe and often effective in managing otherwise uncontrolled pain in both seeing and blind eyes. The greatest limitation of this procedure is the temporary nature of the pain relief.

Selective thermocoagulation of the trigeminal ganglion and its rootlets seems to be an effective alternative method of treating chronic ocular pain. This is now a standard treatment for some forms of facial pain and should be considered in some cases of chronic ocular pain. Only one of our nine patients required a second treatment and all of the patients were free of pain after 44 to 59 months.

The procedure does not require hospitalization or the use of an operating room. Because the treatment is confined to the trigeminal ganglion, there is no risk of even temporary damage to the optic nerve or extraocular muscles. The most attractive feature of this procedure is the preservation of the corneal reflex despite complete analgesia within the ophthalmic division of the trigeminal nerve.

The selective ability to destroy pain fibers without altering most touch fibers is based upon several experimental observations. Animal and human experiments have disclosed that impulses signalling noxious stimuli are conducted in the smaller fibers of the A-delta and C groups (2.5 to 4 μ and less than 2 μ in diameter, respectively). Bessou and Perl[10] demonstrated that about one half of the unmyelinated fibers in a cat's cutaneous nerve are highly responsive to either gentle mechanical or innocuous thermal

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stimuli. The remaining half of the C fibers function at high threshold, requiring noxious stimuli for activation. Zotterman[11] established that light stroking of the skin evokes activity in the A-delta fibers of the cat's cutaneous nerve. Moreover, Maruhashi, Mitzuguchi, and Tasaui[12] proved that touch activates the largest fibers in the cutaneous nerves in the A-alpha-beta range from 8 to 14 μ in diameter in the cat.

The destruction of fibers concerned with pain while those subserving touch remain intact correlates well with the work of Letcher and Goldring.[13] They photographed the compound action potential in the saphenous nerve of the cat after graded heating with radiofrequency current or saline. They found that conduction in both the A-delta and C fibers stopped before that in the A-alpha-beta group. Thus, the relatively easy differential production of analgesia in trigeminal zones in man is associated with lesions of the smaller myelinated and unmyelinated fibers concerned with noxious stimuli. Conversely, the large A-alpha-beta fibers subserving touch can remain intact, perhaps because their heavier myelin sheaths protect them from the heat.

Preserving the corneal reflex minimizes the risk of corneal ulcers after treatment. We have found no changes in corneal structure or function after this procedure. Thus, the advantages of this procedure are the long-term relief of pain from a single treatment and the maintenance of a normal corneal reflex even in patients with total analgesia.

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SERUM CHOLINESTERASE, SERUM LIPASE, AND SERUM AMYLASE LEVELS DURING LONG-TERM ECHOTHIOPHATE IODIDE THERAPY

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Previous studies have found increased pancreatic intraductal pressures in dogs and the development of acute pancreatitis in humans after cholinergic stimulation. However, we did not find abnormally high levels of serum lipase or serum amylase in 44 patients with glaucoma using therapeutic doses of echothiophate iodide for intraocular pressure control on a long-term basis.

In 1979, Dressel and associates[1] described a patient who developed acute and severe pancreatitis as a result of anticholinesterase insecticide intoxication. Additionally, they demonstrated significantly increased pancreatic intraductal pressure and serum amylase and serum lipase levels in dogs after an intravenous bolus of this same anticholinesterase. Further studies[2] correlated depression of pseudocholinesterase in dogs with a statistically significant increase in pancreatic intraductal pressure. Others have also described pancreatic alterations with parasympathomimetic agents.[3,4] Because acelycholinesterase inhibitors are widely used in ophthalmology, we investigated the serum levels of cholinesterase, amylase, and lipase in 44 patients receiving long-term treatment with echothiophate iodide for glaucoma.

MATERIAL AND METHODS

We selected 44 consecutive patients from a glaucoma clinic who had been using topical echothiophate drops for at least one month; all were adults. Of the 44 patients, 38 had been on the same therapeutic regimen for their glaucoma for more than one year. The patients formed four groups based on the concentration of the echothiophate solution they were using and whether one or both eyes were being treated. All drops were used twice daily. No patient was using any cholinesterase inhibitor other than echothiophate. Twenty-one patients were using a 0.25% solution in both eyes, 14 were using a 0.25% solution in only one eye, eight were using a 0.125% solution in only one eye, and one patient was using a 0.0625% solution in one eye. Blood samples were drawn at the time of

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the patients' appointments, and serum lipase, serum amylase, and serum cholinesterase were measured. The cholinesterase levels were determined by the method of Ellman and associates.[5] We determined the mean values of the lipase, amylase, and cholinesterase, along with the standard errors, for the groups and compared them with normal values by Student's *t*-test (one-tailed). Additionally, using linear regression analysis, we attempted to correlate serum cholinesterase activity with serum amylase levels and serum cholinesterase with serum lipase levels for all 44 patients as a group.

Results

The amylase and lipase data for each group were not significantly different from those of a normal population ($P > .05$) (Table). The mean value for serum cholinesterase for the group receiving 0.25% echothiophate in one eye was not significantly different from that of the group receiving 0.25% in both eyes ($P > .05$). There was a significant difference in the cholinesterase level between the group receiving 0.25% echothiophate in one eye and that of the group receiving 0.125% echothiophate in one eye (P/.03). The normal serum cholinesterase level is 3,472 ± 868 units/liter. Therefore, those receiving 0.25% echothiophate iodide in one or both eyes had a cholinesterase level that was 45% of the mean level for a normal population. The group using a 0.125% drop in only one eye showed a cholinesterase level that was 80% of the normal mean level. There was only one patient using a 0.0625% solution, so comparisons could not be made with respect to him.

The linear regression analysis of all the data, plotting serum cholinesterase (P) against serum amylase (A) and then against serum lipase (L), failed to show any significant linear relationship ($r = .087$ P/A; $r = .233$ P/L).

Discussion

Cholinesterase of whole blood is present in two forms. One of these is a membrane-bound form associated with the erythrocyte cell wall, called acetylcholinesterase, and the other is a soluble form found in serum, called butyrylcholinesterase or pseudocholinesterase.[6] Although the physiologic function of pseudocholinesterase is unknown, its importance has been established because of

TABLE

SERUM CHOLINESTERASE, AMYLASE, AND LIPASE LEVELS DURING LONG-TERM ECHOTHIOPHATE IODIDE THERAPY

Group	No. of Patients	Serum Cholinesterase (mU/ml)	Serum Amylase (U/ml)	Serum Lipase (U/ml)
Normal	—	2,604–4,340	4–25	<2.0
0.25% echothiophate in both eyes twice daily	21	1,482 ± 161	18 ± 2	1.2 ± 0.1
0.125% echothiophate in one eye twice daily	14	1,669 ± 187	16 ± 2	1.2 ± 0.1
0.0625% echothiophate in one eye twice daily	8	2,782 ± 508	20 ± 4	1.1 ± 2

its action upon succinylcholine, a commonly used anesthetic agent. Succinylcholine is deactivated by pseudocholinesterase through enzymatic hydrolysis. If serum cholinesterase levels are low, the pharmacologic effects of succinylcholine, including apnea, are prolonged.[7]

The depression of blood cholinesterase activity by topically applied echothiophate iodide has been previously documented. De Roetth and associates[8] showed that the blood cholinesterase levels decrease and reach equilibrium three to four weeks after beginning twice-daily echothiophate therapy in both eyes. They found a 75% to 80% enzyme inhibition in the plasma at equilibrium with a 0.25% echothiophate iodide solution. Our data suggest only a 55% inhibition compared to the normal population with 0.25% drops given in one or both eyes. We did not measure pretreatment plasma cholinesterase levels, but de Roetth and associates[9] found no significant difference between the pseudocholinesterase levels of a normal population and those of a population with glaucoma but not taking anticholinesterase medication.

Dressel and associates[2] showed that substantially decreased plasma cholinesterase activity, at least in dogs, is directly related to increased pancreatic intraductal pressure and suggested that this may be a factor in the pathogenesis of pancreatitis. In our patients, we were unable to correlate reduced serum cholinesterase levels with an increase in serum amylase or lipase. Additionally, none of our groups had mean amylase or lipase values outside of the normal range.

It is possible that serum cholinesterase levels are not sufficiently depressed on standard clinical doses of echothiophate to show significant effects on the pancreas in humans. In canine experiments, intraductal pancreatic pressure began to increase only when the serum cholinesterase activity was inhibited 67% or more.

We performed a brief experiment with a single dog to determine if topical medication produced any pancreatic enzyme elevation. A 15-kg experimental class III dog was given a 1% solution of echothiophate iodide twice a day in both eyes. Serum amylase, lipase, and cholinesterase levels were drawn on two successive days before treatment and at three-day intervals after treatment was begun. After six days of topical echothiophate, the dog's pseudocholinesterase activity was 98% inhibited, the serum amylase was virtually unchanged, and the serum lipase level had increased more than four-fold. The dog died three days later; autolytic changes prevented a meaningful interpretation of pancreatic pathologic abnormalities.

Our clinical data showed that therapeutic dosages of echothiophate iodide do not increase serum pancreatic enzymes above normal levels. The canine pancreas may be more sensitive to cholinergic stimulation than the human pancreas. However, the report of acute pancreatitis in a patient after poisoning with a cholinesterase inhibitor suggested that pancreatitis may occur in humans at higher doses or in susceptible patients. To study this possibility further, we recommend evaluating pancreatic enzymes in patients who develop gastrointestinal symptoms while taking echothiophate iodide.

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OPHTHALMIC MINIATURE

Locke's account of perception is in large part simply a transcription of the doctrine set forth in contemporary scientific text-books. The sense-organs, it was supposed, in perception were mechanically stimulated, either by actual contact with an "external object" or, as in vision, by "insensible particles" emitted or reflected from it: this mechanical stimulus was then transmitted, through the appropriate nerves, to the brain. But when the stimulus reaches the brain something quite different happens; the mechanical cause at this stage produces a non-mechanical effect, namely the occurrence "in the mind" of what Locke calls an "idea," or, more fully, an "idea of sensation." But—and this is a crucial point—in Locke's view to have an idea in one's mind is not, as perhaps one might expect, to be made aware of that object in the world from which the original stimulus emanated; to have an idea is to be aware of the idea; and it is an essential feature of Locke's doctrine that, strictly speaking, we are directly aware only of ideas in our own minds. These mental entities, the only objects of which we are directly aware, "represent" to us those external objects from whose operations they are supposed to originate: of external objects themselves we cannot be directly aware.

George Berkeley,
The Principles of Human Knowledge
Three Dialogues between Hylas and Philonous
Glasgow, Wm. Collins Sons & Co., Ltd., 1979

NOTES, CASES AND INSTRUMENTS

PREVENTION OF CORNEAL BURNS DURING HIGH-ENERGY LASER IRIDOTOMY

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We used a plano hydrophilic bandage contact lens to protect the corneal epithelium and stroma from burns during laser iridotomy. The reduction of energy losses with this method, by the avoidance of corneal epithelial opacities, increases the chance of successful iridotomy when many applications of the laser are needed.

Photocoagulation of the iris has been used for more than 20 years.[1] Experimental iris burns with the laser were first reported in 1964.[2] Since that time, there have been many published reports on the clinical use of the laser in the anterior segment. High levels of irradiance or energy density are required to achieve perforation of the iris with the continuous argon laser. Complications of the high-energy applications are corneal epithelial coagulation and stromal burns.[3] This led us to investigate the use of a common soft contact lens to prevent these burns without loss of energy density delivered to the iris.

MATERIAL AND METHODS

We determined the threshold for a corneal burn for a patient with closed-angle glaucoma using a continuous-wave argon laser, set at 100-μ spot size and one-second applications during attempted iridotomy. We then placed a plano hydrophilic bandage contact lens on the cornea and used similar energy levels to produce burns elsewhere on the iris in the same eye. We later performed laser iridotomy on the second eye of the patient, using the same soft contact lens.

RESULTS

Dense epithelial coagulative lesions occurred at 650 mW (measured at the cornea) for one second, with an irradiance of approximately 8,276 watts/cm^2). The burns coagulated the epithelium, and one burn penetrated to a depth of one third of the corneal stroma, with charring of its edges. These corneal burns prevented full perforation of the iris.

With the plano hydrophilic contact lens in place, no epithelial coagulation occurred up to settings of 700 mW measured at the cornea for one second, with the 100-μ spot in the same eye (irradiance was approximately 8,912 watts/cm^2). A few fine endothelial opacities occurred but these were not visible 24 hours later. A small iridotomy was achieved. In the second eye, no corneal burns occurred at 650 mW, used for one-half second with the 100-μ spot (8,276 watts/cm^2). A large iridotomy was achieved with many 50-μ spots at 525 mW (approximately 26,738 watts/cm^2) for one-half second, also without corneal burns.

DISCUSSION

Corneal epithelial coagulation prevents further laser applications by absorption and reflection from the corneal surface. This is a serious impediment to iridotomy, which often requires many high-energy applications. Some argon lasers have only a medium output, in which case

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transcorneal photocoagulation without a contact lens has been recommended in the past.[4] Special antireflective coated contact lenses have also been suggested as a protection against corneal burns.[3] Increased irradiance may be obtained by including a small convex lens on the surface of the anterior segment contact lens.[5] The special lenses, however, are expensive and not readily available. Loss of energy occurs with the standard anterior segment contact lenses, which do not have antireflective coatings. From our data there was no apparent loss of irradiance through a thin hydrophilic bandage lens. The burn threshold for bare epithelium and stroma was 8,276 watts/cm^2 for one second. With the soft contact lens in place, no epithelial burns occurred at the higher power required to produce a small iridotomy (8,912 watts/cm^2 for one second). Furthermore, increasing the power to 26,738 watts/cm^2 by using the 50-μ spot for one-half second with the soft contact lens in place resulted in a large iridotomy without corneal burns.

One explanation for this is that water retained in the contact lens (38.6% water and 61.4% methylmethacrylate) and in the tear film under the lens acted as a heat sink, conducting heat away from the epithelium and cornea.[5] Another explanation is that the contact lens prevented tear film evaporation. Without the lens, the epithelium may dry out while the eyelids are kept open. The dehydrated epithelium may lose its normal light transmission characteristics, and absorb more energy from the laser.

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ANTERIOR UVEITIS IN KAWASAKI'S DISEASE

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Kawasaki's disease (mucocutaneous lymph node syndrome) is an acute febrile illness primarily affecting children. Slit-lamp examinations of six children with Kawasaki's disease, ranging in age from 22 months to 16 years, showed that five had anterior uveitis during the acute phase of the illness. Two of the children were treated with corticosteroids and cycloplegic drugs and three received no treatment. In all five, the anterior uveitis resolved completely within a few weeks.

Kawasaki's disease is an acute febrile illness of unknown origin that primarily

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affects children. The illness is typically characterized by the following major features[1-4]: (1) fever lasting five days or more; (2) bilateral conjunctival injection; (3) changes of the lips and oral cavity (such as dry, cracked lips; glowing red lips; hypertrophy of tongue papillae with a strawberry tongue appearance; diffuse reddening of the oropharyngeal mucosa); (4) changes of the peripheral extremities (such as reddening of the palms or soles; edema of the hands or feet; generalized desquamation; desquamation from the fingertips); (5) polymorphous exanthem; and (6) acute nonpurulent cervical lymph node swelling.

The Japan Mucocutaneous Lymph Node Syndrome Research Committee has stated that at least five of these six features should be present for a diagnosis of Kawasaki's disease.[3] The Center for Disease Control has stated that the presence of one of the findings listed for features 3 or 4 is sufficient to establish that particular feature.[4]

Bilateral conjunctival injection occurs in 88% to 96% of children with Kawasaki's disease.[1,4] Characteristically, there is discrete engorgement of the bulbar conjunctival vessels, with much less severe involvement of the palpebral conjunctiva. Typically, there is no conjunctival edema, discharge, purulent drainage, pseudomembrane formation, or corneal ulceration.[2]

Recently, Germain and associates[5] reported the occurrence of anterior uveitis in Kawasaki's disease. Their report described two patients who had transient, mild anterior uveitis during the acute phase of the illness. Recently, we began a prospective study of the incidence and nature of uveitis in Kawasaki's disease. We now report our findings to date.

Subjects and Methods

Beginning in December 1979, one of us (M. J. B.) performed slit-lamp examinations on all new patients with Kawasaki's disease examined here. A diagnosis of Kawasaki's disease was not made unless at least five of the six diagnostic criteria were fulfilled. To date, we have prospectively studied six such patients, all during the acute phase of their illness.

Case Reports

Case 1—A 16-year-old girl who had Kawasaki's disease associated with arthritis and angiographically demonstrated coronary artery aneurysms underwent slit-lamp examination on the 16th day of her illness. The examination disclosed mild, bilateral conjunctival injection, 1+ flare and cells in the right eye, 2+ flare and cells in the left eye, and numerous, minute keratic precipitates in both eyes. Although asymptomatic, she was treated with topical corticosteroids and cycloplegic drugs in tapering dosages for a six-week period. Two weeks after treatment was begun, the uveitis cleared markedly. It was completely resolved within two months after the onset of Kawasaki's disease.

Case 2—An 8-year-old boy with Kawasaki's disease underwent slit-lamp examination on the 13th day of his illness. The examination disclosed mild conjunctival injection, mild ciliary injection, and only trace cells in each eye. No treatment was instituted. A second ophthalmologic examination 35 days after the onset of his illness gave normal findings.

Case 3—A 6½-year-old girl with Kawasaki's disease underwent slit-lamp examination on the 19th day of her illness. The examination disclosed mild bilateral conjunctival and ciliary injection, trace flare without cells in the right eye, 1+ cells and trace flare in the left eye, and five to ten keratic precipitates over the inferior third of each cornea. Although asymptomatic, she was treated with topical corticosteroids and cycloplegic drugs in tapering dosages for a three-week period. Within four weeks after the onset of Kawasaki's disease, the uveitis was completely resolved.

Case 4—A 3⅔-year-old boy with Kawasaki's disease underwent slit-lamp examination on the tenth day of his illness. Although he had had mild photophobia and had had moderately severe bilateral conjunctival injection during the first week of his illness, there was no conjunctival or ciliary injection at the time of his slit-lamp examination, which disclosed only rare cells bilaterally. No treatment was instituted. A second slit-lamp examination two weeks later gave normal results.

Case 5—A 2¼-year-old girl with Kawasaki's disease underwent slit-lamp examination on the 11th day of her illness. Although she had mild, resolving bilateral conjunctival injection at that time, there was no evidence of uveitis.

Case 6—A 22-month-old boy with Kawasaki's disease underwent slit-lamp examination on the eighth day of his illness. Although he had had moderately severe bilateral conjunctival injection

during the first week of his illness, there was no conjunctival or ciliary injection at the time of the slit-lamp examination, which disclosed rare cells in the left eye only. He received no treatment, and a second slit-lamp examination two weeks later gave normal results.

Discussion

All six patients in our prospective study fulfilled at least five of the diagnostic criteria for Kawasaki's disease, and one of the patients developed coronary artery aneurysms, which can occur in this disease.[6,7] All six had mild bilateral conjunctival injection during at least the first week of the illness. None noted pain, discharge, or impaired visual acuity. Two had mild photophobia. All six underwent slit-lamp examination during the acute phase of the illness.

Five of the six children had evidence of anterior uveitis. In each instance the uveitis was mild. Two children had keratic precipitates with flare and cells. These two patients were treated with topical corticosteroids and cycloplegic drugs. The other three children had only rare to trace flare or cells without keratic precipitates. These three were not treated. In all five instances, the uveitis fully resolved within two to eight weeks after the onset of the disease.

Our report confirms the recent observations of Germain and associates[5] that mild anterior uveitis can occur in Kawasaki's disease. Furthermore, our prospective series suggests that it may occur frequently. We recommend that slit-lamp examinations be performed during the acute phase of the illness to determine the incidence and significance of anterior uveitis in Kawasaki's disease.

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FOLLOW-UP OPHTHALMOLOGIC EXAMINATIONS IN CHILDREN WITH KAWASAKI'S DISEASE

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We recalled 15 patients who had had Kawasaki's disease with documented bilateral conjunctival injection but who had not undergone slit-lamp examinations during the acute phase of the illness. Although anterior uveitis has been found in the acute phase of Kawasaki's disease, results of the fol-

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low-up studies (including slit-lamp examination, visual acuity testing, and assessment of pupillary reaction, muscle balance, and intraocular pressure) were normal in all 15 children.

Bilateral conjunctival injection occurs in 88% to 96% of children with Kawasaki's disease,[1,2] and is one of the six major diagnostic criteria established by the Japan Mucocutaneous Lymph Node Syndrome Research Committee.[3] Recently, Germain and associates[4] reported that mild anterior uveitis may occur in Kawasaki's disease. In an ongoing prospective study of our own,[5] we documented anterior uveitis during the acute phase of the illness in five of six children. In all five instances, the uveitis was mild and it resolved promptly and completely without sequelae and without recurrence. In three of the five instances this complete resolution occurred without treatment.

This preliminary evidence that mild anterior uveitis may be a common feature of Kawasaki's disease prompted us to study a group of children who had had Kawasaki's disease previously, but who had not undergone slit-lamp examination during the acute phase of their illness. Our purpose was to determine whether any children had evidence of past or present anterior uveitis months after the illness had resolved.

Subjects and Methods

All children with Kawasaki's disease who had been examined here between November 1978 and September 1980 were recalled for ophthalmologic examination. The group consisted of 16 patients; all had fulfilled at least five of the six diagnostic criteria for Kawasaki's disease.[3,5] Although all 16 patients had had documented bilateral conjunctival injection during the acute phase of their illness, none had undergone slit-lamp examination or taken ophthalmologic medications. Fifteen of the 16 returned for examination; the 16th lived too far away to return conveniently. The ophthalmologic examination, performed by one of us (M. J. B.) consisted of visual acuity testing, slit-lamp examination, and assessment of pupillary reaction, muscle balance, and intraocular pressures. We also obtained a history of the period between the onset of the disease and the recall.

Results

For the 15 children who returned, the mean age at which Kawasaki's disease had occurred was 5.13 years (range, 0.5 to 14.2 years). The time interval between the onset of Kawasaki's disease and the follow-up ophthalmologic examination ranged from six months to 24 months (mean, 15.3 months). Twelve of the 15 had experienced no ocular symptoms since their acute illness had resolved. Three of the 15 had experienced brief, mild ocular redness during an upper respiratory tract infection.

All 15 patients had normal visual acuities and no evidence of synechiae, glaucoma, or recurrent anterior uveitis.

Discussion

An ongoing prospective study of Kawasaki disease at our institution has suggested that mild anterior uveitis may be a common feature during the acute phase of the illness.[5] Because none of the 15 children in the present study had undergone a slit-lamp examination at the time of the acute illness, we do not know whether any initially had anterior uveitis. In any case, results of follow-up ophthalmologic examinations were normal in all 15 children. Although our studies suggested that the anterior uveitis associated with Kawasaki's disease is usually mild and probably self-limited, continued prospective documentation is necessary.

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MODIFIED ZEISS BEAM SPLITTER FOR OPERATING MICROSCOPE AND TELEVISION CAMERA

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I have designed an asymmetrical 50/70 beam splitter that allows a television camera to be coupled directly to the Zeiss operating microscope. This device provides more light for the surgeon.

The usefulness of television cameras coupled to an operating microscope[1] has been limited because the sensitivity of most color cameras on the market is lower than that of the human eye, and, therefore, they demand more light than the eye of the surgeon. Parel and associates[2] solved the problem by replacing the ordinary 50 beam splitter of the Zeiss operating microscope with a 70 beam splitter. With the latter, each of the two reflecting prisms transmits 12.5% of the light to the surgeon and reflects 87.5% to the television camera and to the assistant (Carl Zeiss, personal communication). The loss of light for the operator is considerable. With the 50 beam splitter, for example, the transmission of these prisms amounts to 42.85% and the reflection to 57.15%. This difference is particularly noticeable when the illumination is barely sufficient, as in the case of a vitrectomy.

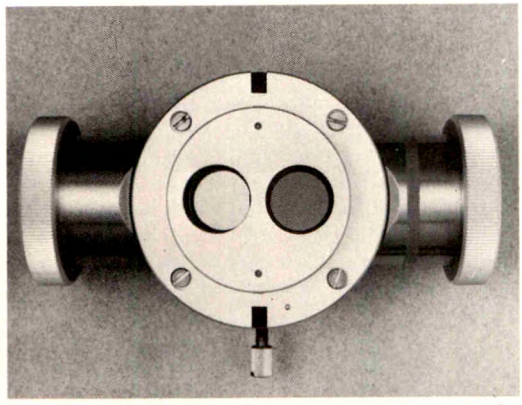


Figure (Gonvers). Light transmission between the right and the left optic systems of the 50/70 beam splitter is asymmetrical.

I have overcome this inconvenience by combining a 70 beam splitter with a 50 beam splitter (Figure). The reflecting prism of the 70 beam splitter is placed on the side of the camera connection, whereas the reflecting prism of the 50 beam splitter is placed on the assistant's side. Theoretically, this set-up is a disadvantage to the surgeon because one eye receives more light than the other when he looks through an asymmetrical optic system. In practice, this difference is hardly noticed by the surgeon who prefers a greater degree of light even if its distribution is asymmetrical.

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WHICH TYPE OF SALINE SOLUTION FOR SOFT CONTACT LENSES IS BEST?

Several published reports have described severe corneal ulcerative bacterial infections in wearers of soft lenses.[1-5] Important underlying factors in this uncommon but serious adverse event are bacterial contamination of the saline solutions used to rinse and store these lenses and the failure of some wearers to follow disinfection procedures correctly.[5,6]

In late 1978 the major manufacturer of the 250-mg sodium chloride tablets used to prepare these solutions withdrew its tablets from the marketplace at the request of the Food and Drug Administration. The reason for this market withdrawal was concern that the unpreserved solutions prepared from those tablets could become contaminated with bacteria, particularly if stored for longer than a day or if prepared from contaminated distilled water. As salt tablets disappeared from the marketplace, consumers were forced to switch to premixed thimerosal-containing saline solutions or unpreserved sterile unit-dose solutions.

A controversy then developed over the withdrawal of salt tablets. The Food and Drug Administration and some physicians felt that the salt tablet technique carried an unwarranted risk, at least at that time, of producing corneal infections. Conversely, others believed that solutions prepared from salt tablets did not carry a significant risk if used correctly and that consumer choice should not be limited to premixed solutions; the latter are more expensive and not infrequently cause eye irritation in patients who are sensitive to thimerosal.

After reviewing the situation with an advisory committee, the Food and Drug Administration again approved salt tablets in mid-1980, but this time in a kit intended to eliminate the concerns of the past. This kit contains 135-mg sodium chloride tablets and a plastic vial suitable for preparing only 15 ml (a one-day supply) of saline; a detailed consumer brochure emphasizing proper technique is also included. (The 250-mg sodium chloride tablets still offered by a few suppliers are not approved.)

The controversy over saline solutions for soft lenses has been detailed in the lay press and has been the subject of two recent Congressional hearings.[7,8] Regrettably, an interim report from the National Registry of Drug-Induced Ocular Side Effects[9] contributed to this controversy

and should to be corrected. This report stated that 201 cases of corneal infections in users of the salt tablet technique had been reported to the Registry. This was subsequently cited in promotional information sent to physicians by a manufacturer as evidence of the risk of salt tablets. Other manufacturers and the House Subcommittee on Oversight and Investigations[7,8] then objected that the information on salt tablets in the previous report was not based on well-documented case reports submitted in the usual manner to the Registry. This objection is well taken. In fact, 196 of the cases in the report from the Registry were oral descriptions of corneal infections collected by a single ophthalmologist from conversations with colleagues. The other five cases were reported independently to the Registry.

Surveys by clinical investigators are valuable as scientific information in their own right. However, from this experience the Registry learned that the risk of introducing epidemiologic bias into the Registry is great if information collected from an informal survey is entered into the general data file. The 196 cases have thus been removed from the Registry's file. Registries are most useful when they collect well-documented case reports from a broad population of practicing physicians. Timely reports of this type can disclose trends and provide information that is at least suggestive on the comparative risks of different products.

The previous report from the Registry should therefore be taken only as an alert about the possibility of corneal infections in wearers of soft contact lenses using solutions prepared with the older type (250-mg) of salt tablets. Because new salt tablet kits have now replaced the older tablets, this alert may well not be relevant to the current marketplace.

Request for case reports of severe corneal infections in wearers of soft contact lenses—The key question is whether the newer solutions now being used carry a significant risk of producing severe corneal infections or ulcers in wearers of soft contact lenses and, if so, whether the risk among them is meaningfully different. No culturally proven cases of severe bacterial infection or corneal ulcers have yet been reported to the Registry in users of the new salt tablet kits or preserved premixed saline or unpreserved sterile saline, but this may be because the Registry has not focused in the past on over-the-counter products. If such cases are occurring, however, now is the ideal time to report them. The three types of products in the marketplace are all relatively new. They are each used widely enough that some idea of their comparative risk might be gained if reported cases were well-documented and the reporting unbiased with respect to the type of solution used in each case.

The National Registry for Drug-Induced Ocular Side Effects therefore invites all ophthalmologists to help answer the question posed in the title. Any case of severe bacterial infection or corneal ulcer in an individual who wears soft contact lenses should be reported on the form (FD 1639a) previously supplied by the Registry. Both the infected eye and the solution used by the wearer should be cultured if possible. It is essential that the type of solution or brand name, or both, be reported. Names of patients, practitioners, and any geographical identifiers are protected from public disclosure.

The Registry will summarize the data received and report the results at a later date.

FREDERICK T. FRAUNFELDER
Executive Director,
National Registry for Drug-Induced Ocular Side Effects
J. RICHARD CROUT
Director,
Bureau of Drugs,
Food and Drug Administration

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8. Hearing before the Subcommittee on Oversight and Investigations of the Committee on Interstate and Foreign Commerce, House of Representatives, 96th Congress, Dec. 12, 1980.
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CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Centenary of Credé Prophylaxis

Editor:

I would like to offer several comments on the editorial (Am. J. Ophthalmol. 90:874, 1980) marking the centenary of Credé prophylaxis against ophthalmia neonatorum. As the editorial noted, the revision of the recommendations of the American Academy of Pediatrics and the Center for Disease Control to include topical erythromycin or tetracycline for the prevention of gonococcal conjunctivitis and the recognition that *Chlamydia trachomatis* is currently the most common identifiable cause of neonatal conjunctivitis make it imperative that the efficacy of various prophylactic regimens against chlamydial infections be evaluated.

The efficacy of topical silver nitrate is unknown but is probably minimal. Although 0.1% to 1% silver nitrate protected embryonated eggs from lethal infection with a trachoma strain of *Chlamydia*,[1] there are no data from control studies supporting its efficacy *in vivo*. Most infants born to mothers with chlamydial colonization of the cervix have received traditional Credé prophylaxis. However, cohort studies have shown that 21.5%[2] to 44%[3] of such infants develop conjunctival colonization with *C. trachomatis*, with more than 50% of these infants exhibiting clinical conjunctivitis.

Hammerschlag and associates[4] have recently shown that topical application of 1% erythromycin ointment protects against conjunctival infection (0 of 24 infants) but not against nasopharyngeal colonization (five of 24 infants) or subsequent pneumonia. We (P. Patamasucon, P. Rettig, and J. D. Nelson, unpublished data) observed 36 infants with culture-positive chlamydial eye infection who had received topical tetracycline, a number equivalent to the 30 neonates who received intramuscular penicillin for postnatal gonococcal prophylaxis at Parkland Memorial Hospital, Dallas.[5] These findings suggest that single-dose postnatal application of tetracycline is no more effective than parenteral penicillin, an agent of at best moderate in vitro and in vivo potency against *C. trachomatis*. The explanation of the apparent discrepancy between topical erythromycin and tetracycline in this setting remains unknown. These agents have otherwise exhibited equivalent laboratory and clinical antichlamydial effectiveness.

The widespread implementation of any form of antimicrobial prophylaxis against neonatal chlamydial infection requires the design of a regimen which will prevent ocular and extraocular colonization and awaits the development of rapid, sensitive, and inexpensive methods of identifying mothers and infants at risk.

PHILIP J. RETTIG, M.D.
Oklahoma City, Oklahoma

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Unilateral Rieger's Anomaly

Editor:

In their article, "Unilateral Rieger's anomaly" (Am. J. Ophthalmol. 90:725, 1980), W. E. Bundy III, P. L. Kaufman, G. A. Stainer, and J. C. Prensky stated that their patient was the first with unilateral Rieger's anomaly to be described. However, I described a patient with similar findings in 1977.[1]

KHALID J. AWAN, M.D.
Norton, Virginia

REFERENCES

1. Awan, K. J.: Peters-Rieger's syndrome. J. Pediatr. Ophthalmol. 14:112, 1977.

Reply

Editor:

The patient described by Dr. Awan exhibited several of the findings of Peters' anomaly, including a central corneal leukoma and an anterior polar cataract, as well as those of Rieger's anomaly. Our patient exhibited only those findings characteristic of Rieger's anomaly. We appreciate Dr. Awan's calling attention to his interesting patient, but we hold to our contention that, to the best our knowledge, our patient represented the first reported instance of pure Rieger's anomaly occurring unilaterally.

PAUL L. KAUFMAN, M.D.
Madison, Wisconsin

BOOK REVIEWS

1980 Year Book of Ophthalmology. Edited by William F. Hughes. Chicago, Year Book Medical Publishers, 1980. Hardcover, 374 pages, index, 66 black and white figures. $31.50

The 1980 Year Book of Ophthalmology provides the reader with the 44 key questions of the year, a selection of brief reviews, and pertinent articles from ophthalmic and scientific publications. Representative selections from Hughes' "current literature quiz" include: "How should one manage a capillary hemangioma of the lids in infants?" "Is timolol a miracle drug?" "Should we correct refractive errors surgically?"

Each of the 14 sections begins with brief reviews written by authors expert in these areas. The editor has provided a representative selection of articles from current American ophthalmologic journals and some foreign publications. Although fewer in number, there are articles from related medical fields. The editor has done an admirable job in selecting and organizing clinically relevant and laboratory investigative articles. Articles on current topics such as refractive keratoplasty, extracapsular cataract surgery, intraocular lens implantation, extended-wear contact lenses, glaucoma therapy, HLA antigens, and ocular diseases are reviewed. The illustrations are appropriately selected and reproduced. The editor's discerning comments are interspersed throughout the text. His sagacious remarks reflect the broad range of Hughes' knowledge.

The last section of the book refers the reader to references for the 44 key questions of the year. In ophthalmology, as in

life, the questions remain the same; it is only the answers that change with time. The 1980 Year Book of Ophthalmology is an excellent survey of the ophthalmologic field.

ZIBUTE ZAPARACKAS

Ptosis, 3rd ed. By Crowell Beard. St. Louis, C. V. Mosby Co., 1981. Hardcover, 276 pages, index, 230 black and white figures. $44.50

The third edition of Crowell Beard's book continues to be the most comprehensive text on the subject of blepharoptosis. Because it is so well written and illustrated, the book precisely explains the anatomy, pathology, diagnosis, and surgical management of blepharoptosis. The text enables the reader to understand the different types of blepharoptosis and the preferred techniques for treating them.

This third edition adds several new blepharoptosis procedures, including Small's A-frame technique, Singh and Singh's superior rectus muscle transplantation, and the Collin-Beard operation to shorten the levator palpebrae superioris muscle. Whereas superior rectus muscle transplantation into the eyelid previously led to severe exposure keratopathy, the newer procedure seems to avoid this complication. If the long-term results of this operation prove to be good, blepharoptosis surgeons may begin using it again. The Collin-Beard operation attempts to preserve Müller's muscle in a technique similar to but simpler than those of the Jones and Mustardé procedures.

In general, the third edition is almost the same as the second. Unfortunately, some of the photographs used previously are now lighter and of poorer quality. However, one distinct advantage of the third edition is that photographs of late results are included. These help to prove that fascia lata frontalis slings provide more lasting results in severe blepharoptosis with poor levator palpebrae superioris muscle function than resection procedures do. In the initial photographs of maximum levator palpebrae superioris muscle resections the results appear to be excellent, but after five and 18 years there is a gradual recurrence. Also, the photographs comparing the results of jaw-winking blepharoptosis treated with levator resections to those managed by levator excisions and frontalis slings are convincing evidence of the superiority of slings in relieving the jaw-winking deformity. The levator resection tends to exaggerate the jaw-winking component.

I recommend this third edition of Beard's textbook to all surgeons who perform blepharoptosis surgery. If you already own the second edition, it may be difficult to justify buying the newer one for the few added procedures and photographs; however, the ease of handling this thinner and lighter (1 lb) book may convince you that the purchase is worthwhile.

ALLEN M. PUTTERMAN

Basic Anatomy for the Allied Health Professions. By Royce L. Montgomery. Baltimore, Urban and Schwarzenberg, 1980. Hardcover, 455 pages, index, approximately 417 black and white figures and 167 color figures. $19.50

This is essentially an atlas with some appended text intended "to provide a useful, appropriately-illustrated learning tool for students in introductory courses in human anatomy." The illustrations are culled from a number of German anatomical atlases and are the highlight of this volume. In the preface, the text is stated to be "specifically written for students seeking careers in nursing and allied health professions"; this may be so, but to state that it is "supplemented with easy-to-comprehend explanations" is not correct, in my opinion. The text is no more than a descriptive glossary of terms that, by and large, is a disservice to anatomists

in that this format must make anatomy boring, tedious, and uninteresting for any student. Tables with lists of muscles and columns of origins, insertions, actions, and innervations are obviously meant to be learned by rote, as nowhere in the text is there even a hint of what muscles are, how they operate, group and agonistic and antagonistic actions, and the like. Similarly, the section on the skeletal system does not mention of growth, plasticity, etc. The musculoskeletal system, for example, can hardly be understood "by students in . . . physical education." The book is only "basic" if basic means "bare minimum"; to me, basic means something else—the foundation of a general understanding of anatomical concepts, principles, and functions to which detail can be added as needed.

Each chapter begins with a number of "student objectives" and ends with some review questions phrased in such a way as to encourage memorization rather than to check the student's understanding of the material. There is also a short section at the end of each chapter labeled "clinical applications." In general, these are useless glossaries of selected clinical terms without much relevance to the text. Clinical applications are usually included in books to emphasize morphologic concepts or to highlight the value of anatomical knowledge in clinical conditions. This book fails both these tests.

There are many minor mistakes throughout the text. In Figure 2-4, for example, cuticle is spelled "cutical," while in the adjoining text the "lunula" of the nail and an invented term, "crescent," are described as separate items although they turn out to be the same thing. In Figure 5-17C, "anterior" and "posterior" should be reversed. In Figure 6-37G, "profundus nerve" should read "profundus branch, ulnar nerve" to coincide with "superficial branch, ulnar nerve" just below. Similarly, in Figure 6-39A, "genital branch" should be "genital branch of the genitofemoral nerve"; one cannot know this from the figure. In Figure 6-39D, "common peroneus muscle" (which does not exist) should read "common peroneal nerve."

I have seen "Purkinje" spelled in many ways (including the original Czech) but never as "Purkinge" (Fig. 9-9B). In the same figure, the "right branch bundle" arrow does not point to the correct structure. Similarly, in Figure 9-33A the "ductus arteriosus" pointer does not lead to the correct structure, and "ductus venosis" should be "ductus venosus." The branches of the internal thoracic artery are called perforating branches, not "perforant arteries" (Fig. 9-11D). Again, in Figure 9-30B, "popliteal vein" incorrectly labels a minor communicating vein, and in Figure 9-31A, "superficial" should be added to "inguinal lymph nodes" because the superficial inguinal and superficial subinguinal nodes are subgroups of the inguinal lymph nodes. The text does not mention the important communications between the superficial and deep veins of the leg; in Figure 9-31B, the illustration displays these channels but they are not labeled. The author lost a good opportunity to emphasize some basic functions of veins and the importance of valves, gravity, and the pumping action of muscles. Figure 10-4 inaccurately depicts the "lateral thoracic vein" as virtually subcutaneous in the axilla. The same vein is labeled lower down as the "thoracoepigastric vein" (which is actually the anastomosis between the lateral thoracic and inferior epigastric veins); there is no reference to the thoracoepigastric vein in either the text or index. Again, although there are detailed illustrations (Figs. 11-6F and 11-6G) of the bronchopulmonary segments, there is no reference in the text to this important part of the "basic anatomy" of the lungs.

The atlas part of this book may be worth the price (although there are other excellent atlases selling for approximately

the same price), but the allied health professions deserve a better text.

RONALD SINGER

Diabetic Retinopathy. Clinical Evaluation and Management. By Francis A. L'Esperance, Jr., and William A. James, Jr., St. Louis, C. V. Mosby Co., 1980. Hardcover, index, 257 black and white figures. $44.50

This text provides a comprehensive review of concepts indispensable to the understanding and management of diabetic retinopathy.

The authors, with contributions from David Apple, Eli Friedman, Hunter Little, and Jesse Sigelman, have logically organized their presentation into 11 chapters: History, Epidemiology, Pathology, Pathogenesis, Classification, Medical Treatment, Pituitary Ablation, Photocoagulation, Diabetic Maculopathy, Vitrectomy, and Future Management of Diabetic Retinopathy.

A particular strength of each chapter is the review of published reports; there are 40 to 70 references per chapter.

This volume is a welcome addition to the reference library of all physicians attempting to understand the complexity of this important and growing area.

DANIEL FINKELSTEIN

SYMPOSIA

Developments in Ophthalmology. Vol. 1. Ocular Microsurgery. Edited by A. S. M. Lim. Basel, S. Karger AG, 1981. Hardcover, 96 pages, 29 black and white figures. $30

ROPER-HALL, M. J.: **Extended use of microsurgery in ophthalmology**

ROPER-HALL, M. J.: **The ideal suture**

PIERSE, D. J., AND MCSTEELE, D. G.: **Diamonds—Their development for and influence on modern microsurgery**

MISHIMA, S.: **Injuries to the iris and corneal endothelium in intraocular surgeries**

WATSON, P. G.: **Microscopes and glaucoma**

MINODA, K.: **Trabeculoiridocyclectomy of tumors of the iris and ciliary body**

BONNET, M.: **Microsurgery of retinal detachment (vitreous surgery excluded)**

WATSON, P. G.: **Trabeculectomy**

CONSTABLE, I.: **Vitrectomy in ocular trauma**

CONSTABLE, I.: **Options for visualisation in vitrectomy**

LIM, A. S. M., AND KHOO, C. Y.: **Soft cataracts—Surgical management**

LIM, A. S. M., AND ANG, B. C.: **Microsurgery of peripheral iridectomy**

Current Concepts in Cataract Surgery. Selected Proceedings of the Sixth Biennial Cataract Surgical Congress. Edited by Jared M. Emery and Adrienne C. Jacobson. St. Louis, C. V. Mosby Co., 1980. Hardcover, 466 pages, index, approximately 300 black and white figures. $64.50

Preliminary Considerations
- Introduction
- Anesthesia
- Instrumentation

Intracapsular Cataract Extraction
- General principles
- Intracapsular extraction. Wound closure technique

Extracapsular Cataract Extraction

Intraocular Lenses
- Status of intraocular lenses. Indications for intraocular lenses
- Determination of intraocular lens power
- Surgical techniques for intraocular lenses

Special Problems in Cataract Surgery
- Cataract surgery in glaucomatous eyes
- Cataract surgery combined with keratoplasty
- Pseudophakic keratoplasty
- Refractive keratoplasty
- Management of corneal astigmatism

Operative Complications
- Management of the vitreous

Postoperative Complications
- Management of complications using surgery through the pars plana
- Opacified posterior capsule
- Cystoid macular edema
- Retinal detachment
- Complications with intraocular lenses
- Miscellaneous complications

Correction in Aphakia
- Refractive correction
- Strabismus and amblyopia

Results of Cataract Surgery

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Ophthalmologica

RESPONSE OF HUMAN CORNEAL ENDOTHELIAL CELLS TO INCREASED INTRAOCULAR PRESSURE. A SPECULAR MICROSCOPIC STUDY. (Thesis) Setälä, K. (Dept. Ophthalmol., Univ. Helsinki, Helsinki, Finland). Acta Ophthalmol., suppl. 144, 1980.

In the first portion of this study endothelial cell counts were done in patients who had uniocular capsular glaucoma. Of 27 such patients, 15 had a lower cell density in the affected eye. Ten patients had the same cell density in both eyes and only two patients had a lower cell density in the normotensive eye. In the second portion of this study 46 patients with acute increases in ocular pressure were studied. Some of these were simple angle-closure glaucoma; others had glaucomatocyclitic crises. In both groups there was a decrease in cell density of about 48% if the acute attack lasted more than three days. To rule out the possibility of the keratic precipitates having a deleterious effect, 60 normotensive patients with unilateral iridocyclitis were examined. If the keratic precipitates were small and round there was no effect on the endothelial cell count but if there were large mutton-fat precipitates with severe iridocyclitis, then there was a distinctly lower endothelial cell density. Finally, in three patients with essential iris atrophy, it was noted that the endothelial cells were large, hazy, and pleomorphic, although biomicroscopy of the corneas appeared normal. (4 figures, 3 tables, 51 references)—David Shoch

Acta Paediatrica Scandinavica

CORTICAL BLINDNESS IN A CHILD WITH ACUTE LEUKEMIA. Ha, K., Kanaya, S., Ikeda, T., Ushio, T., Nakao, Y., and Yabuuchi, H. (Depts. Pediatr., Neurosurg., and Ophthalmol., Osaka Univ. School of Med., Osaka, Japan). Acta Paediatr. Scand. 69:781, 1980.

Ophthalmic complications are reported in about 9% of children suffering from acute leukemia. The typical complications are retinal bleeding and leukemic infiltration of the optic nerve, retina, iris, or orbit. Cortical blindness has not been described. Diagnosis of acute promyelocytic leukemia was made in an 18-month-old baby with exophthalmos, a retrobulbar tumor, and an abnormal blood picture. Response to chemotherapy was good with complete remission of the findings. Thirteen months after diagnosis the patient developed a left hemiplegia and bilateral loss of vision. She was given whole-brain irradiation but six months after she became blind she developed intraventricular bleeding and died. Autopsy disclosed atrophy of the visual cortex and necrosis of the white matter. Microscopic findings showed leukemic infiltration into the subarachnoid space around the visual cortex. In the cortex extensive degeneration of neurons and gliosis were found. (8 references)—David Shoch

Archives of Disease in Childhood

VISUAL DETERIORATION AS PRESENTATION OF SUBACUTE SCLEROSING PANENCEPHALITIS. Johnston, H. M., Wise, G. A., and Henry, J. G. (Regional Neurol. Ctr., Newcastle Gen. Hosp., Westgate Rd., Newcastle upon Tyne NE4 6BE, U.K.). Arch. Dis. Child. 55:899, 1980.

A focal chorioretinitis has been described frequently in patients with subacute sclerosing panencephalitis. However, visual symptoms preceding the more common features of the disease—myoclonic jerks, mental deterioration, and progressive motor deficit—are rare. The authors present a 7-year-old boy who was referred because of deteriorating

vision. His visual acuity was 6/60 (20/200) in both eyes and bilateral macular degenerative changes were seen and thought to be consistent with hereditary dystrophy. However, within a month he developed unusual behavior and increasing forgetfulness. An encephalogram showed periodic complexes and high measles complement-fixation titers were found in the cerebrospinal fluid and blood, confirming the diagnosis of subacute sclerosing panencephalitis. Four months after the onset of visual symptoms he started having myoclonic jerks. (2 figures, 6 references)—David Shoch

Archives of Ophthalmology

THE NATURAL HISTORY OF DIABETIC EXTRAMACULAR TRACTION RETINAL DETACHMENT. Charles, S., and Flinn, C. E. (Vitreo-Retinal Res. Found., 909 Ridgeway Loop Rd., Memphis, TN 38138). Arch. Ophthalmol. 99:66, 1981.

There is a difference of opinion concerning the advisability of surgery for extramacular traction retinal detachment in diabetic patients before development of macular detachment. Ninety-nine eyes with extramacular traction retinal detachment of 84 diabetic patients observed without surgery had a detachment rate of 13.8% in one year. This rate compares favorably with the reported 30% to 47% failure rate for diabetic eyes subjected to vitrectomy. (2 figures, 4 tables, 3 references)—Authors' abstract

INCREASED INCIDENCE OF CATARACTS IN MALE SUBJECTS DEFICIENT IN GLUCOSE-6-PHOSPHATE DEHYDROGENASE. Orzalesi, N., Sorcinelli, R., and Guiso, G. (Clinica Oculistica Univ., Ospedale Civile, 09100 Cagliari, Italy). Arch. Ophthalmol. 99:69, 1981.

Glucose-6-phosphate dehydrogenase (G6PD) deficiency in RBCs was found significantly more frequently in 210 male cataractous patients than in 672 control subjects of Sardinian origin. The frequency of the deficiency was increasingly higher in presenile cataracts. In the G6PD-deficient group, the incidence of cortical and total cataracts was also increased. It is suggested that decrease of the G6PD activity in the lens, which accompanies its deficiency in the erythrocyte, might play a role in the cataractogenesis of these patients. Moreover, G6PD deficiency should be added to other conditions, such as the galactosemic states and riboflavin deficiency, where cataracts represent a sensitive indicator of metabolic abnormalities of the RBC. (3 figures, 17 references)—Authors' abstract

KERATOCONJUNCTIVITIS AND SOFT CONTACT LENS SOLUTIONS. Binder, P. S., Rasmussen, D. M., and Gordon, M. (Sec. Ophthalmol., Veterans Adm. Med. Ctr., and Univ. California, San Diego, CA). Arch. Ophthalmol. 99:87, 1981.

Three patients with soft contact lenses had acute onset of a red eye that clinically appeared consistent with an adenoviral keratoconjunctivitis. The signs and symptoms cleared within five to ten days in all three patients. When the patients reinserted their lenses after their initial episodes, the same signs and symptoms again developed, including follicular conjunctivitis, punctate keratitis, and subepithelial infiltrates. All three were using preserved solutions for soft lens disinfection, and when switched to thermal disinfection with saline tablets, a recurrence of the syndrome did not develop when the lenses were replaced. This syndrome seems related to a toxic or allergic reaction to the preservatives in the contact lens disinfecting solutions. Similar patients who have an acute or chronic red, irritated eye should stop their lens wear and later should change their disinfection technique to one that uses unpreserved saline solution to eliminate the problem. (1 table, 50 references)—Authors' abstract

Clinical Endocrinology

AN EVALUATION OF SUPERVOLTAGE ORBITAL IRRADIATION FOR GRAVES' OPHTHALMOPATHY. Teng, C. S., Crombie, A. L., Hall, R., and Ross, W. M. (Dept. Med., Welsh Natl. School of

Med., Health Park, Cardiff CF4 4XN, U.K.). Clin. Endocrinol. 13:545, 1980.

Twenty patients with moderately severe ophthalmopathy due to Graves' disease or ophthalmic Graves' disease were treated by supervoltage orbital radiotherapy generated by a linear accelerator. Seven patients (35%) showed some response within three weeks of the treatment, four patients (20%) improved minimally while nine patients (45%) were unchanged. Improvement was noted mainly in soft tissue changes while proptosis decreased in only four patients. With one exception, ophthalmoplegia did not improve after the radiotherapy. The benefit obtained with the treatment was not impressive. (1 figure, 4 tables, 15 references)—Authors' abstract

Current Therapeutic Research

A TREATMENT OF THE CORNEAL GRAFT REACTION WITH AZATHIOPRINE. Fronterré, A., and Bordin, R. (Dept. Ophthalmol., Pavia Univ. Med. Ctr., Pavia, Italy). Curr. Ther. Res. 28:618, 1980.

Fifteen patients with corneal graft reaction occuring between one and ten months after surgery were treated with oral azathioprine, 300 mg/day, methylprednisolone, 16 mg/day, and topical dexamethasone. Ten of the corneas cleared. It should be noted that the topical dexamethasone was used only three times a day and no mention is made of the interval between the onset of the graft reaction and the beginning of therapy. (1 table, 13 references)—David Shoch

Developmental Medicine and Child Neurology

DISSOCIATED VISUAL DEVELOPMENT. ELECTRODIAGNOSTIC STUDIES IN INFANTS WHO ARE "SLOW TO SEE." Mellor, D. H., and Fielder, A. R. (City Hosp., Hucknall Rd., Nottingham, England). Dev. Med. Child. Neurol. 22:327, 1980.

The authors describe four children who seemed to have poor vision in the early months of life. The pupil reflexes were normal and the fundus examination appeared to be normal but the visual-evoked potentials were definitely reduced or absent. The possibility of mental retardation was entertained but all four children appeared to have normal mentation and further development showed this to be so. As time went on, these children developed completely normal vision and their visual-evoked responses became normal. Thus there appears to be an entity of dissociated visual development, that is, the development of vision lagging behind the development of other systems. In these children the visual-evoked response may in fact be abnormal and may falsely indicate a poor prognosis. It is difficult to explain the slowness in the development of vision but one theory is that the optic nerves have not completely myelinated at birth and do so in postnatal life, at which time vision becomes normal. It is important to follow these children without making a definite statement as to prognosis in spite of an abnormal visual-evoked response, because the visual-evoked response becomes normal as the vision improves. (3 figures, 25 references)—David Shoch

Headache

OPHTHALMOPLEGIC MIGRAINE. ISCHEMIC OR COMPRESSIVE NEUROPATHY? Vijayan, N. (Dept. Neurol., Univ. California, Davis Med. Ctr., 2315 Stockton Blvd., Sacramento, CA 95817). Headache 20:300, 1980.

It is currently believed that the ophthalmoplegia in ophthalmoplegic migraine is caused by compression of the cranial nerves by dilated and swollen carotid or basilar arteries. One of the author's patients did not fit the pattern of a compressive neuropathy because the pupil was spared during three episodes of ophthalmoplegic migraine. Review of published reports disclosed that only in about one third of the patients with ophthalmoplegic migraine is there in fact involvement of the pupil. This suggests that the ophthalmoplegia is caused by a delayed ischemic neuropathy leading to border zone infarction of the nerve rather than direct compression from an enlarged vessel. (1 figure, 2 tables, 15 references)—David Shoch

Investigative Ophthalmology and Visual Science

SCHLEMM'S CANAL. THE EFFECT OF INTRAOCULAR PRESSURE. Moses, R. A., Grodzki, W. J., Etheridge, E. L., and Wilson, C. D. (Dept. Ophthalmol., Washington Univ. School of Med., 660 S. Euclid Ave., St. Louis, MO 63110). Invest. Ophthalmol. Vis. Sci. 20:61, 1981.

In an excised eye the trabecular meshwork was made impermeable by introducing oil in the anterior chamber. Schlemm's canal was cannulated and it was noted that resistance to circumferential flow along the canal increased moderately as transmural pressure was increased. At the same time, resistance to outflow from the canal through the collector channels increased markedly as the transmural pressure was increased to 35 or 40 mm Hg. This suggested that the usual primary defect in open-angle glaucoma is reduced facility of the inner canal wall and that collapse of the canal with reduction of filtering area and plugging of collector channels is a secondary effect. (6 figures, 1 table, 14 references)—David Shoch

ENDOTHELIAL FUNCTION IN PATIENTS WITH CORNEA GUTTATA. Burns, R. R., Bourne, W. M., and Brubaker, R. F. (Mayo Clinic, Rochester, Minn. 55901). Invest. Ophthalmol. Vis. Sci. 20:77, 1981.

The authors compared the corneas of two groups of patients. One group had early Fuchs' dystrophy without epithelial edema and the second group had normal corneas. Statistically significant differences were found between the two groups of corneas as regards corneal thickness, endothelial cell size, and endothelial permeability to fluorescein. There was also a difference in intraocular pressure in the two groups. Although this was statistically significant, it was clinically insignificant. The results suggests that the earliest defect in Fuchs' dystrophy is a breakdown in the barrier function and, thus, increasing permeability that results in a thickened cornea. (4 figures, 3 tables, 39 references)—David Shoch

Journal of the American Medical Association

HIGH-ALTITUDE RETINOPATHY. McFadden, D. M., Houston, C. S., Sutton, J. R., Powles, A. C. P., Gray, G. W., and Roberts, R. S. (77 Ledge Rd., Burlington, VT 05401). J.A.M.A. 245:581, 1981.

Thirty-nine healthy subjects were examined by ophthalmoscopy and retinal photography before and after a stay at 5,360 m. Vascular engorgement and tortuosity and optic disk hyperemia were seen in all subjects at altitude. This was apparently a "normal" response to hypoxia. Twenty-two (56%) of the subjects had retinal hemorrhages and one showed cotton-wool spots. These changes are abnormal reactions and are considered high-altitude retinopathy. After maximal exertion on a cycle ergometer, fresh hemorrhages were observed in seven of 34 subjects. The incidence of hemorrhage associated with exercise was significantly greater than predicted. Fluorescein leakage was noted after exercise in eight of 20 persons tested and was associated with exercise-induced hemorrhages. Two persons developed premacular hemorrhages. The high-altitude retinal hemorrhages described here are not important clinically except for those which are premacular. The authors therefore recommend descending from altitude only if the hemorrhages are in the macular area of the retina. (3 figures, 3 tables, 27 references)—David Shoch

PHYSICIANS MEASURE UP: A STUDY OF 13 SURGICAL PROCEDURES. Elliott, R. V., Kahn, K. A., and Kaye, R. (1601 E. 19th Ave., Denver, CO 80218). J.A.M.A. 245:595, 1981.

A professional standards review organization in Colorado established indications for cataract surgery. The major indications were a lens opacity decreasing the visual acuity to 6/22 (20/70) or less or a diminished visual acuity substantially interfering with the patient's ability to function. There were three other indications related to the presence of uveitis and glaucoma and inability to see the interior of the eye. When the above criteria were used, the examiners found that 99.3% of

the 1,768 cases reviewed met the indications for the procedure. In 98% the indication was the first given, that is, visual acuity reduced to 6/22 (20/70) or less in the eye under consideration for surgery or interference with the patient's lifestyle. (14 tables, 16 references)—David Shoch

Journal of Gerontology

NEURON LOSS IN THE AGING VISUAL CORTEX OF MAN. Devaney, K. O., and Johnson, H. A. (Dept. Pathol., Tulane Univ., New Orleans, LA 70112). J. Gerontol. 35:836, 1980.

Measured by a cell dispersion technique, the neuron population densities of the macular projection areas of 23 individuals aged 20 to 87 years fell from about 46 million neurons per gram of tissue at age 20 to about 24 million neurons per gram at age 80. A corresponding increase in glial cells was found in the gray and subjacent white matter. Since the older brains showed a significant decrease in mass, the decrease in neuron population density could only reflect an absolute loss of neurons proportional to or greater than the nearly 50% loss indicated by these data. Neuron death in the macular projection area of the human visual cortex is evidently a major factor in the loss of visual acuity in old age. (5 figures, 4 tables, 21 references)—Authors' abstract

Journal of Pediatric Ophthalmology and Strabismus

MARCUS-GUNN JAW-WINKING PTOSIS. CLASSIFICATION AND SURGICAL MANAGEMENT. Bullock, J. D. (33 W. First St., Suite 590, I.B.M. Bldg., Dayton, Ohio 45402). J. Pediatr. Ophthalmol. Strabismus 17:375, 1980.

The author divides his patients into various categories depending on whether the blepharoptosis is mild or severe and whether the jaw-winking is mild or severe. If the jaw-winking is mild, then the surgery is limited to repair of the blepharoptosis, but when the jaw-winking is severe then a complete excision of the levator palpebrae superioris muscle is done from the tarsus back to the orbital apex. The eyelids are then repaired with a frontalis sling. Because this frequently results in excess skin, the blepharoplasty is done at the same time. (11 figures, 6 references)—David Shoch

VISUAL EVOKED RESPONSE AS AN AID IN DIAGNOSING OPTIC NERVE HEAD DRUSEN. Bishara, S., Feinsod, M. (Dept. Ophthalmol., Hadassah Univ. Med. Ctr., Jerusalem, Israel). J. Pediatr. Ophthalmol. Strabismus 17:396, 1980.

An 11-year-old child was referred with a presumptive diagnosis of papillitis of the right optic nerve head. Although vision was correctible to 6/9 (20/30) the optic disk was irregular and there was mild engorgement of the veins. No real disk swelling was noted. The visual field showed irregular lower nasal and upper temporal defects. Visual-evoked potentials demonstrated conduction abnormalities in both eyes. Therefore, fluorescein angiography was performed. This disclosed buried drusen in both optic nerve heads even though there were no visual symptoms in the left eye. (4 figures, 6 references)—David Shoch

Journal of Theoretical Biology

MODELING OF THE VERTEBRATE VISUAL SYSTEM. I. WIRING DIAGRAM OF THE CONE RETINA. Siminoff, R. (Lab. Ocular Physiol., Dept. Ophthalmol., College of Physicians and Surgeons of Columbia Univ. 630 W. 168th St., New York, NY 10014). J. Theor. Biol. 86:673, 1980.

The author postulates four features to his model. (1) An informational flow diagram depicting the interconnections in the retina. (2) A cone mosaic organized into unit hexagons. (3) The effect of strayed light and electrical coupling between neighboring cones depicting the polarization changes in excess to those resulting from direct activation. (4) Polarization factors relating cone

hyperpolarization to changes in the direction and extent of polarization produced in retinal elements. The fundamental unit is the mosaic of regularly formed unit hexagons in the vertebrate cone retina. The center field of a bipolar cell receives direct input from the cones of these unit hexagons and the antagonistic surround field of the bipolar cells receives indirect inputs from the cones of six unit hexagons via horizontal cells. Ganglion cells may be tonic, formed by direct conversions of bipolar cells, or phasic, formed indirectly by convergence onto amacrine cells of hyperpolarizing and depolarizing types of bipolar cells. The complete blueprint consists of spatial organization of unit hexagons, weight inputs for each cone, taking into account strayed light effect and electrical coupling between cones, and polarization factors determined by assuming linearity up to the bipolar cell. (14 figures, 3 tables, 71 references)—David Shoch

Nature

CORTICAL BINOCULARITY IN INFANTS. Braddick, O., Atkinson, J., Julesz, B., Kropfl, W., Bodis-Wollner, I., and Raab, E. (Dept. Exp. Psychol., Univ. Cambridge, Downing St., Cambridge CB2 3EB, U.K.). Nature 288:363, 1980.

The primate visual cortex, including that of man, receives separate input from each eye and these interact in binocular cortical neurons. This organization is known to be vulnerable to disruption in early life. The authors assessed the binocular function of infants by recording visual-evoked potentials to the onset and offset of binocular correlation in a large-screen dynamic random dot display. They report that the human infant has a functional binocular visual cortex by 3 months of age, with some individuals showing cortical binocularity even earlier. (1 figure, 1 table, 13 references)—David Shoch

Quarterly Journal of Medicine

THE USE OF COLOUR VISION MEASUREMENT IN THE DIAGNOSIS OF DIGOXIN TOXICITY. Aronson, J. K., and Ford, A. R. (MRC Unit and Univ. Dept. Clin. Pharmacol., Radcliffe Infirm., Woodstock Rd., Oxford OX2 6HE, U.K.). Q. J. Med. 49:273, 1980.

Color vision has been measured, using the Farnsworth-Munsell 100-hue test, in control subjects and in patients taking digoxin. In ten patients with digitalis toxicity, of whom only two described symptomatic abnormalities, color vision was impaired compared with that of both control subjects and patients who had been taking digoxin for more than two months without developing toxicity. Withdrawal of digoxin from patients with toxicity led to improved color vision. Color vision scores correlated well with plasma digoxin concentrations. These results suggest that color vision assessment may be of use in screening for digitalis toxicity at the bedside and in assessing the degree of digitalis intoxication in an individual patient. (6 figures, 19 references)—David Shoch

Correction

In the article, "Treatment of macular subretinal neovascularization with the red-light krypton laser in presumed ocular histoplasmosis syndrome," by Yuval Yassur, Emil Gilad, and Isaac Ben-Sira (Am. J. Ophthalmol. 91:172, 1981), the term "polychromatic light of the xenon photocoagulator" should have been used. THE JOURNAL regrets this error.

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least four months before the date of occurrence.

FIFTH INTERNATIONAL CONGRESS OF EYE RESEARCH

The Fifth International Congress of Eye Research will be held Oct. 3–8, 1982, in Eindhoven, The Netherlands. For further information, write Prof. Dr. S. L. Bonting, Vth ICER Secretariat, Dept. of Biochemistry, University of Nijmegen, P.O. Box 9101, 6500 HB Nijmegen, The Netherlands.

OPHTHALMOLOGICAL SOCIETY OF THAILAND: EIGHTH CONGRESS OF THE ASIA PACIFIC ACADEMY OF OPHTHALMOLOGY

The Ophthalmological Society of Thailand will host the Eighth Congress of the Asia Pacific Academy of Ophthalmology in Bangkok, Thailand, Nov. 29–Dec. 4, 1981. For further information, write The Secretary General, Eighth Congress, Asia Pacific Academy of Ophthalmology, Dept. of Ophthalmology, Chumbhot Building, Chulalongkorn Hospital, Bangkok 5, Thailand.

CANADIAN OPHTHALMOLOGICAL SOCIETY

The Canadian Ophthalmological Society will hold its 44th annual meeting June 21–24, 1981, in Montreal, Canada.

CANADIAN IMPLANT ASSOCIATION: ANNUAL MEETING

The Canadian Implant Association will hold its annual meeting June 20, 1981, in Montreal, Canada. The registration fee is $125. For further information, write Marvin L. Kwitko, M.D., Program Chairman, 5591 Cote Des Neiges Rd., Montreal, H3T 1Y8, Canada.

MOORFIELDS EYE HOSPITAL: MICROSURGICAL COURSES

The Department of Clinical Ophthalmology, Institute of Ophthalmology, will conduct courses in microsurgery of the cornea and closed intraocular microsurgery to be held concurrently Oct. 8 and 9, 1981. Fees for each course will be $240. The closing date for applications is Aug. 31, 1981. For further information, write to Microsurgical Course Secretary, Dept. of Clinical Ophthalmology, Moorfields Eye Hospital, City Road, London, EC1V 2PD England.

STATE UNIVERSITY OF NEW YORK: OPHTHALMOLOGY ALUMNI DAY

The 13th annual ophthalmology alumni day of the State University-Kings County Hospital of Brooklyn, New York, will be held on June 11, 1981. For further information, write Mrs. Rita Belsky, State University of New York, Downstate Medical Center, Box 58, 450 Clarkson Ave., Brooklyn, NY 11203.

ROCHESTER EYE ISTITUTE AND DEPARTMENT OF OPHTHALMOLOGY, PARK RIDGE HOSPITAL: SIXTH ANNUAL OPHTHALMIC SEMINAR

The Rochester Eye Institute and the Department of Ophthalmology at Park Ridge Hospital will hold the Sixth Annual Ophthalmic Seminar June 12 and 13, 1981. For further information, write Miss Katherine Sgabelloni, R.N., Coordinator, Ophthalmic Services, Park Ridge Hospital, 1555 Long Pond Rd., Rochester, NY 14626.

MEDICAL SOCIETY OF THE STATE OF NEW YORK, SECTION ON OPHTHALMOLOGY: ANNUAL CONTINUING MEDICAL EDUCATION MEETING

The Section on Ophthalmology of the Medical Society of the State of New York will hold its annual Continuing Medical Education meeting June 27, 1981. For further information, write New York State Ophthalmological Society, 82 Kakely St., Albany, NY 12208.

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY: ANNUAL SCIENTIFIC MEETING

The Pennsylvania Academy of Ophthalmology and Otolaryngology will hold its annual scientific

meeting, May 20–23, 1981, in Bedford, Pennsylvania. Registration fee is $75 for nonmembers. For further information, write Donald B. Kamerer, M.D., Secretary, 3600 Forbes Ave., Pittsburgh, PA 15213.

Cleveland Clinic Foundation: Course on Neuro-ophthalmology for the Practitioner

The Center for Continuing Medical Education of the Cleveland Clinic Foundation will present a course, "Neuro-ophthalmology for the Practitioner," May 29 and 30, 1981. For further information, write Center for Continuing Medical Education, Cleveland Clinic Educational Foundation, 9500 Euclid Ave., Cleveland, OH 44106.

University of Michigan Medical Center: Department of Postgraduate Medicine and Health Professions Education Course

The Department of Postgraduate Medicine and Health Professions Education, University of Michigan Medical Center, will sponsor a course, "Current Perspectives in Ophthalmology," 1981, May 7 and 8, 1981, in Ann Arbor, Michigan. For further information, write Ms. Jan Reisinger, Towsley Center for Continuing Medical Education, Ann Arbor, MI 48109.

University of Illinois Eye and Ear Infirmary: Annual Residents/Alumni Day

The University of Illinois Eye and Ear Infirmary will hold its annual residents/alumni day on May 20, 1981. For further information, write Jacob Wilensky, M.D., University of Illinois Eye and Ear Infirmary, 1855 W. Taylor, Chicago, IL 60612.

Minnesota Academy of Ophthalmology and Oto-Laryngology: 12th Annual Benedict Lecture

The Minnesota Academy of Ophthalmology and Oto-Laryngology presented its 12th Annual William L. Benedict Lecture March 13, 1981, in Bloomington, Minnesota. Robert D. Yee spoke on the topic, "A clinical approach to nystagmus." Dr. Benedict was president of the ophthalmic publishing company at the time of his death in 1969.

Boston University Medical Center Ophthalmic Technician Program

A two-year training program for ophthalmic technicians begins June 1, 1981; there are openings for three students. For further information, write Ms. Norma Garber, Boston University School of Medicine, Dept. of Ophthalmology, 80 E. Concord St., L907, Boston, MA 02118.

INSTRUCTIONS TO AUTHORS

For the preparation of manuscripts for THE AMERICAN JOURNAL OF OPHTHALMOLOGY

THE AMERICAN JOURNAL OF OPHTHALMOLOGY publishes original and timely articles dealing with clinical and basic ophthalmology. Every typescript submitted is evaluated by two or more scientific referees who recommend that the paper be (1) accepted as submitted, (2) returned for revision, or (3) rejected. Acceptance is determined by such factors as the originality, significance, and validity of the contribution; the suitability of the subject matter for subscribers of THE JOURNAL; and the editorial care with which the manuscript has been prepared. Manuscripts that require extensive editorial correction or mechanical preparation will be returned for that purpose.

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1. Fuxe, K., and Jonsson, G.: The histochemical fluorescence method for the demonstration of catecholamines. Theory, practice and application. J. Histochem. Cytochem. 21:293, 1973.

2. Bjorklund, A., Flack, B., and Owman, C.: Fluorescence microscopic and microspectrofluorometric techniques for the cellular localization and characterization of biogenic amines. In Berson, S. A. (ed.): Methods in Investigative and Diagnostic Encocrinology, vol. 1. London, North-Holland Publishing Co., 1972, p. 318.

3. Duke-Elder, S., and Leigh, A. G.: Diseases of the Outer Eye. Cornea and Sclera. In Duke-Elder, S. (ed.): System of Ophthalmology, vol. 8, pt. 2. St. Louis, C. V. Mosby, 1965, pp. 811-856.

Abbreviations of periodicals are listed in Index Medicus: Am. J. Ophthalmol.; Surv. Ophthalmol.; Br. J. Ophthalmol; and the like. If there is any doubt about an abbreviation, the name of the publication should be spelled out completely.

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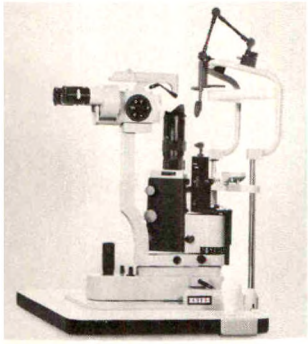
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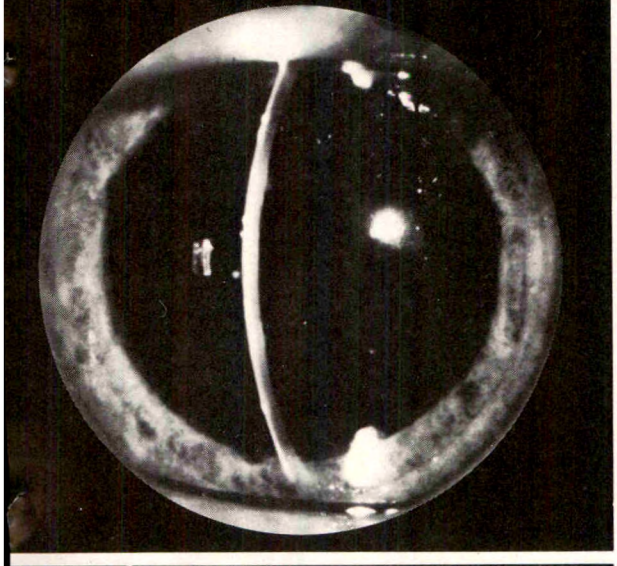
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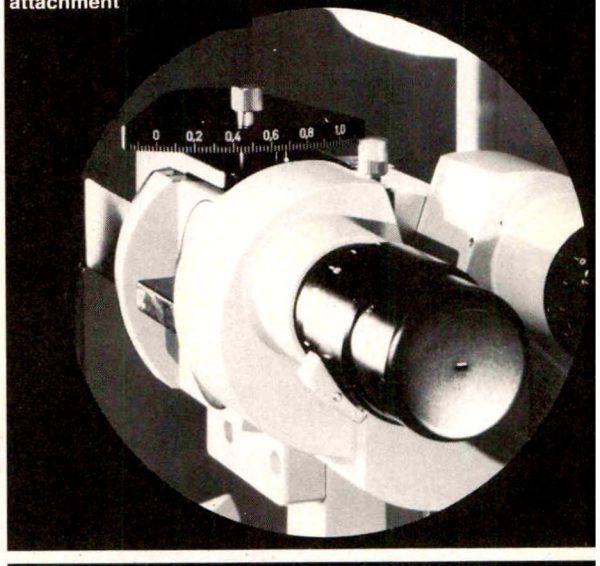
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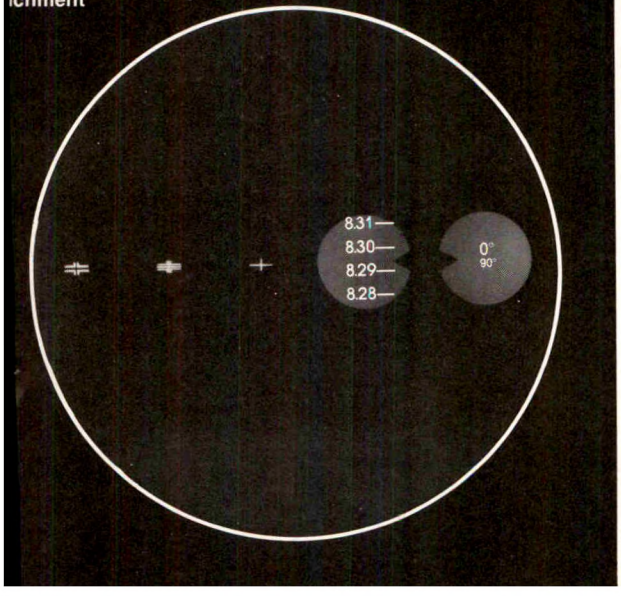
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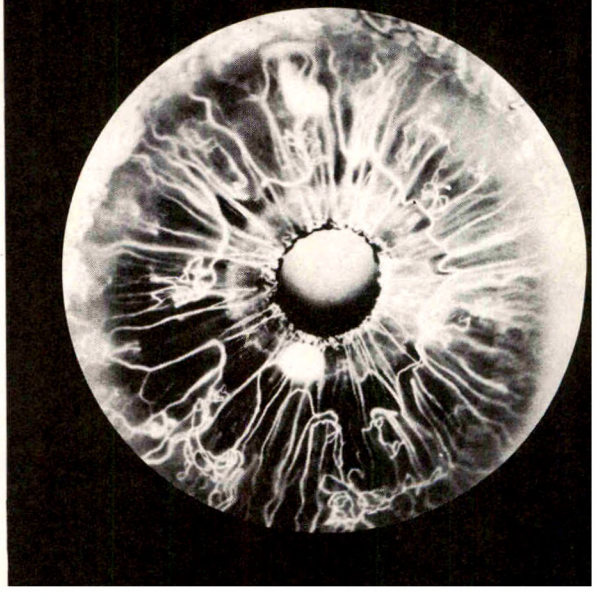
Corneal thickness measurement with Pachometer attachment



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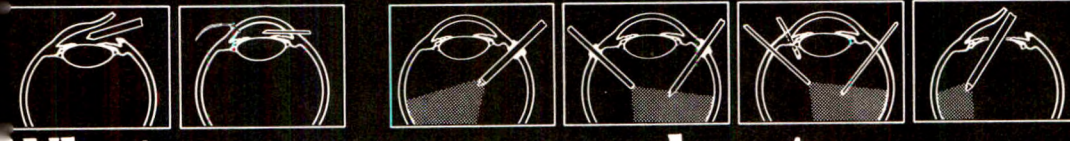
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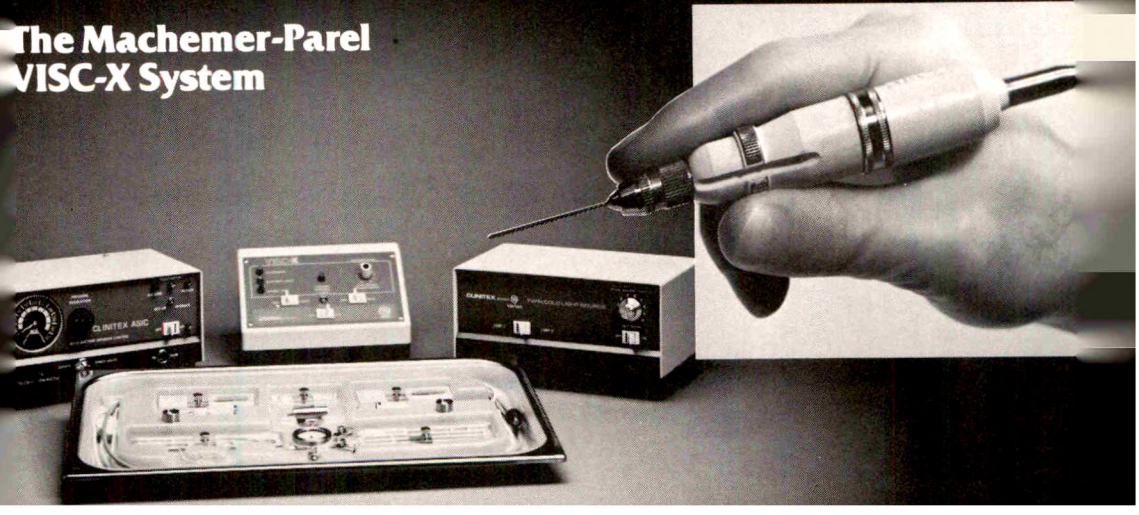
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Current Concepts in Ophthalmology

COURSES: NOVEMBER 21, 1981
Neuro Ophthalmology.
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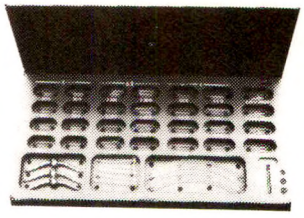
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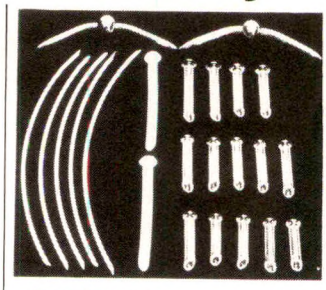
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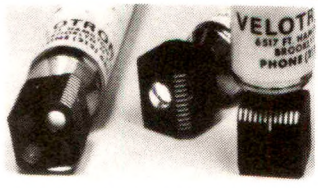
* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
J.C. Mustarde', L.T. Jones M.D. and A. Callahan M.D., Ophthalmic Plastic Surgery---Up-To-Date, Aesculapius Publishing Company, 1970.
L.T. Jones M.D. and J.L. Wobig M.D., Surgery of the Eyelids and Lacrimal System, Aesculapius Publishing Company, 1976.

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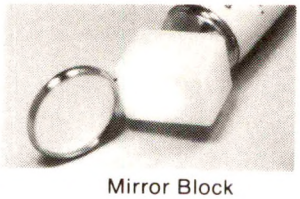
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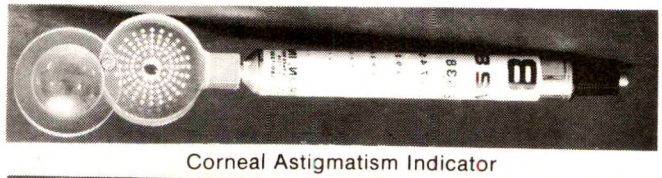
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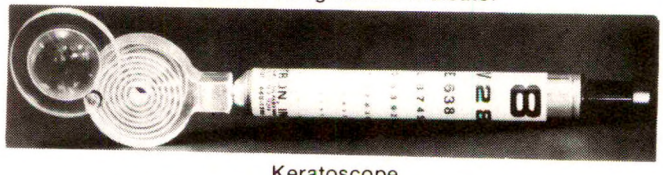
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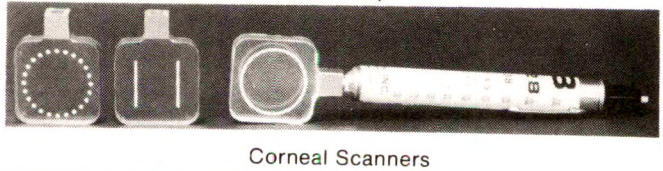
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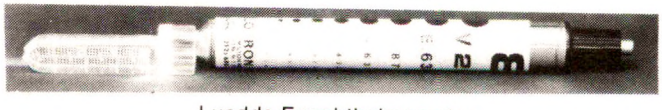
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British Journal of Ophthalmology February 1981 Vol. 65 No. 2

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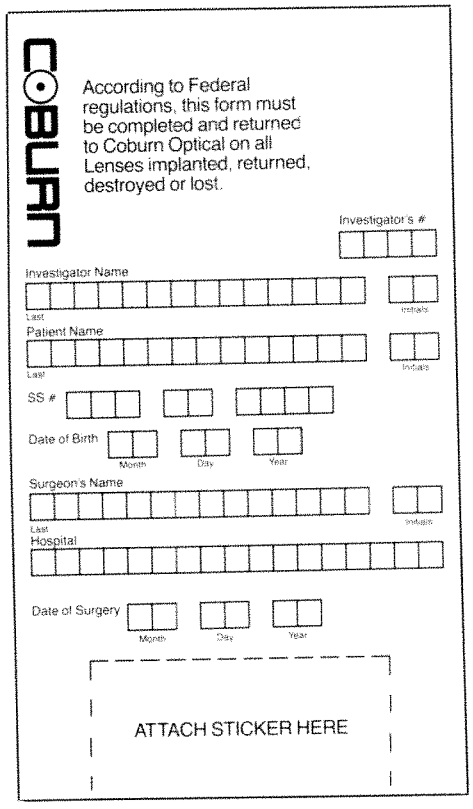
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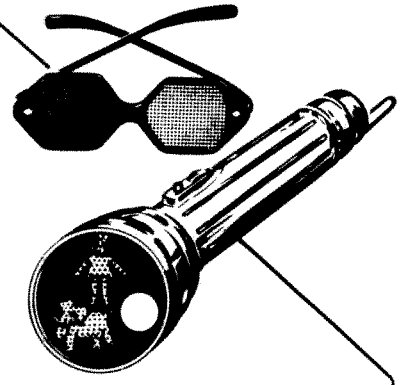


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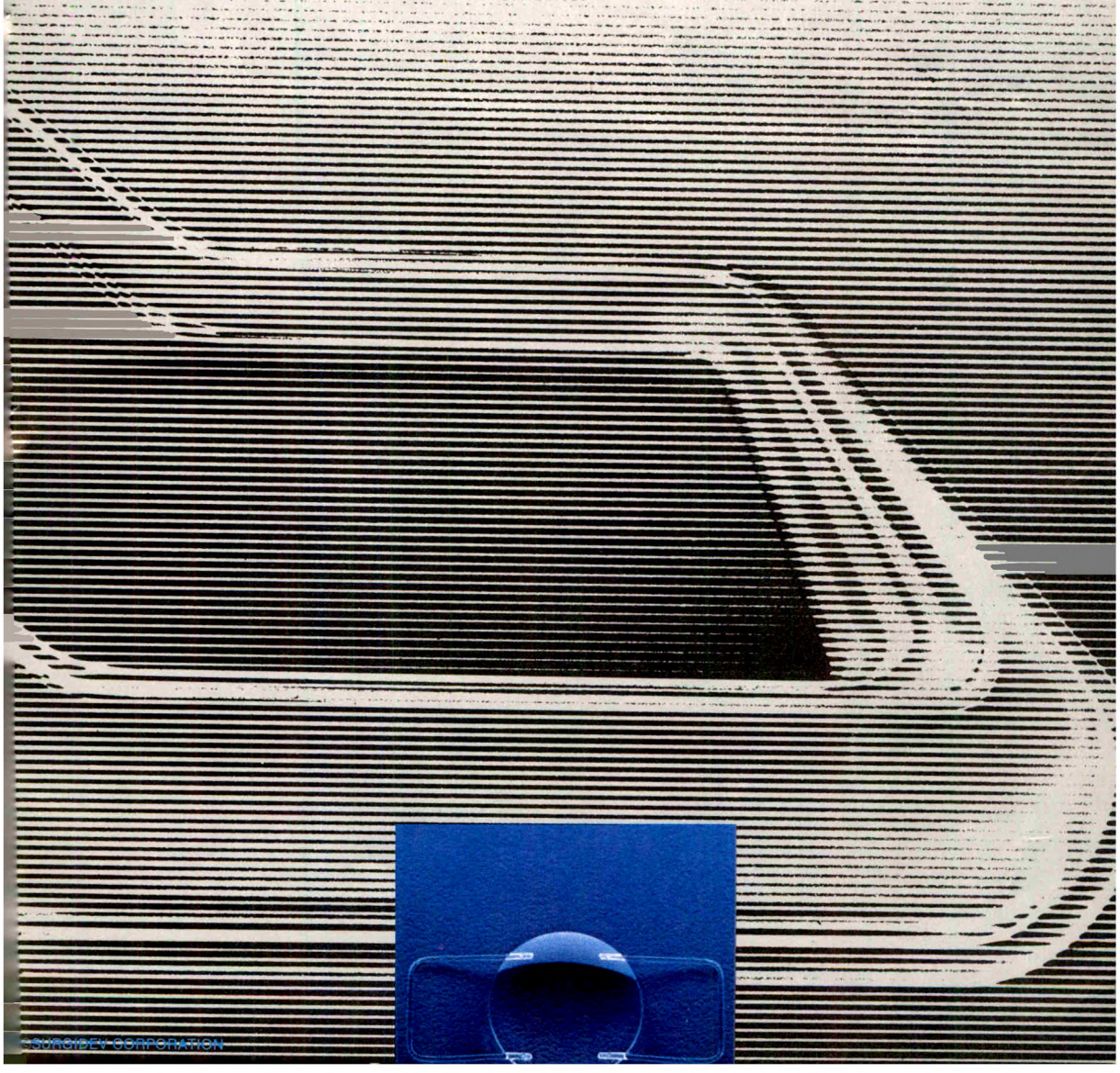
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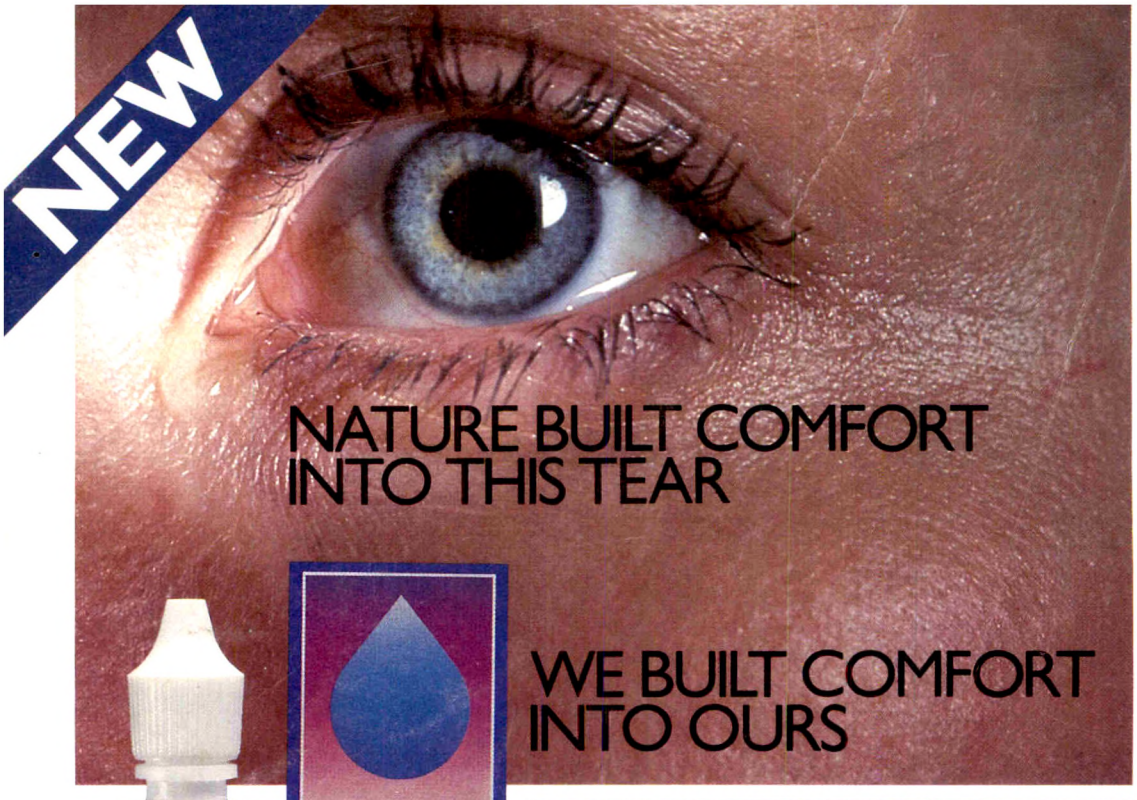
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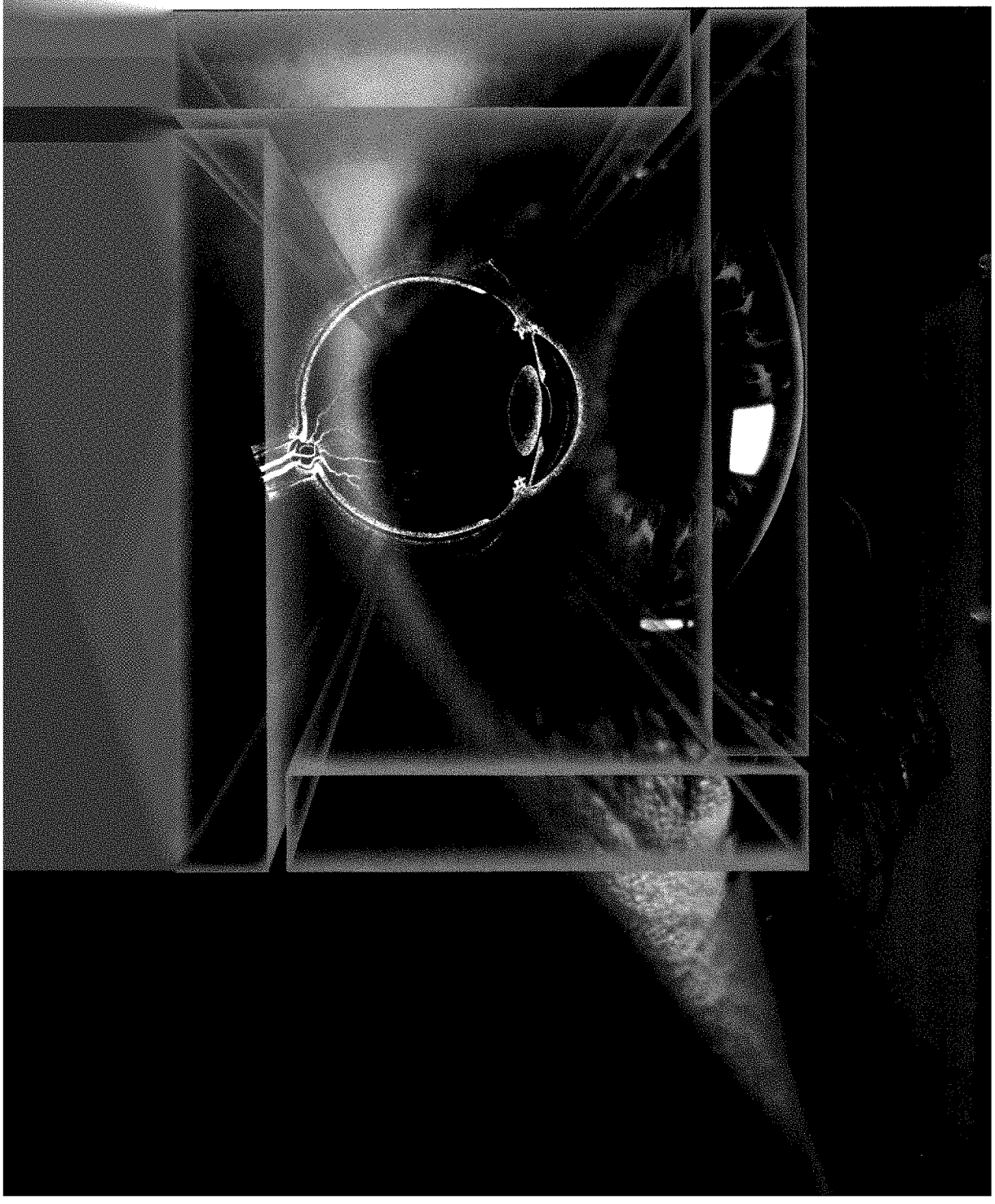
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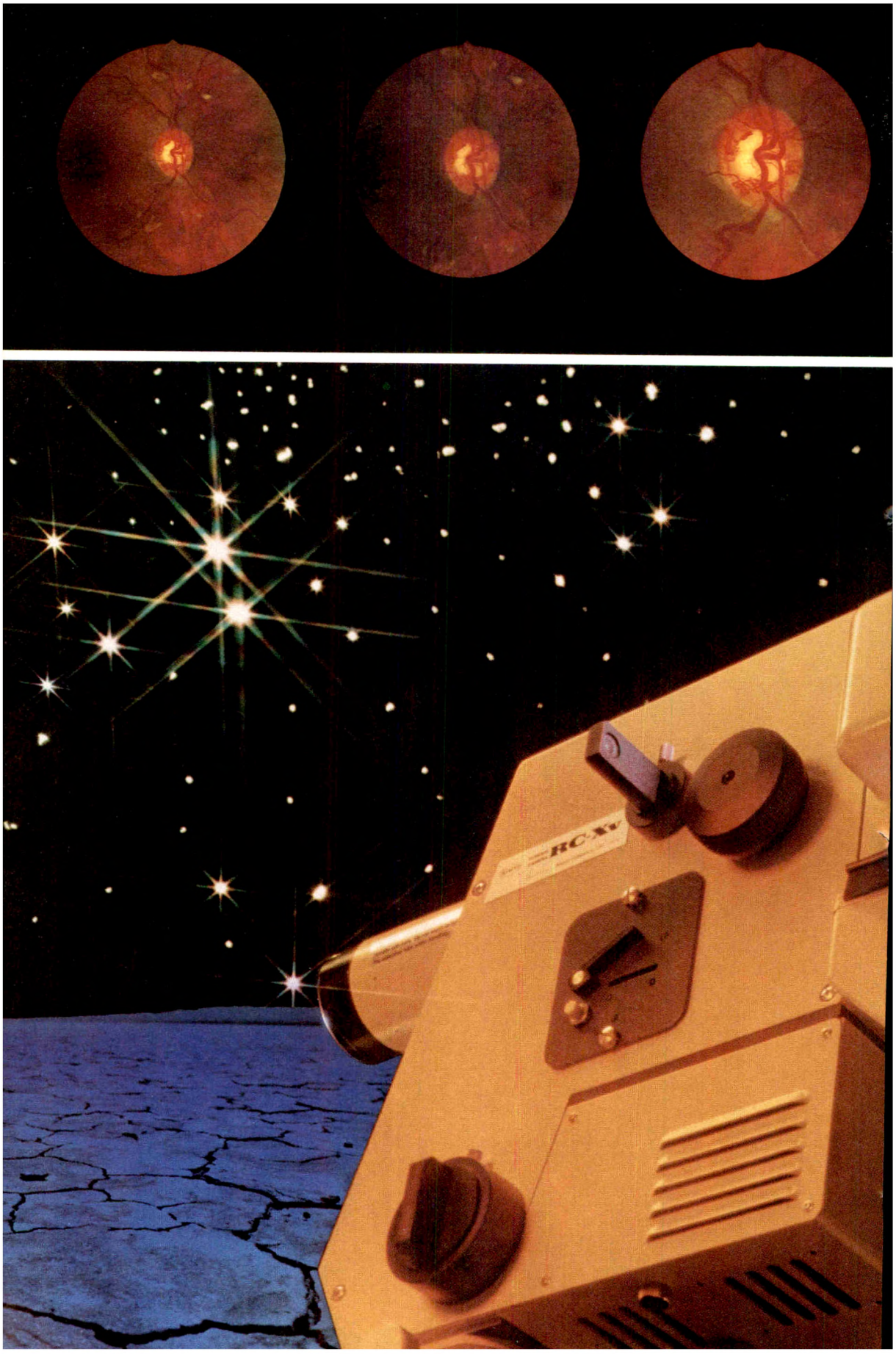


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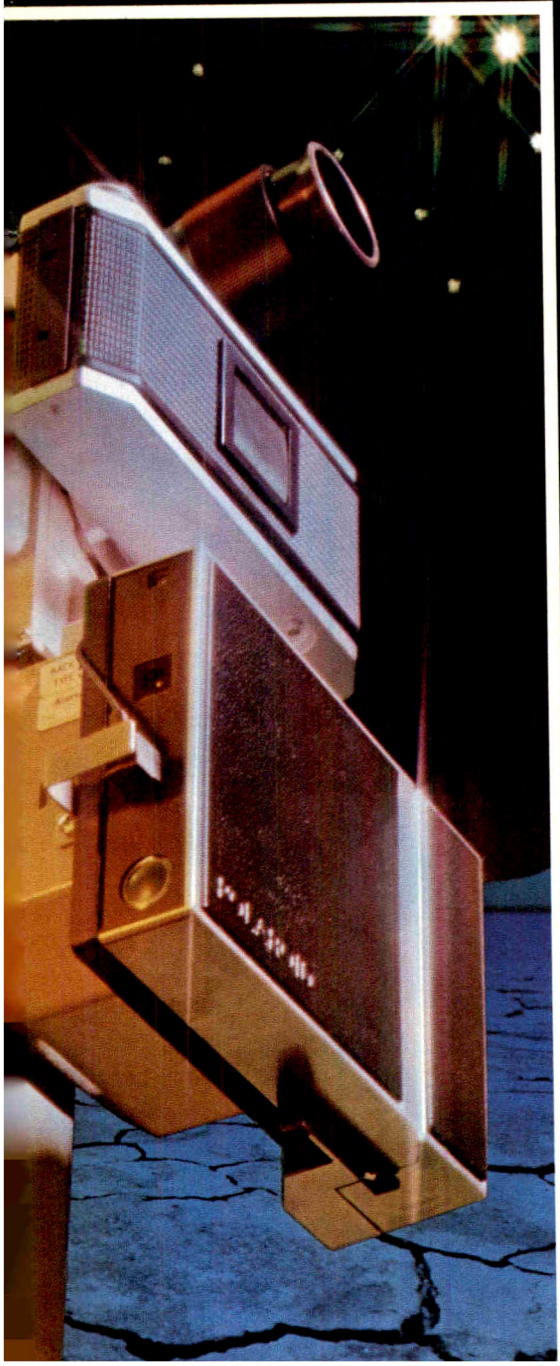
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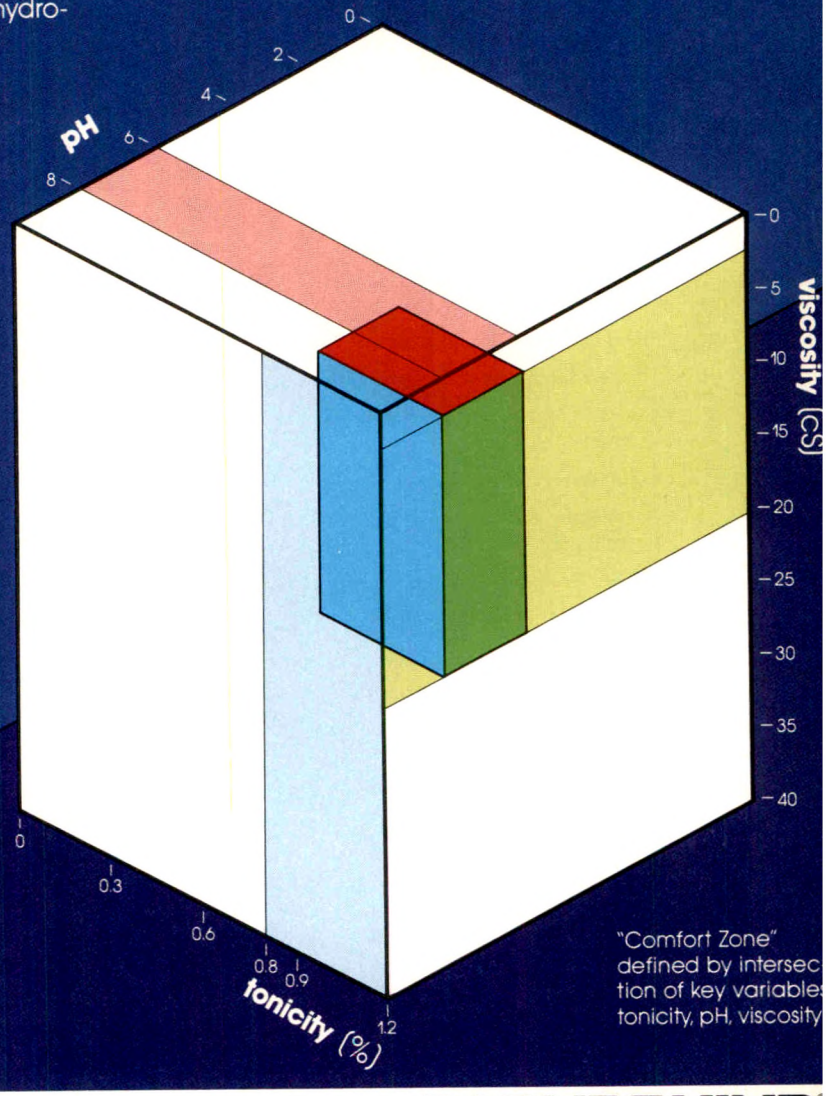
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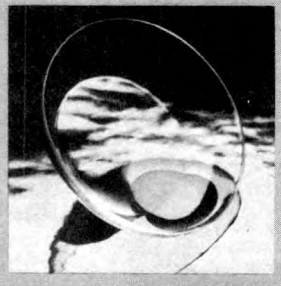
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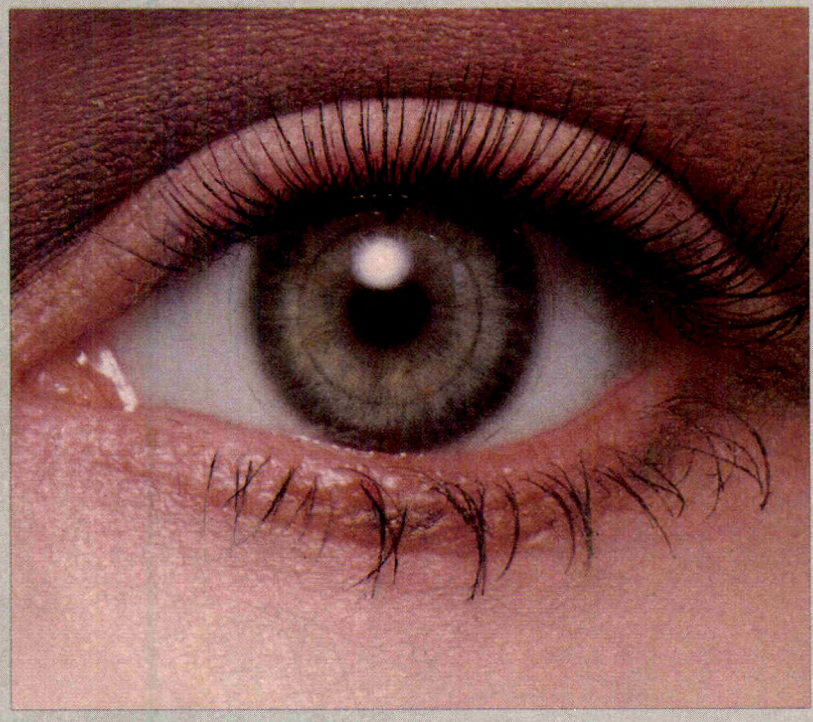
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[1]Clinical Data on file, Heyer-Schulte Medical Optics Center

[2]Cavanaugh, H.D., Bodner, B.I., Wilson, L.A. "Extended Wear Hydrogel Lenses, Long-Term Effectiveness and Costs," Ophthalmology, 1980, 87:9—871.

[3]Fatt, I. and Morris, J., "A Survey of Gas-Permeable Contact Lenses." The Optician, November 1977.

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GLAUCOMA, 2nd ed.

Edited and with contributions by PAUL A. CHANDLER, M.D., *Massachusetts Eye and Ear Infirmary, Boston, Massachusetts; and* W. MORTON GRANT, M.D., *Harvard Medical School, Boston, Massachusetts. (8 Contributors).* Now completely updated to provide recent findings and up to date techniques and procedures, this new edition is an important text for effective solutions to problems concerning glaucoma. Unusually clear instruction and practical guidance for the recognition and management of the many forms of glaucoma are the hallmarks of this exceptionally fine book. "There is no better book on glaucoma."—*American Journal of Ophthalmology. 381 pp. (7 x 10), illus., 1979, $26.00.*

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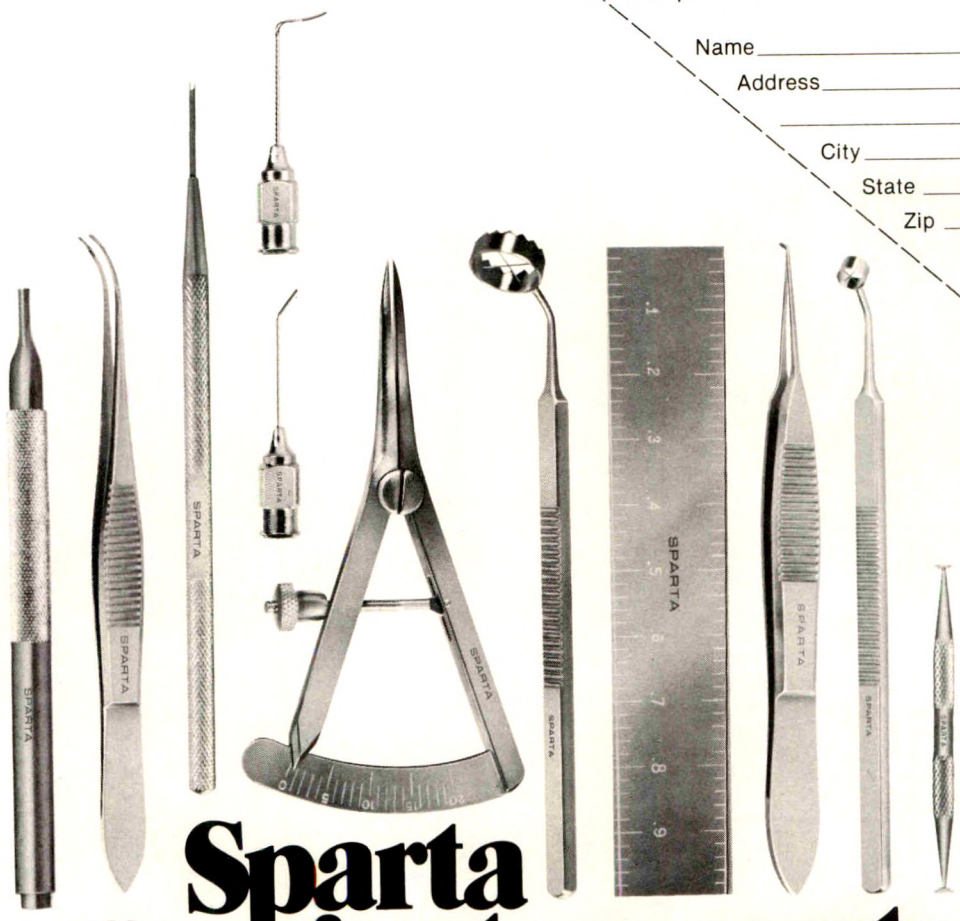
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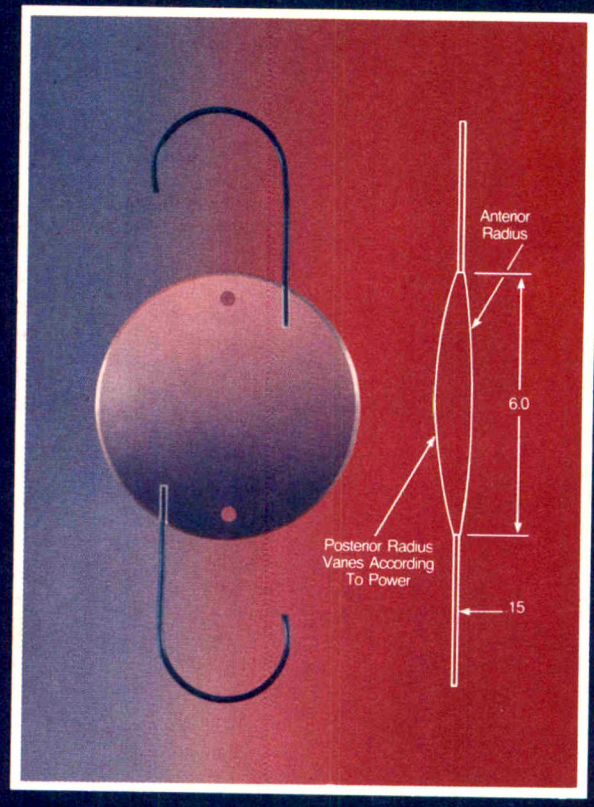
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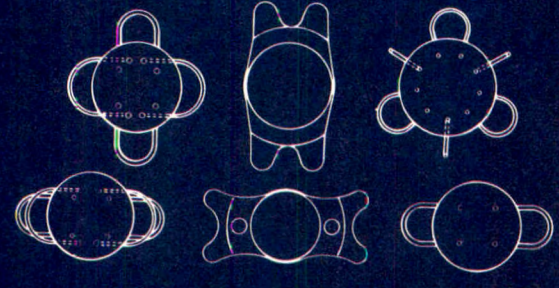
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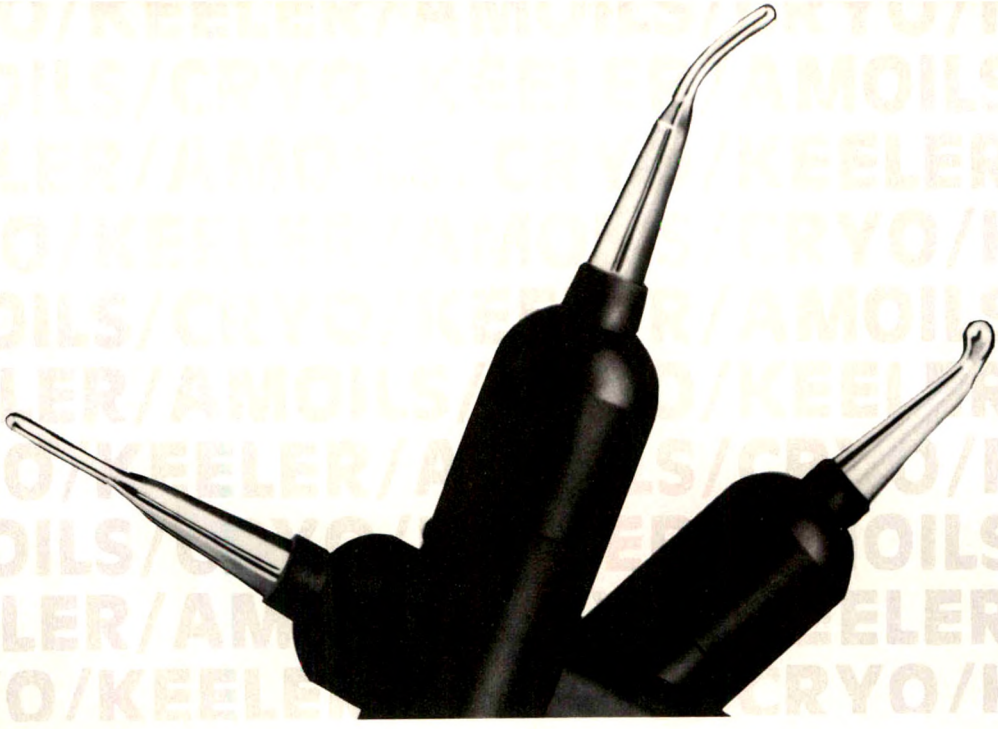
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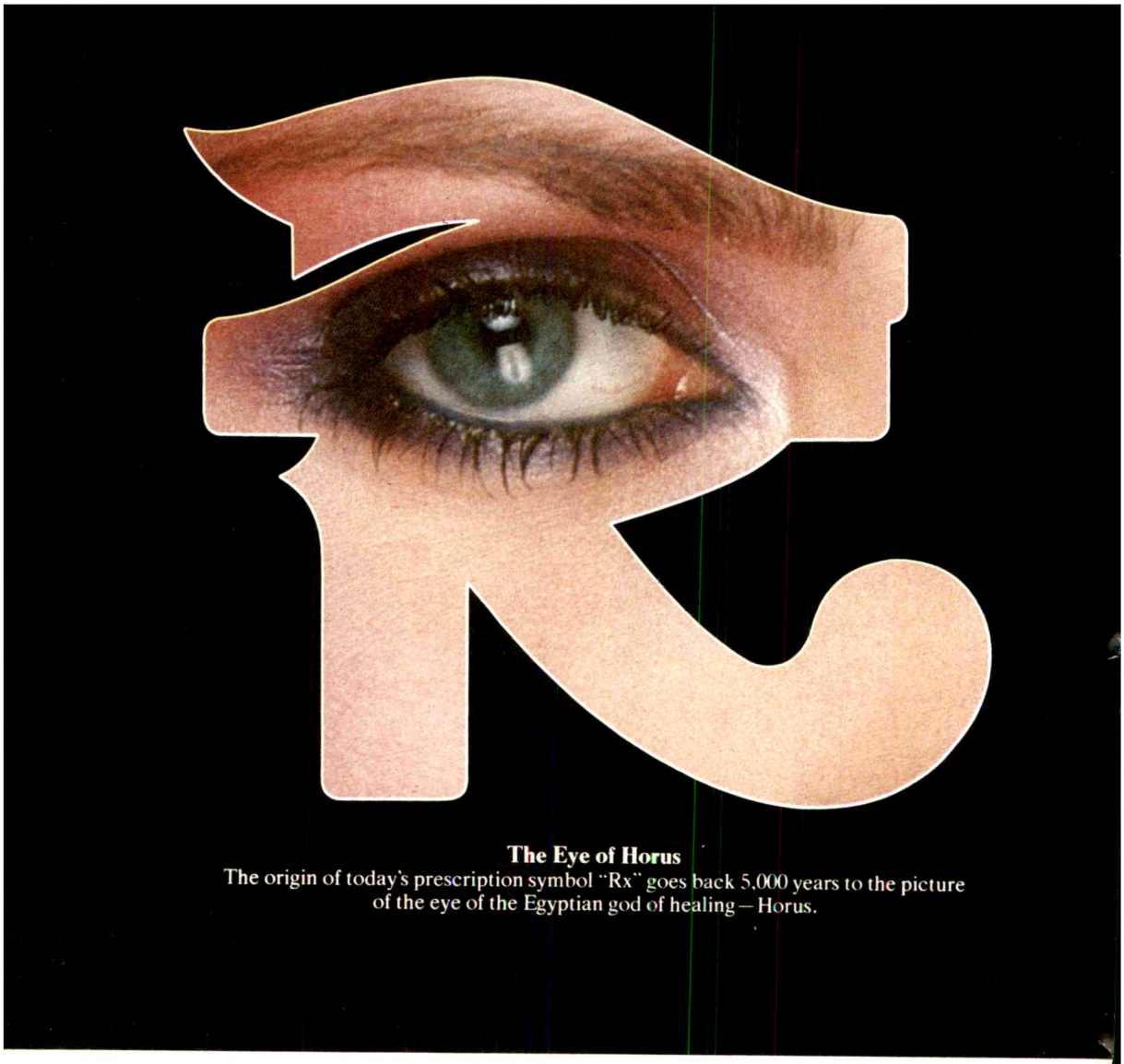
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DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2′-deoxyuridine.

VIROPTIC sterile opthalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro*.

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro*, 5-carboxy-2′-deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

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Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml. Store under refrigeration 2° to 8° C (36° to 46° F).

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Jack Hartstein, M.D. will discuss the concept of extended-wear. Dr. Hartstein has participated in numerous extended-wear clinical evaluations since studies began in 1976. Dr. Hartstein is Assistant Professor of Clinical Ophthalmology at Washington University School of Medicine and Chairman of the Department of Ophthalmology, Missouri Baptist Hospital.

Louis A. Wilson, M.D. will discuss the performance of available extended-wear products. Dr. Wilson's extensive background includes numerous contact lens clinical studies and continuous involvement in extended-wear since 1976. Dr. Wilson is a Professor of Ophthalmology and Director of the Contact Lens Clinic at Emory University School of Medicine.

Harold A. Stein, M.D. will discuss considerations involved in extended-wear practice management. Dr. Stein has had a great deal of practical experience with extended-wear in Canada, where the lenses were introduced in the mid 1970's. Dr. Stein is Chief of the Department of Ophthalmology at Scarborough General Hospital and is Assistant Professor of Ophthalmology at the University of Toronto.

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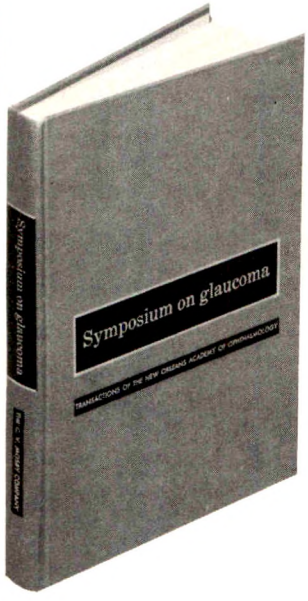
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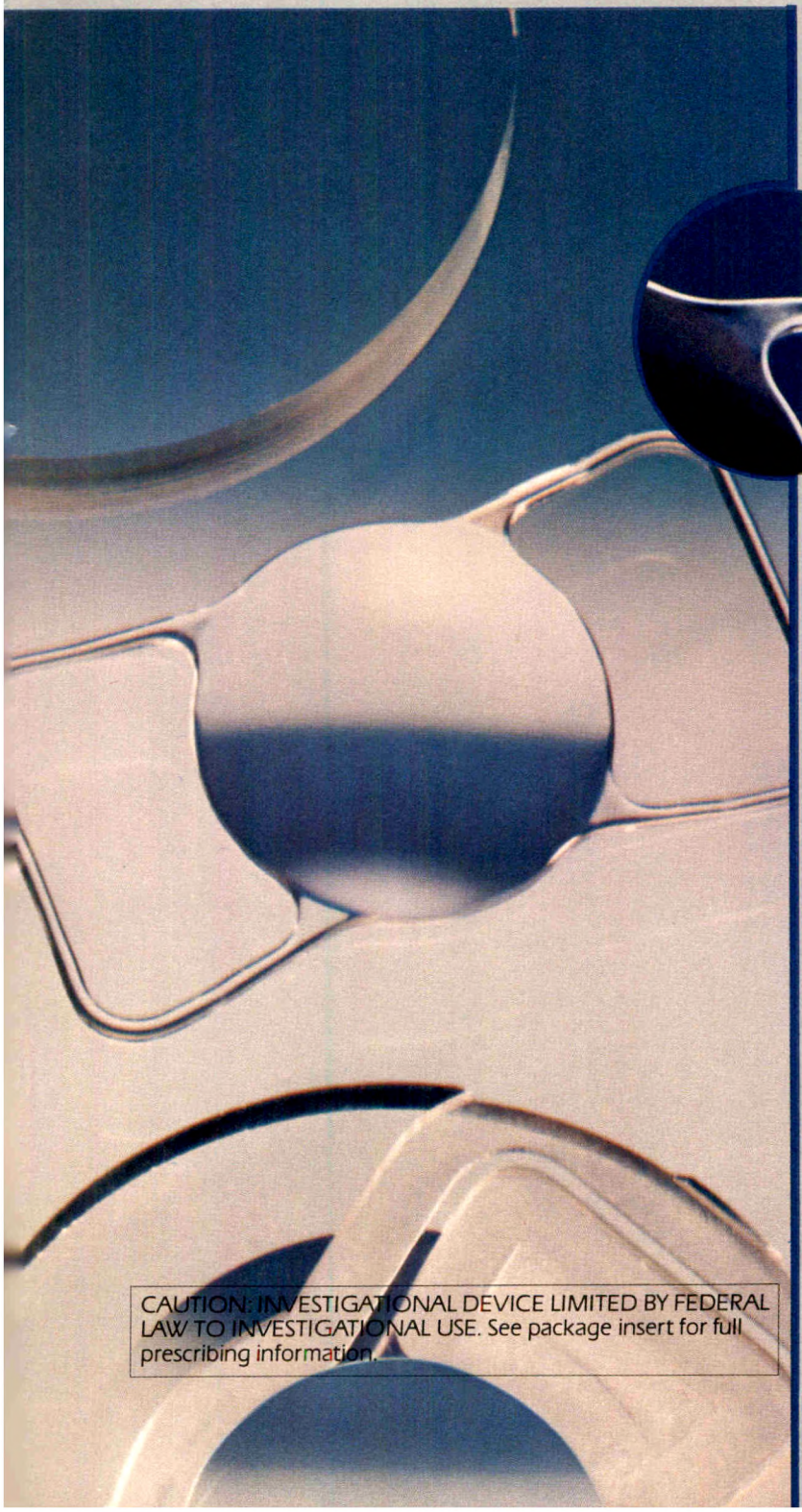
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The small photograph at left shows how the loops are an integral part of the lens body, producing a lens without drill holes or welds.

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Smoothness on edges and optical surfaces is achieved by proprietary polishing technology. CILCO's quality-conscious approach to lens manufacture also includes stringent control of optical power, resolution and physical dimensions.

For intraocular lenses, look to CILCO, your single source for the single-piece Optiflex anterior chamber lens. Please turn the page for additional lens styles.

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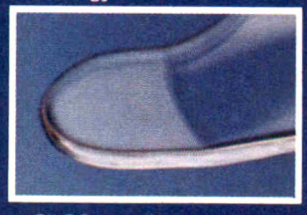
The Standard and Micro Series

The CILCO anterior chamber lenses are available in two series. The standard lens (Style AC — 6mm wide) is shown at upper right. The micro lens for small incisions (Style AM — 5mm wide) is shown at lower right.

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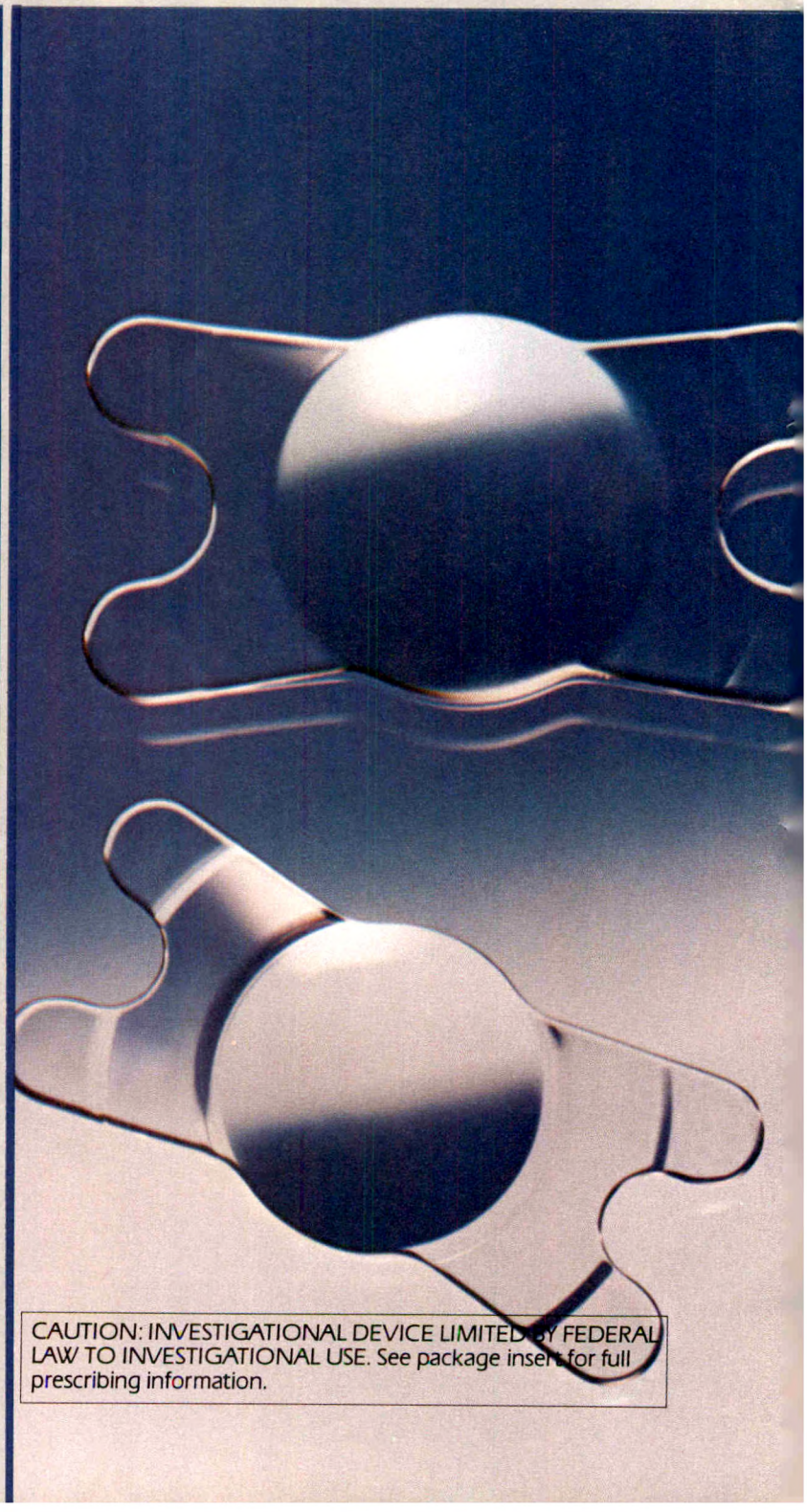
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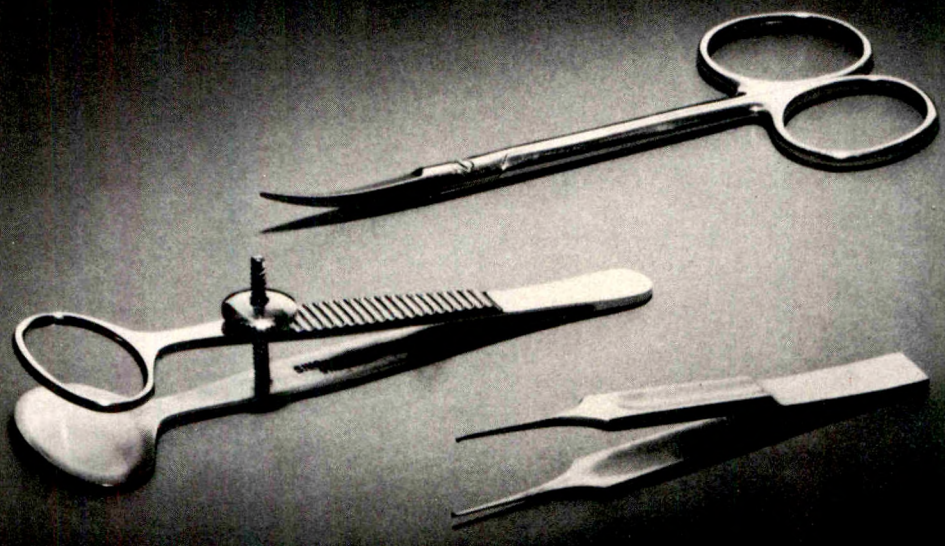
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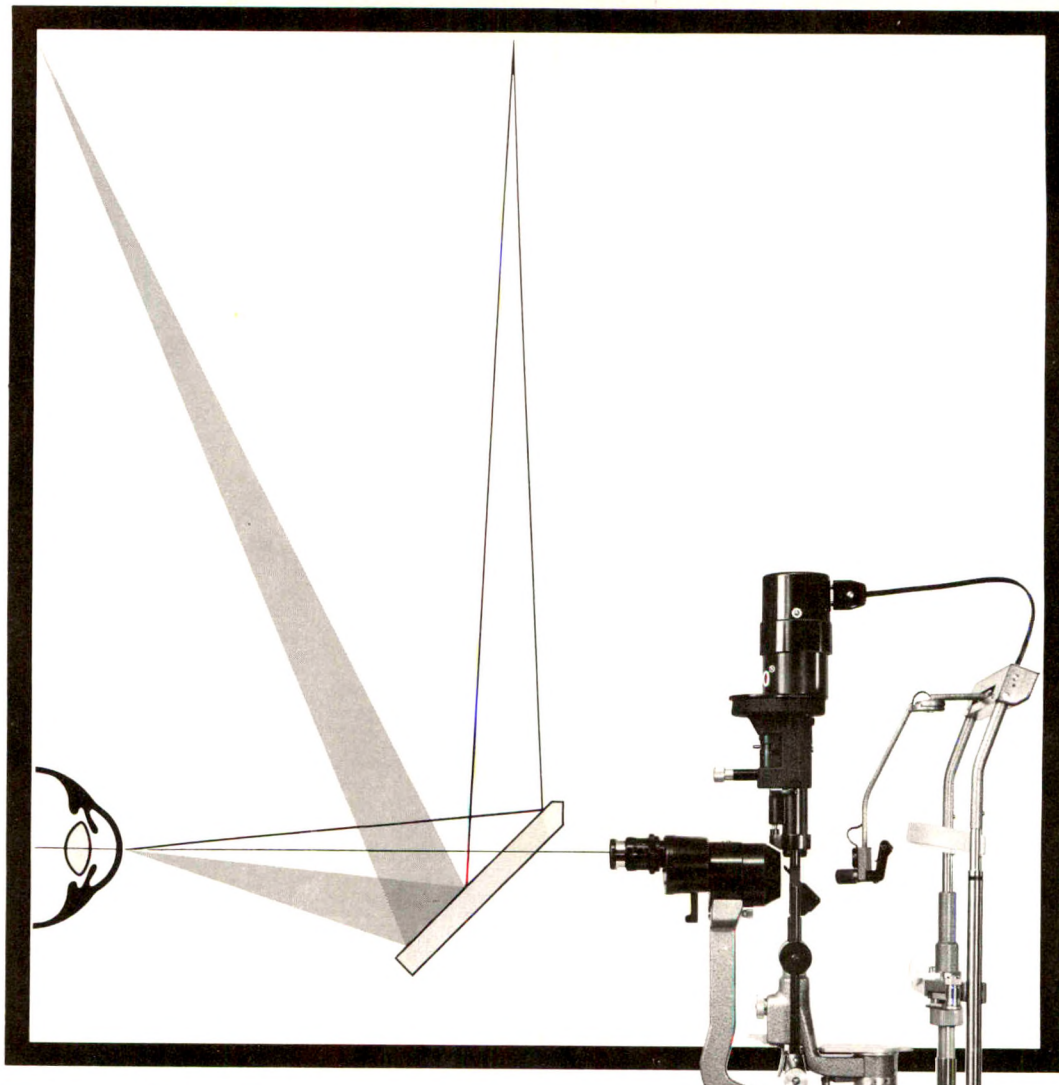
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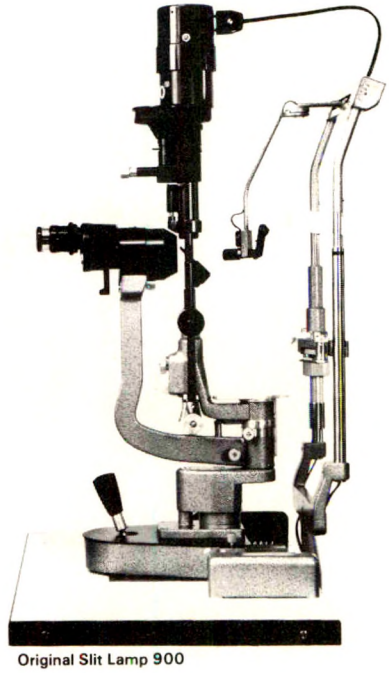
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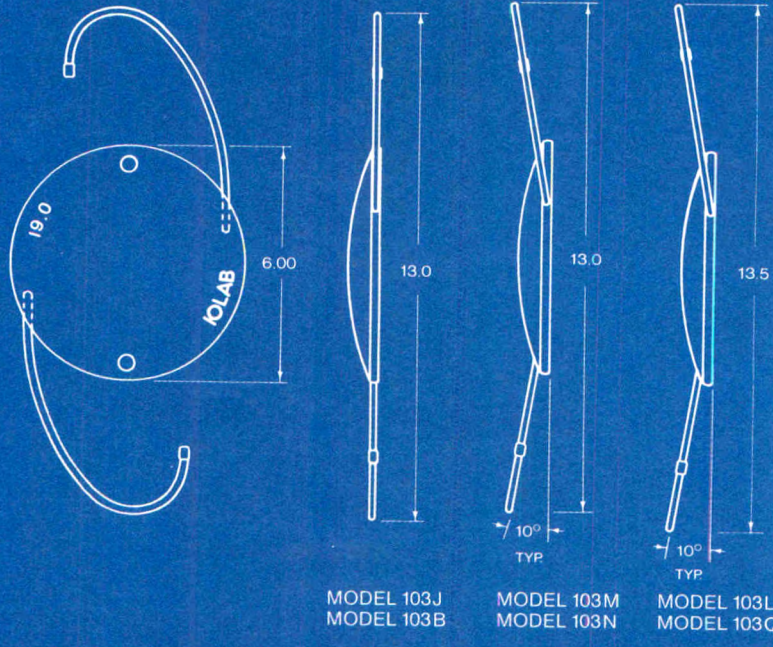
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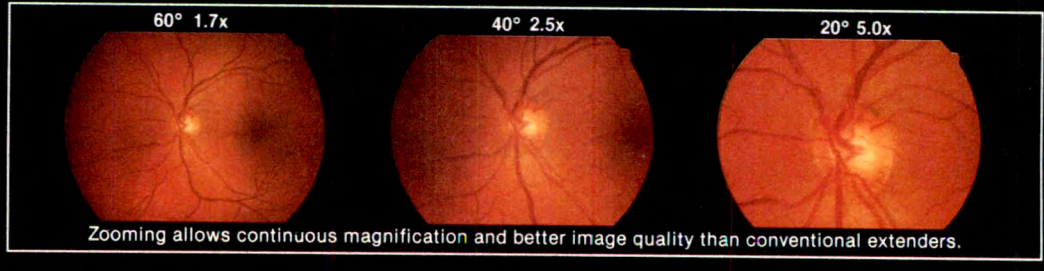
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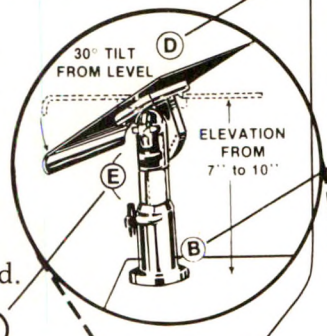
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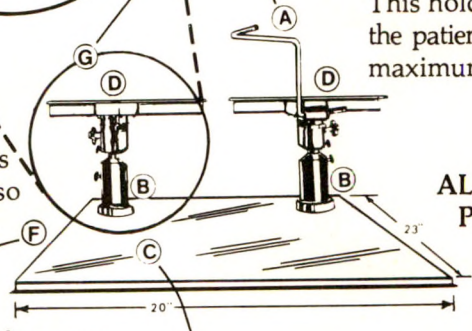
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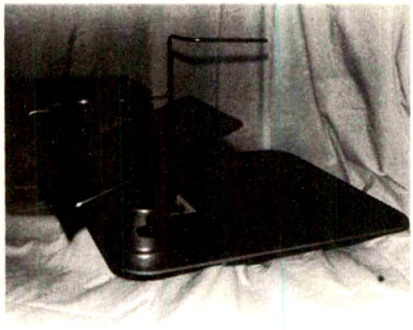
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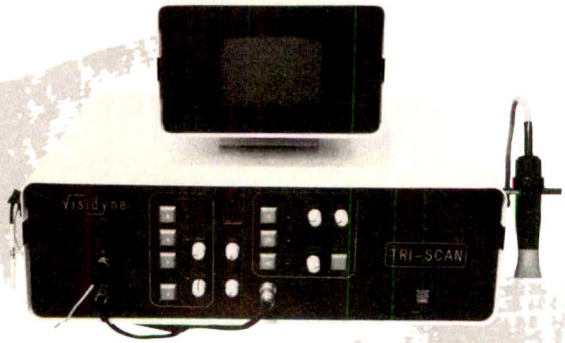
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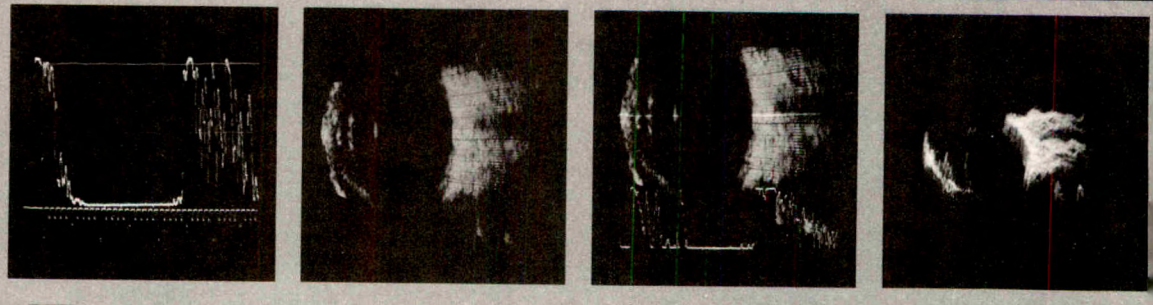
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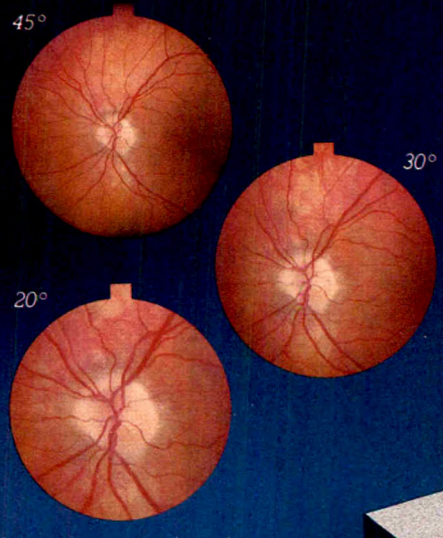
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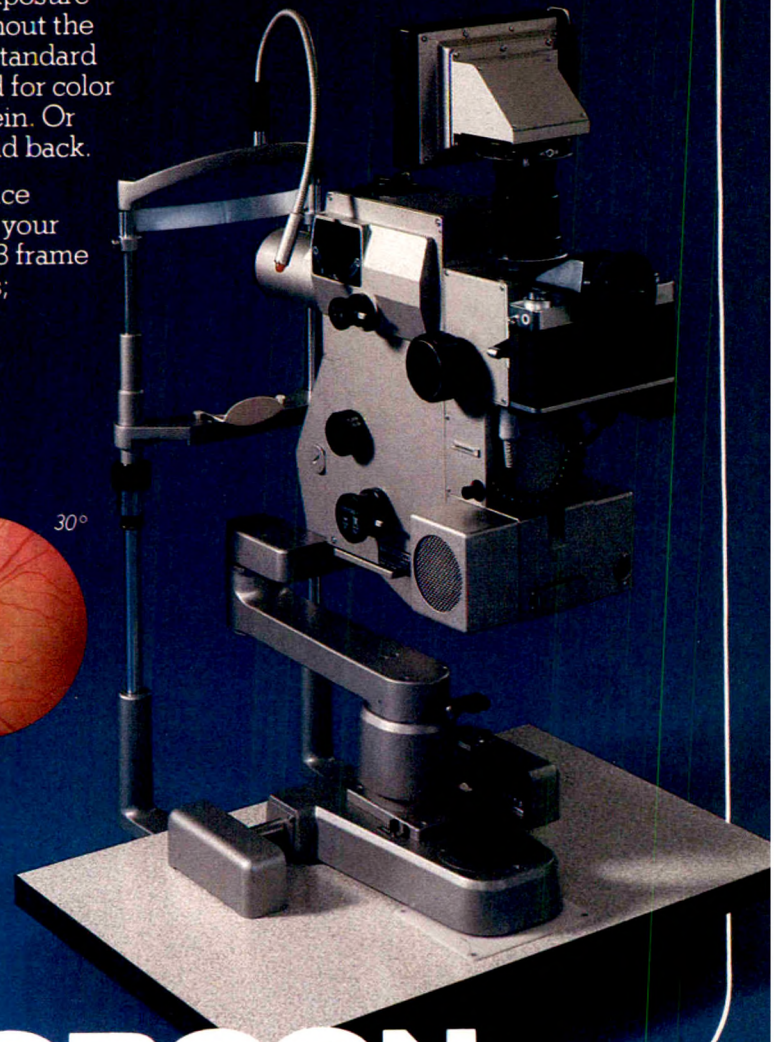
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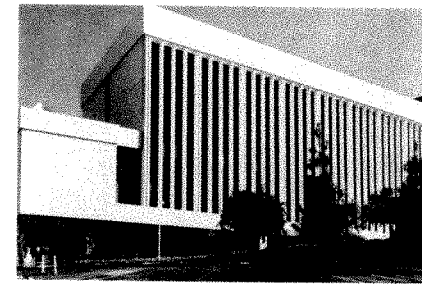
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Assistant Professor of Ophthalmology
USC—School of Medicine, Los Angeles, California

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Diseases Treated by Photocoagulation
Complications of Photocoagulation
Clinical Results of Treatment
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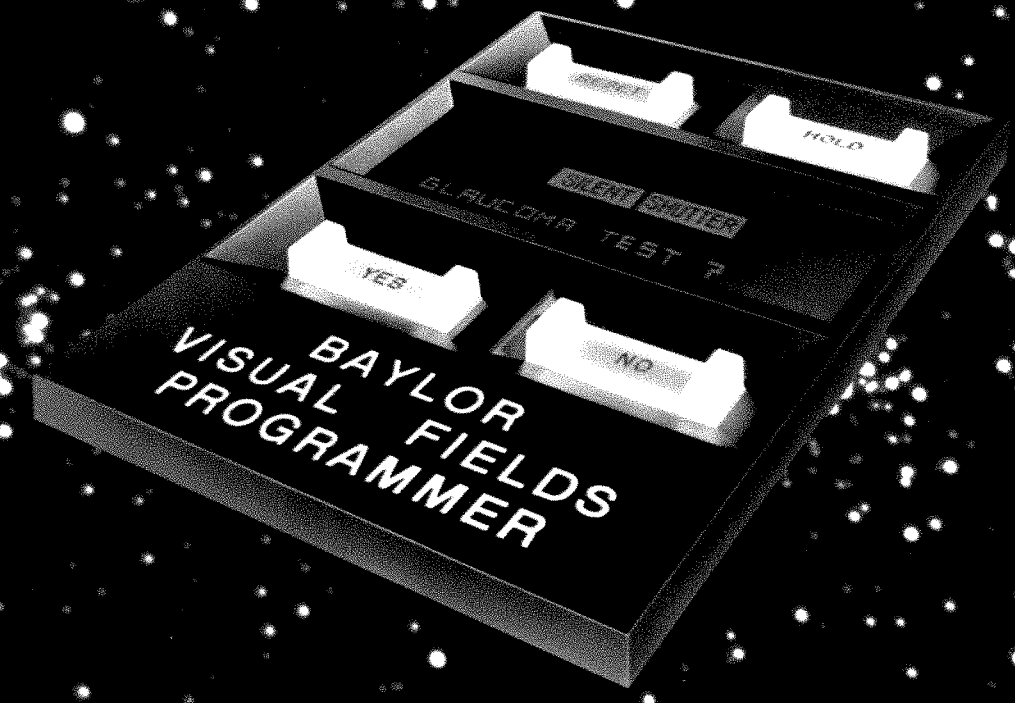
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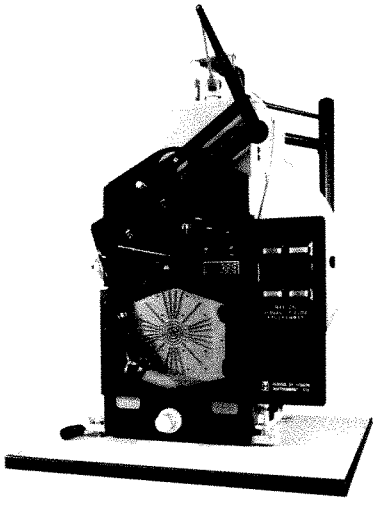
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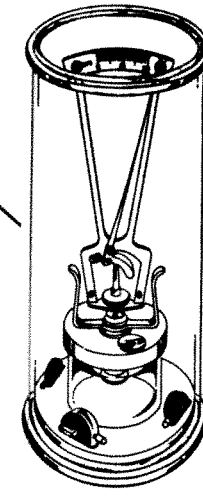
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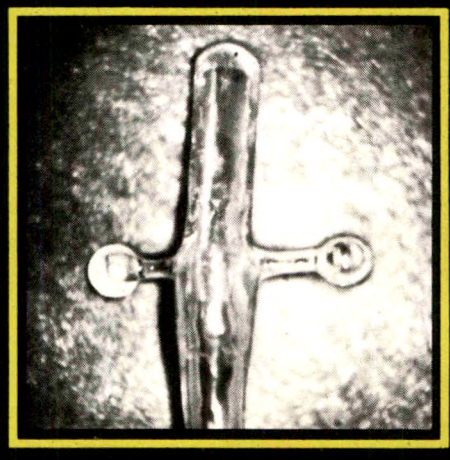


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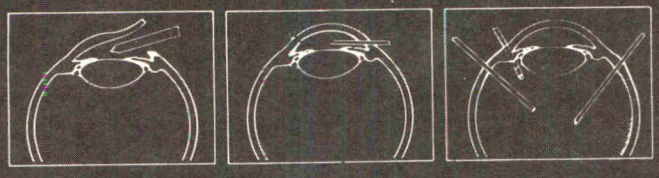
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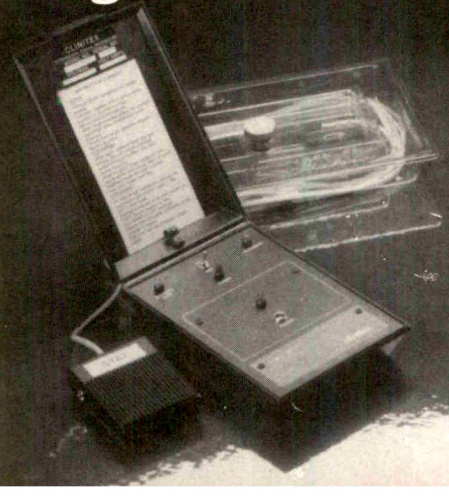


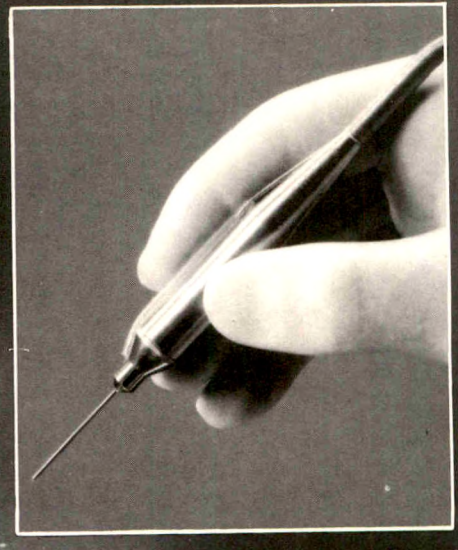
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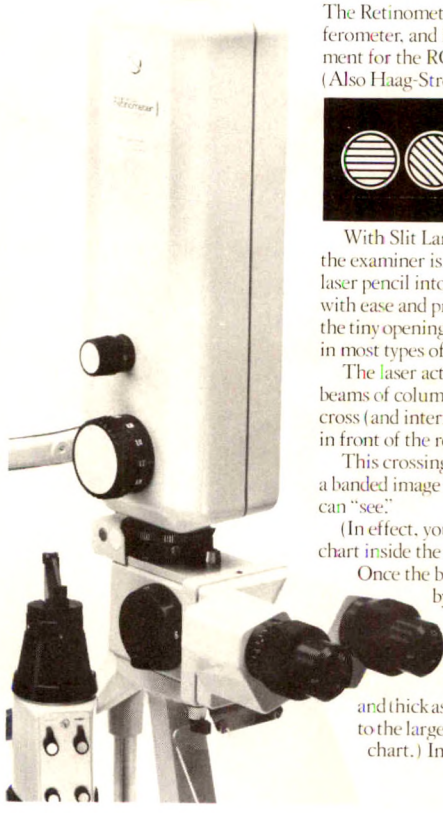
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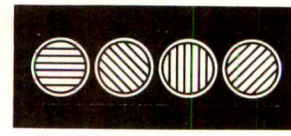
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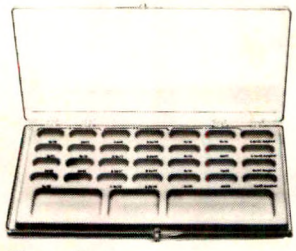
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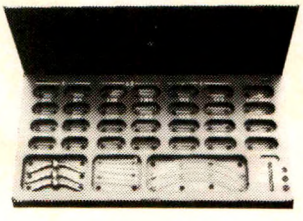
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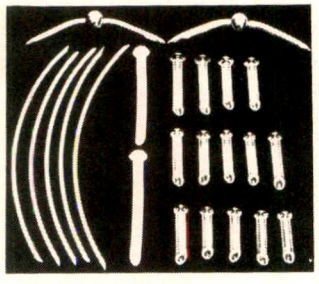
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* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
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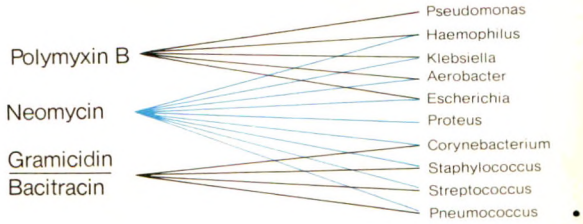
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See adjacent page for brief summary of prescribing information.

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Each cc contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, neomycin sulfate 2.5 mg (equivalent to 1.75 mg neomycin base), gramicidin 0.025 mg. Vehicle contains alcohol 0.5%, thimerosal (preservative) 0.001% and the inactive ingredients propylene glycol, polyoxyethylene polyoxypropylene compound, sodium chloride and purified water.

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Each gram contains: Aerosporin® (Polymyxin B Sulfate) 5,000 units, bacitracin zinc 400 units, neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base), special white petrolatum qs.

Brief Disclosure below applies to the solution and ointment.

INDICATIONS: For the short-term treatment of superficial external ocular infections caused by organisms susceptible to one or more of the antibiotics contained therein.

CONTRAINDICATIONS: Contraindicated in those persons who have shown sensitivity to any of the components.

WARNINGS: Prolonged use may result in overgrowth of nonsusceptible organisms. Ophthalmic Ointment may retard corneal healing.

PRECAUTIONS: Culture and susceptibility testing should be performed during treatment.

Allergic cross-reactions may occur which could prevent the use of any or all of the following antibiotics for the treatment of future infections: kanamycin, paromomycin, streptomycin, and possibly gentamicin.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Complete literature available on request from Professional Services Dept. PML.

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[1] Sinclar, S., Riva, E., Loebl, M.: Blue field entoptic phenomenon in cataract patients. Archives of Ophthalmology 97: 1092, 1979.

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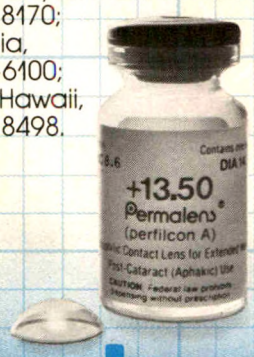
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May 22-23	Pacific Medical Center San Francisco, CA	Arthur W. Allen, MD	Sally Dunmeyer (415) 563-4321 ext. 2761
June 11-12	Georgetown University Washington, DC	Leonard Parver, MD	Mary J. Day (202) 625-2306
June 25-27	University of Iowa Iowa City, IA	Thomas Weingeist, MD	Terry Fanning (319) 356-2215
July 10-11	Hyatt Regency, Dearborn Detroit, MI	Howard Joondeph, MD	Marion Lynch (313) 882-4002
July 24-25	Truman Medical Center Kansas City, MO	Felix Sabates, MD L. Piebenga, MD	Harry Kuba (816) 556-3595
July 27-29	Sanatorio San Camilo Angel Gallardo 899 Buenos Aires, ARGENTINA	Dr. Alberto Zambrano Dr. Edgardo Manzitti Dr. Gustavo Mayorga Argañaraz	Delfor Horacio Cervo Uruguay 390-Piso 13-OF B 1015 Buenos Aires, Argentina Phone 45-3043
Aug. 3-7	Australian Society for Ophthalmology Melbourne or Sydney, AUSTRALIA	G. Waring, MD	
Aug. 7-8	Sheraton Plaza Hotel Rush Presbyterian Medical Center Chicago, IL	Thomas Bennett, MD	Barbara Trejo (312) 942-7095
Aug. 21-22	USC—Estelle Doheny Eye Foundation Los Angeles, CA	Kenneth R. Diddie, MD	Nancy Call (213) 224-7752
Sept. 4-5	The Broadmoor Colorado Springs, CO	Mylan Van Newkirk, MD	Delores Dailey (308) 635-3911
Sep. 18-19	Emory University Atlanta, GA	Travis Meredith, MD Henry Kaplan, MD	Debbie Seay (404) 329-4271
Oct. 1-3	Devers Eye Clinic Portland, OR	Richard Chenoweth, MD	Suzanne Meyers (503) 225-7881
Oct. 16-17	St. Louis Eye Hospital St. Louis, MO	Orion T. Ayer, MD Anwar Shah, MD	Karen Maroney (314) 644-6800
Nov. 19-20	Philadelphia, PA	Alexander J. Brucker, MD	Pat Shengle (215) 662-8675
1982 Feb. 12-13	University of North Carolina Chapel Hill, NC	David Eifrig, MD	Betty Neilson (919) 966-4161
Feb. 26-27	Emory University Atlanta, GA	Travis Meredith, MD Henry Kaplan, MD	Debbie Seay (404) 329-4271
Mar. 26-27	University of South Carolina Columbia, SC	Mark Hammer, MD	(803) 765-7356
Apr. 23-24	Jules Stein Eye Inst., UCLA Los Angeles, CA	Thomas Hanscom, MD	Lu Hendricks (213) 825-4617

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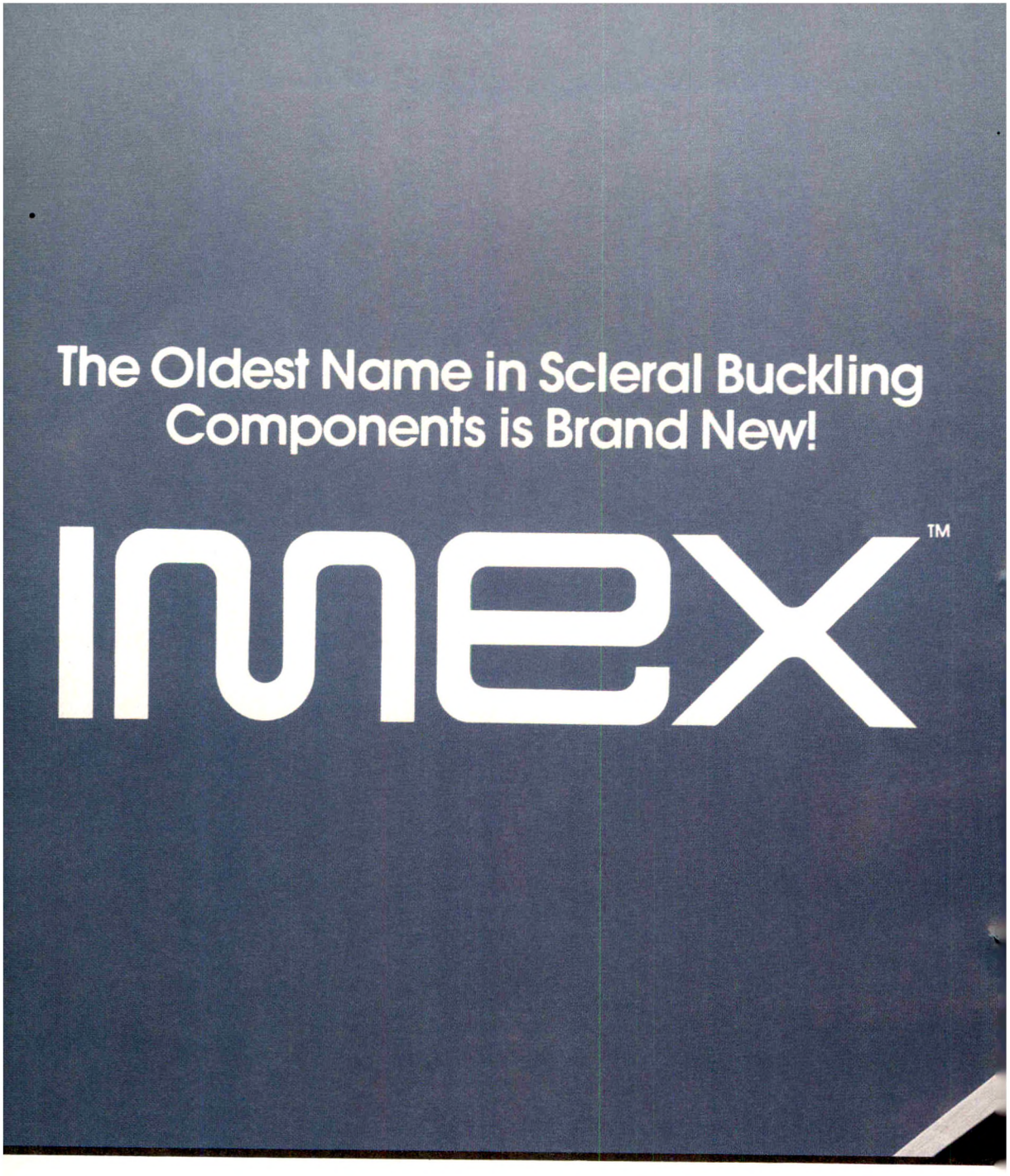
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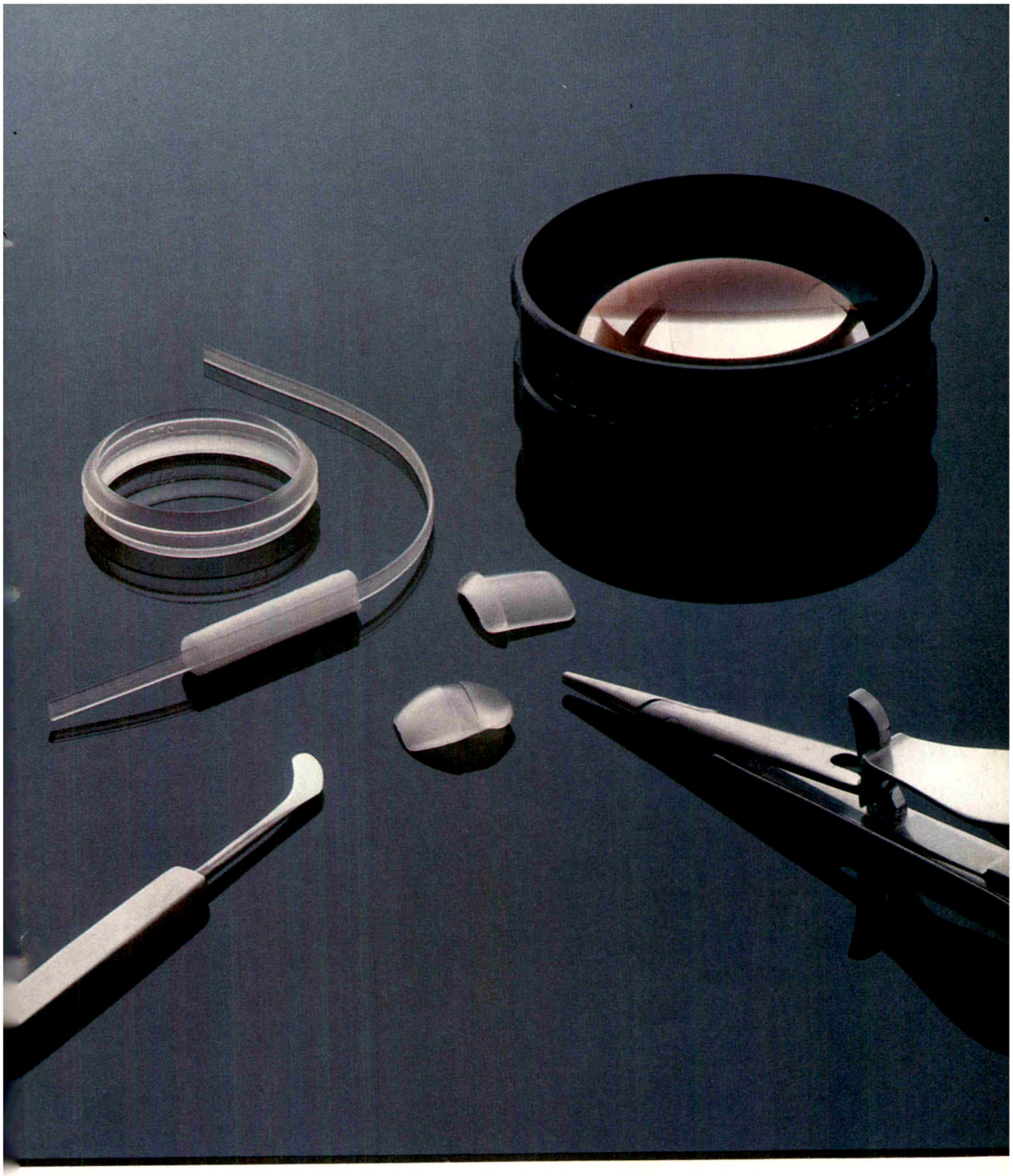
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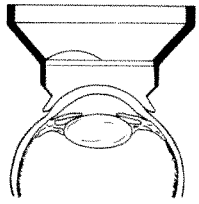
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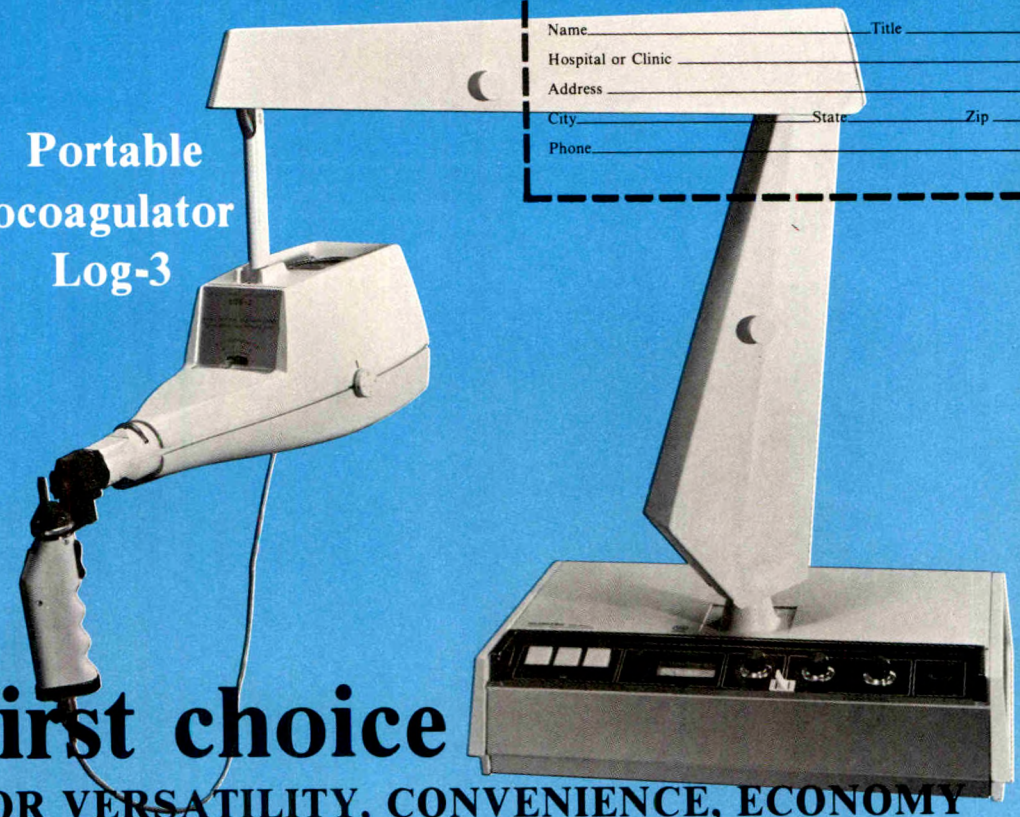
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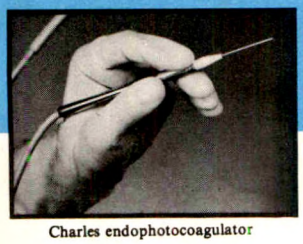
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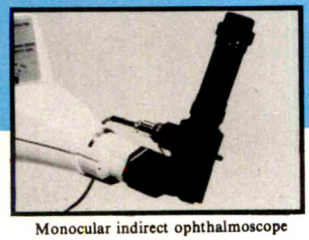
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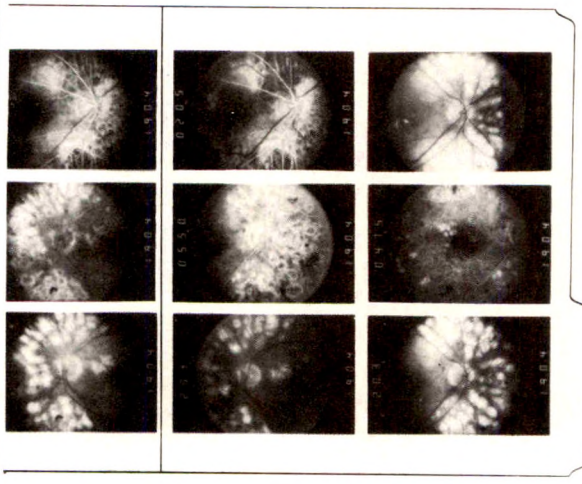
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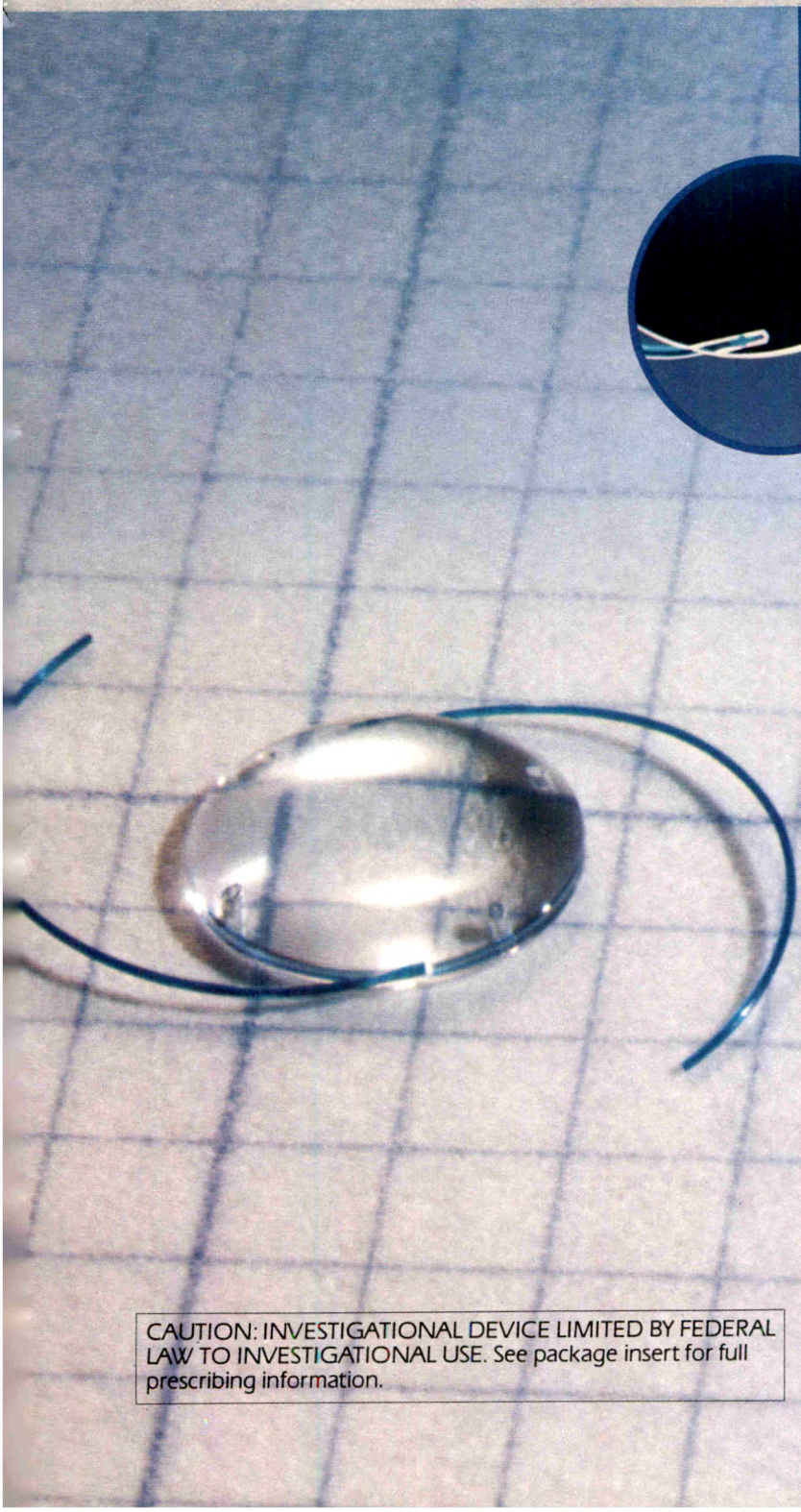
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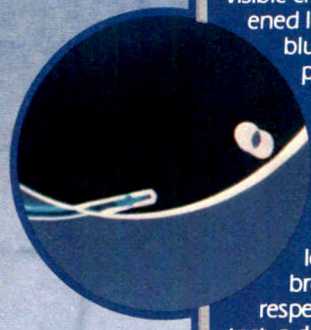
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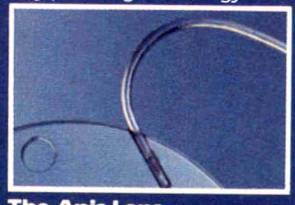
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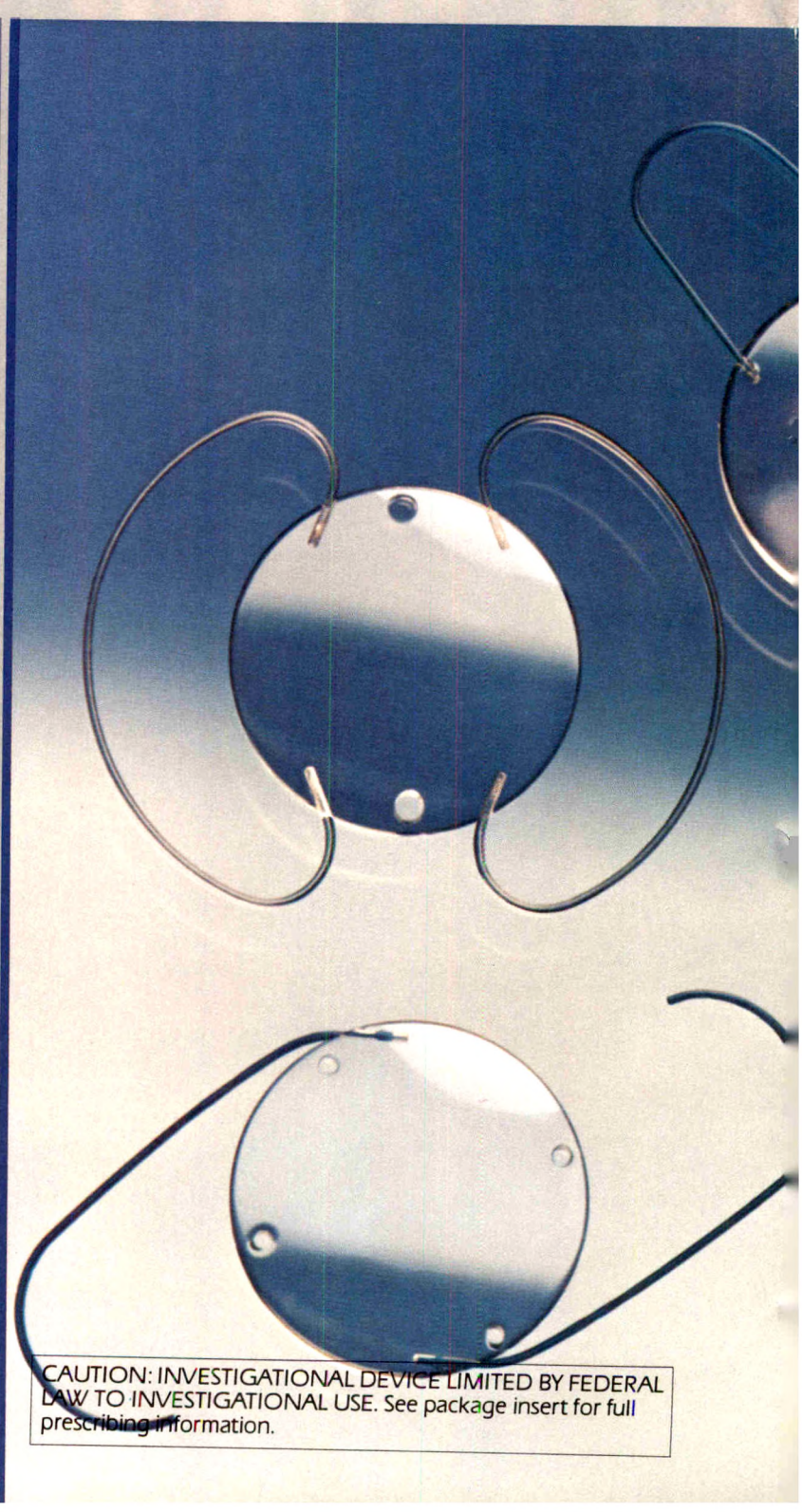
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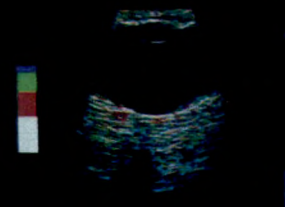


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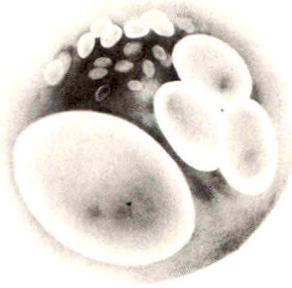
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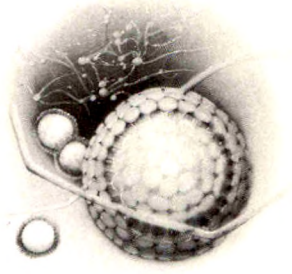
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VOLUME 91 NUMBER 5 MAY, 1981

GOOD VISUAL FUNCTION AFTER NEONATAL SURGERY FOR CONGENITAL MONOCULAR CATARACTS

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AND

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Berkeley, California

We treated eight neonates who had total, monocular, congenital cataracts with surgery, occlusion, and contact lenses. Visual results in all eight patients were good. In five patients visual acuities improved to 6/9 (20/30) or better in the aphakic eye. In three patients visual acuities improved to 6/24 (20/80) or better. Problems with contact lenses probably accounted for the poorer results in two of these three patients. Binocularity was not demonstrated in any of our patients.

Most investigators have considered the monocular congenital cataract a nearly hopeless clinical problem in which successful visual rehabilitation is virtually unknown.[1-17] The failure to obtain good visual acuity in these eyes has been ascribed to the high incidence of associated ocular anomalies, including microphthalmos, nystagmus, foveal dysplasia, and strabismus.[1-3] Such anomalies occur in 30% to 70% of eyes with monocular congenital cataracts.[3] Surgical technique, timing, and complications have also been considered significant factors in determining the final visual outcome.[6-17] However, no consistent relationship between the type of surgery performed or the associated ocular abnormalities and the final visual result has been demonstrated.

Several investigators have suggested that surgery performed in the first few days of life followed by immediate optical correction and amblyopic therapy might result in a favorable visual outcome in these patients.[6-8,14,18] Experiments with animal models disclosed that unilateral eyelid closure causes amblyopia comparable to that occurring in humans, producing morphologic and functional anomalies in several visual centers.[19-22] Von Noorden has argued that if surgery is performed and the aphakic eye optically corrected in the first few days of life, a patient with

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a congenital monocular cataract can be spared the irreversible neural consequences of visual deprivation.[18] A few reports of individual cases have documented the success of such a treatment plan.[23-25] We achieved favorable long-term visual results in eight infants with total, monocular, congenital cataracts who underwent surgery as neonates.

SUBJECTS AND METHODS

From August 1977 to December 1980, 31 patients with monocular congenital cataracts were referred to one of us (C.S.H.) for treatment. Because the published results of treatment of these patients are so pessimistic about a favorable outcome, we decided to treat only the 15 infants who were 6 weeks of age or younger. The parents of four of these children elected not to have surgery performed after they were informed that we considered our treatment plan to be a clinical research project with only theoretical data to suggest that a good visual result might be possible. Therefore, 11 neonates were treated. Eight of these children are now old enough to read a standard Snellen chart or the illiterate E test. These eight children are the subject of this report.

Our attempts to obtain a red-light reflex through dilated pupils showed that each of the infants had a complete, monocular, congenital cataract. In four of the eight patients, the cornea of the cataractous eye was at least 1 mm smaller in its horizontal dimension than in the normal eye (Table 1). In none of the children was rubella, trauma, or persistent hyperplastic primary vitreous suspected of causing the lens opacity. All the neonates were products of uncomplicated pregnancies and none of them had any evidence of systemic disease.

The parents were counseled before the surgery about the necessity of postoperative occlusion therapy and diligent contact-lens care. If the parents elected to have their child participate in the study, surgery was carried out as soon as possible, usually within 24 hours of our first examination of the child and never more than 60 hours later. Preoperative, binocular, total occlusion was instituted in the two patients who did not undergo surgery within 24 hours of the initial examination.[26]

Lens extraction was performed by one of us (C.S.H.); the ages of the infants are shown in Table 1. Initially, we planned to assign these children randomly to two groups to compare the results of standard lens aspiration with those obtained by the newer technique of combined lensectomy and vitrectomy performed with the O'Malley Ocutome. However, preliminary data from our center have indicated that the combined lensectomy and vitrectomy is associated with a significant incidence of cystoid macular edema (unpublished data). Therefore, we have abandoned this technique except for the treatment of complicated infantile cataracts. Hence, six children in this study underwent a standard lens aspiration and only two had a combined lensectomy and vitrectomy (Table 1). In those children who were treated by aspiration we used a constant infusion technique and performed a complete posterior capsulotomy as part of the initial surgery. Keratometry and retinoscopy were performed after the corneal wound was closed and the anterior chamber reestablished. There were no surgical or anesthetic complications in any of the infants.

Postoperatively, binocular total occlusion was maintained until the contact lens was successfully fitted. Contact-lens fitting was initiated in all patients within four days of surgery. Various soft contact lenses were used (Table 1) with initial powers of +26.00 to +30.00 diopters. The power of the contact lens used was calculated to produce a focused image at

TABLE 1

SUMMARY OF CLINICAL DATA FOR EIGHT PATIENTS WITH MONOCULAR CONGENITAL CATARACTS TREATED DURING THE NEONATAL PERIOD

Patient No.	Age		Type of Surgery	Present Contact Lens	Final Visual Acuity in Aphakic Eye	Comments
	At Surgery	Present (yrs)				
1	6 days	3.3	Aspiration	Hydrocurve	6/9 (20/30)	Cornea 1.5 mm smaller in aphakic eye
2	3 days	3.1	Aspiration	Hydrocurve	6/7.5 (20/25)	Dense plaque on posterior lens capsule
3	20 days	3.1	Aspiration	Hydrocurve	6/24 (20/80)	Cornea 1 mm smaller in aphakic eye
4	41 days	3.0	Lensectomy, vitrectomy	Permalens	6/12 (20/40)	—
5	7 hrs	2.9	Aspiration	Hydrocurve	6/6 (20/20)	Cornea 1 mm smaller in aphakic eye
6	10 days	2.9	Lensectomy, vitrectomy	Bausch & Lomb	6/6 (20/20)	—
7	16 days	2.7	Aspiration	Hydrocurve	6/12 (20/40)	Cornea 2 mm smaller in aphakic eye
8	5 days	2.6	Aspiration	Permalens	6/9 (20/30)	—

33 cm from the child. The contact lenses were removed only once a week, at the time of our examination, whenever possible. Retinoscopy was repeated on a monthly basis and changes in the powers of the contact lenses were made whenever necessary. However, frequent fitting problems interfered with this planned protocol.

Because of the extreme steepness of the neonatal cornea and the thickness of these high-plus contact lenses, a contact lens too tightly fitted often led to corneal irritation, edema, or focal epithelial lesions. Nonetheless, permanent corneal damage did not occur in any of the patients as a result of these problems. In two infants (Patients 3 and 7), the contact-lens fitting was initially successful, but for at least six weeks of their first year of life they both were without a contact lens because of complications attributable to contact-lens wear or because of repeated contact-lens loss. Problems of contact-lens tolerance and retention were encountered in several of the other children; in some instances frequent changes in the type, as well as in the power, of the contact lens were required. In the first year after surgery, the average number of contact lenses required by each infant was nine.

Patching therapy was initiated at the same time as contact-lens fitting. Initially, we recommended total occlusion of the phakic eye for 96 hours. After this period of total occlusion, we estimated the visual acuity of both the phakic and the aphakic eye with visual-evoked cortical potentials. We described previously the technique we used for measuring visual-evoked potentials.[27] Briefly, the visual-evoked potentials were elicited with a pattern onset-offset presentation. Presentation of the pattern initiated averaging for 64 iterations. We calculated visual acuity by extrapolating visual-evoked potential amplitude as a function of the fundamental spatial frequency of the checkboards to the baseline voltage or to the noise level of the control obtained by presenting the smallest pattern slide out of focus.[28]

Patching therapy during the infants'

preverbal development was tailored to maintain equal visual-evoked potentials in fellow eyes. Because of the frequent problems with contact-lens fitting and retention, we could not formulate a uniform patching schedule for these children. However, we realized that total occlusion of the phakic eye for 48 hours or more in children less than 2 months old was not necessary to maintain the phakic and aphakic eyes at equal levels of visual acuity. Thus, we tended to patch the phakic eye for four to eight hours per day, depending on the visual-evoked potential measurements. These measurements were repeated at least once each month until the child was able to read the Snellen chart or the illiterate E, at which time patching therapy was monitored by these standard clinical tests. In none of the children, however, was there a major discrepancy between the visual acuity measured with the standard subjective reading charts and that measured with the visual-evoked potential. Strabismus surgery, if necessary, was performed when the visual acuities of the two eyes had remained stable at three consecutive monthly examinations (Table 2).

RESULTS

The visual acuities of the aphakic eye in the eight patients old enough to use either a Snellen chart or the illiterate E are summarized in Table 1. In five of the eight patients, visual acuities improved to 6/9 (20/30) or better after a mean follow-up period of 2.8 years. In the three children visual acuities improved to 6/24 (20/80) or better. Two of the three children with visual acuities of less than 6/9 (20/30) in the aphakic eye had serious problems with their contact lenses and were without optical correction for at least six weeks in the first year of life (Patients 3 and 7). The other patient in the series with a visual acuity of less than 6.9 (20/30) in the aphakic eye was the oldest patient (41 days old) at the time of surgery (Patient 4). The corrected visual acuities in the phakic eyes of all eight patients were 6/6 (20/20) or better. We did not find occlusion amblyopia in any of our patients.

Table 2 summarizes the ocular alignment of these eight patients. We found ocular deviations before surgery in only two patients (Patients 4 and 7). In con-

TABLE 2

OCULAR ALIGNMENT IN PATIENTS WITH MONOCULAR CONGENITAL CATARACTS

Patient No.	Aphakic Eye	Ocular Alignment		
		Before Cataract Surgery	After Patching	After Strabismus Surgery
1	Right	Orthophoric	10° left esotropia	Left monofixation syndrome
2	Right	Orthophoric	20° left esotropia	Left monofixation syndrome
3	Left	Orthophoric	15° left esotropia	Left monofixation syndrome
4	Right	5° right esotropia	15° right esotropia	Right monofixation syndrome
5	Left	Orthophoric	5° right esotropia	No surgery
6	Left	Orthophoric	15° right esotropia	Right monofixation syndrome
7	Left	10° left esotropia	20° left esotropia	10° left esotropia
8	Right	Orthophoric	5° right esotropia	No surgery

trast, all eight patients had clear heterotropia after patching therapy was instituted. This would be expected from the experimental animal models in which monocular occlusion has been shown to reduce the number of binocular units and the degree of binocularity. Four of the eight patients preferred fixation with their aphakic eye once good visual acuity had been attained (Patients 1, 2, 5, and 6). Six of the eight patients underwent surgery to correct the heterotropias (Patients 1 to 4 and 6 and 7) but none of the patients demonstrated binocularity.

Discussion

Monocular congenital cataracts present a frustrating clinical challenge. Although the surgical cataract removal can cause such complications as retinal detachment, membrane formation, glaucoma, and hemorrhage, no definite relationship between the type or timing of surgery performed and the final visual outcome has been established. Most investigators treating monocular congenital cataracts have agreed with Helveston, Saunders, and Ellis[17] that there is "virtually no chance of achieving a good visual result despite early surgery, prompt aphakic correction, and agressive amblyopia therapy." The failure to achieve good visual results has been attributed primarily to the frequency of other ocular defects[3] or to the necessity of surgery in the first few days of life in order to avoid the irreparable consequences of visual deprivation.[18]

The association of monocular congenital cataracts with microphthalmos, strabismus, nystagmus, and other ocular anomalies has been cited frequently as an essential factor in the disappointing visual outcome.[3] These abnormalities have been noted in 30% to 70% of eyes with congenital cataracts.[3,17] In our series of patients, four of the eight patients had microphthalmos in the cataractous eye. Although this made surgery somewhat more difficult in these tiny infants and did account for some of the contact-lens fitting problems, our data did not support the thesis that microphthalmos is an insurmountable obstacle to attaining good visual function.

We detected strabismus before surgery in only two of the eight neonates in this series. They were older neonates, 16 and 41 days old at the time of surgery. In contrast, all eight patients had a significant amount of heterotropia after occlusion. We believe, therefore, that the strabismus associated with monocular congenital cataracts is a secondary phenomenon caused by visual deprivation, and not an intrinsically allied condition.

Although there are undoubtedly cases of monocular congenital cataracts associated with visually disabling retinal or optic nerve anomalies, we do not believe that associated ocular anomalies can account for more than a small percentage of the dismal visual results. Moreover, in eyes without structural or functional anomalies, the visual results have been equally poor.

We agree with von Noorden[8,18] that the major factor responsible for the uniformly poor visual prognosis of monocular congenital cataracts is irreversible deprivation amblyopia. In recent years, animal experiments have shown that the visual deprivation syndrome may be produced during a critical period of sensitivity. This visual deprivation results in a predictable set of behavioral, electrophysiologic, and histologic anomalies of the visual system.[19-22,29,30] These include a decrease in visual acuity in the deprived eye, a reduction in cortical neurons receiving binocular input and in those connected with the amblyopic eye, and a decrease in cell sizes in all layers of the lateral geniculate body that are innervated by the amblyopic eye.

The severity of changes produced at the cortical level after a single week of unilateral eyelid closure in the first month of life is remarkable.[31] However, the extraordinary rapidity of maximal alteration of normal cortical physiology is not paralleled by the slower changes in the lateral geniculate body. Most pertinent to the clinical problem of treating monocular congenital cataracts is the observation that early in this sensitive period the originally deprived eye can recapture its cortical connections completely while the lateral geniculate body still shows the effects of visual deprivation.[31]

The timing and duration of the critical sensitive period in humans has yet to be determined. However, Vaegan and Taylor have argued that the critical period may be deduced by studying children with amblyopia occurring after uncomplicated unilateral cataract and the subsequent aphakic blur.[32] These authors argued that "in unilateral congenital cataract, surgery and optical correction, if indicated, should be completed within the first four months of life." Although we have treated very few children with monocular cataracts who were more than 3 months old, our preliminary data showed that surgery and optical correction are urgent. Certainly, extensive data from previously published series suggested that good visual results are rare in children 6 months old or older.[6,8,10,13,14]

Although we are convinced that good visual acuity can be obtained with some degree of regularity if early surgery is performed in children with monocular congenital cataracts, we are not satisfied with our current treatment program. None of our patients demonstrated any binocular interaction. This may have been the result of severe deprivation of binocular input to cortical cells during the early period of intensive patching. Therefore, we are currently investigating alternative patching regimens that allow more binocular stimulation while maintaining good visual acuity in the aphakic eye. Another possible reason for the lack of binocularity in our patients was the aniseikonia induced by the high-plus contact lenses fitted. We intend to investigate whether the reverse Gallilean telescopic correction advocated by Enoch[33] results in better binocularity.

Despite these problems in our current treatment protocol, we believe our results showed that surgery during the neonatal period is not only justified but probably essential in any successful treatment of monocular congenital cataracts.

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AN ANALYSIS OF TREATMENT OF CONGENITAL GLAUCOMA BY GONIOTOMY

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We used goniotomy as the initial surgical procedure in 34 patients (50 eyes) with congenital glaucoma. An overall success rate of 88% was achieved with one or more goniotomies. The age at disease onset, level of intraocular pressure, and corneal diameter did not significantly influence the surgical outcome. The refractive error proved to be a valuable indicator of operative success or disease progression. A satisfactory visual outcome was associated with treatment by goniotomy.

The surgical treatment of congenital glaucoma by goniotomy can offer a favorable prognosis in this rare condition. Reports of a poor success rate with goniotomy as the initial procedure,[1,2] however, led several investigators to seek alternative surgical techniques.[3,4] These early reports[1,2] mostly involved patients operated on without the benefit of direct angle visualization, which resulted in a high complication rate. The de-emphasis of goniotomy performed with angle visualization in favor of trabeculectomy or trabeculotomy prompted us to review our experience with goniotomy during the last 20 years. Retrospectively, we studied the clinical characteristics of congenital glaucoma and the surgical results obtained by goniotomy in 50 eyes.

SUBJECTS AND METHODS

In children under 3 years old, the diagnosis of congenital glaucoma was established by the presence of signs of increased intraocular pressure such as breaks in Descemet's membrane, loss of corneal transparency, buphthalmos, megalocornea, or an increased cup-disk ratio. The diagnosis was confirmed at the time of surgery by measurement of the intraocular pressure or visualization of the anterior chamber angle structures consistent with congenital glaucoma. In children who were over 3 years old, the presence of megalocornea or buphthalmos was essential for the diagnosis. All patients operated on since 1960 by one of us (M. M. P.) with goniotomy as the first surgical procedure were considered for the study; patients with glaucoma associ-

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ated with ocular or systemic disease were excluded. These conditions included anterior segment dysgenesis, aniridia, rubella, persistent hyperplastic primary vitreous, or Sturge-Weber disease. However, one patient with trisomy 21 and one clinically normal patient with pericentric inversion of chromosome 11 were included. Patients previously operated on were also excluded. A minimum of six months of follow-up care after the first surgery was necessary for inclusion in the study. All goniotomies were performed with the Worst goniolens, which allowed microscopic visualization of the angle structures. Patients were divided into groups according to onset of disease symptoms as determined historically or by initial examination. Five groups of patients were thus determined: (1) those from birth to 5 days old; (2) those from > 5 days to 1 month old; (3) those from > 1 month to 6 months old; (4) those from > 6 months to 3 years old; and (5) those whose ages were unknown.

Visual acuity, corneal diameter, cup-disk ratio, refractive error, and intraocular pressure were obtained, when possible, before surgery and during follow-up care. Corneal diameters were measured to the nearest 0.5 mm, with the patient under anesthesia, by calipers applied to the horizontal corneoscleral limbus. Refractive error was determined by retinoscopy after cycloplegia with 2% cyclopentolate during outpatient examinations on a six-month basis. Intraocular pressure was measured by applanation tonometry with the patient under anesthesia until cooperation permitted measurements during office visits. Statistical analysis was performed to determine any correlative significance of the surgical results to the various clinical findings.

Clinical characteristics—The male to female ratio of the 34 patients (50 eyes) included in the study was 1:1. Eighteen patients had unilateral disease, involving the left eye in 12 patients, and the right eye in six. There was a family history of congenital glaucoma in four of the 34 patients, including two sisters. Two of the patients were premature and one had Down's syndrome.

The most common symptoms or signs that prompted ophthalmologic attention are shown in Table 1.

On initial examination, corneal cloudiness or breaks in Descemet's membrane were noted in 42 of 50 eyes. In all cases, the anterior chamber angle appeared to be consistent with our experience with primary congenital glaucoma. No significant anomalies of the iris or cornea were noted. Additional clinical findings included an impatent nasolacrimal duct in several patients, an ipsilateral cataract in one patient, and strabismus in three patients.

RESULTS

In this study, the criteria for success were a decrease in intraocular pressure to less than 21 mm Hg and the absence of evidence of disease progression (optic nerve cupping, refractive error, and the like) for a minimum period of six months after surgery. All patients were eventually cured surgically, although six eyes required alternative surgical procedures. Nineteen eyes required a second operative procedure; goniotomy was used in 17. Seven eyes, in which the intraocular pressure was normal for at least six

TABLE 1

INITIAL SIGNS OR SYMPTOMS OF CONGENITAL GLAUCOMA

Sign or Symptom	Patients	
	No.	%
Change in size of eyes	19	56
Corneal cloudiness	17	50
Tearing	10	29
Photophobia	3	9

months and thus were considered to be successfully treated by the initial goniotomy, later developed a recurrence of their disease. The first occurred nine months after the initial goniotomy and the remainder during the next five years. Three of these eyes required alternative surgical procedures and are included in the six eyes mentioned previously. Only one patient underwent a third goniotomy, which was successful, that followed recurrence of disease seven years after a successful second goniotomy. The overall success rate (± 1 S.E.) for one or more goniotomies was 88% (± 4.6). The surgical success rate by age at onset of disease is presented in Table 2. Normal intraocular pressure after six months was the criterion for success. When evaluated from the standpoint of surgical success during the entire period of follow-up care, the cumulative chance of success after the initial goniotomy was 0.597 or 60%. Of the 19 failures (39% of 50 eyes), 13 occurred within the first six months and the remaining six during the next five years (Table 3).

The success rate, although highest in the group of patients aged 1 to 6 months old, was not significantly different at various ages of onset, either for the first ($P > .75$) or the second ($P > .38$) goniotomy. Additionally, the overall success rates of 74% for the first and 82% for the second goniotomy were not significantly different ($\chi^2_c(1) = .136$; $P > .7$).

The surgical success rate after the primary procedure was compared to various clinical factors to determine any positive associations.

No statistically significant difference was found for the success rate at the various ages of initial treatment ($P > .1$) (Table 4). Additionally, no statistically significant association was observed with either the corneal diameter or the duration or level of intraocular pressure and the surgical outcome (Tables 5 and 6). The median corneal diameter, when first clinically measured, was 13.0 mm (range, 11.0 to 15.0 mm). All corneal diameters greater than 14 mm were successfully treated with the first surgery except one which was successfully treated by the

TABLE 2

SUCCESS OF GONIOTOMY AT AGE OF DISEASE ONSET

Age	First Goniotomy $\chi^2(4) = 1.41$, $P>.75$			Second Goniotomy $\chi^2(4) = 4.15$, $P>.38$		
	Success		Failure	Success		Failure
	No.	%	No.	No.	%	No.
0–5 days	9	75	3*	1	100	0
5 days–1 mo	6	67	3	3	60	2†
1 mo–6 mos	14‡	82	3	4§	100	0
6 mos–3 yrs	5	71	2	2	67	1‖
Unknown	3¶	60	2	4#	100	0
Total	37	74	13	14	82	3

*Two trabeculectomies as second procedure.
†Two trabeculectomies as third procedure.
‡One of these patients had recurrence of disease and required a second goniotomy.
§One patient required a third goniotomy six years later.
‖Trabeculectomy as third procedure.
¶Two of these patients had recurrence of disease and required a second goniotomy.
#One recurrence treated with cyclocryotherapy.

TABLE 3

SUCCESS OF INITIAL GONIOTOMY DURING ENTIRE FOLLOW-UP PERIOD*

Postoperative Interval	No. of Eyes at Interval Beginning	No. of Failures During Interval	No. of Eyes Withdrawn During Interval	Chance of Failure	Chance of Success	Cumulative Chance of Success
0–1 mo	50	10	0	.2	.8	.800
1 mo–6 mos	40	3	0	.075	.925	.740
6 mos–1 yr	37	1	0	.027	.973	.720
1 yr–2 yrs	36	3	4	.088	.912	.656
2 yrs–3 yrs	29	1	8	.040	.960	.630
3 yrs–4 yrs	20	0	1	0	1.0	.630
4 yrs–5 yrs	19	1	0	.053	.947	.597
5 yrs–10 yrs	18	0	0	0	1.0	.597
10 yrs–15 yrs	18	0	9	0	1.0	.597

*Prepared according to "Life Table" approach; from Corron, T.: Statistics in Medicine, 5th ed. Boston, Little, Brown and Co., 1974, p. 244.

second goniotomy. The initial intraocular pressure, although lower for the patient group that was successfully treated by the first goniotomy (Table 6), was not significantly ($P > .1$) different than that of the failure group.

The mean ± 1 S.D. of the follow-up period for all patients from the last surgical procedure was 65.9 months (± 54 months; median, 60.0 months). Observations of the preoperative intraocular pressure, refractive error, and cup-disk ratio and the postoperative changes determined at the patient's last visit are listed in Table 7.

The mean intraocular pressure at the final visit was significantly ($P < .001$) lower than its initial value. A paired comparison in 41 eyes, where initial and final measurements were available, showed a statistically significant ($P < .03$) reduction of the refractive error from −6.24 diopters (spherical equivalent) to −4.81 diopters. A small yet statistically significant ($P < .003$) decrease in the mean cup-disk ratio was found in a paired comparison between the preoperative observation and the last follow-up observation.

All patients with unilateral glaucoma had anisometropia; the mean refractive difference was 4.93 diopters of myopia on the affected side (S.D. = 3.63; range, −0.75 to 14.5 diopters). The treated eye became normal during the follow-up period. Snellen visual acuity, rather than fixation responses, could be determined in 29 of 50 eyes at the patient's last visit. Two patients (four eyes) had nystagmus. Fifteen eyes had visual acuities equal to or better than 6/12 (20/40); visual acuities were 6/18 (20/60) or better in 20 eyes. None of the eyes developed a cataract or other significant postoperative complication. No patients required adjunctive

TABLE 4

SUCCESS OF FIRST GONIOTOMY

Age at Time of Surgery	No.	%	Total Cases
0–2 mo	5	50	10
2–6 mos	14	78	18
6 mos–1 yr	8	100	8
1 yr	10	71	14
Total	37	75	50
	$\chi^2(3) = 5.99$; $P > .1$		

TABLE 5

INFLUENCE OF CORNEAL DIAMETER ON SUCCESS OF FIRST GONIOTOMY

	Mean Corneal Diameter (mm)	S.D.	Range	Median	No. of Cases
Success	13.14	1.01	11.00–15.00	13.00	35
Failure	12.94	0.86	11.00–14.25	13.00	13
	t_{46} = .791 (unpaired); P >.5				

medical therapy for control of intraocular pressure after surgical success.

DISCUSSION

Since Barkan[5] introduced the technique of goniotomy under direct visual control, it has become the standard in surgical therapy and has significantly decreased the complication rate from "blind" techniques.[6] Reports on the surgical treatment of congenital glaucoma, therefore, have been designed to compare the success of other procedures to goniotomy and to delineate the clinical factors that may influence the surgical success rate.

Most studies have shown an overall success rate for reduction of intraocular pressure to normal in congenital glaucoma to be between 60% and 80% after repeated goniotomies.[7,8] Dissatisfaction with the technique, however, has been ascribed to its poor success rate after the initial procedure.[1,2] Additionally, clinical factors such as an early age at onset of disease and a corneal diameter greater than 14 mm have each been reported to affect adversely the success of goniotomy.[2,9]

In a large combined series of 253 eyes operated on by goniotomy,[1] less than one half were successfully treated by the initial procedure, even though a final success rate of 77% was achieved by five or fewer goniotomies.[1] The largest failure group was that in which the age at onset was from birth to 2 months. In this group, only 55% achieved normal results. Douglas[7] similarly found a success rate of 76%, but two thirds of the eyes required more than one procedure. Additional studies have further supported the impression of a low success rate after the first procedure and a poor success rate in the early age group.[10,11] Other techniques, such as trabeculectomy, have, therefore, been explored as alternative first procedures and have been found to have a higher success rate.[3,4,12]

In this study we found a high success rate (74%) for reduction of intraocular pressure to normal for at least six months after the first procedure with a visualized goniotomy. This rate compares favorably with those for other more complicated techniques. However, the reduction of intraocular pressure after a goniotomy does not guarantee a long-term success

TABLE 6

INFLUENCE OF INITIAL INTRAOCULAR PRESSURE ON SUCCESS OF FIRST GONIOTOMY

	Mean Intraocular Pressure (mm Hg)	S.D.	Range	Median	No. of Cases
Success	28.71	5.15	22–36	30.5	14
Failure	33.25	7.78	20–44	34	8
	t_{20} = 1.652 (unpaired); P >.1				

TABLE 7

CLINICAL FACTORS INFLUENCED BY SURGICAL SUCCESS

	Intraocular Pressure (mm Hg)	Refractive Error (Diopters)	Cup-Disk Ratio
	Initial Measurement		
Mean	30.36	−6.24	0.53
S.D.	6.44	6.75	0.31
No. of patients	22	41	23
	Final Measurement		
Mean	14.97	−4.81	0.42
S.D.	4.36	5.60	0.37
No. of patients	50	41	23
	t_{70} = 11.84 (unpaired) P <.001	t_{40} = 2.23 (paired) P <.03	t_{22} = 3.36 (paired) P <.003

because recurrence of glaucoma can be expected in 10% to 18% of operated eyes.[8] In our study, seven eyes (14%) developed a recurrence of glaucoma after six months of normal intraocular pressure, and five eyes (10%) after one year. The period of normal intraocular pressure necessary for consideration as a successful procedure thereby becomes an arbitrary designation. Thus, we chose six months as an index for success in our study. Based on our data, the cumulative chance of success for a 15-year period after having been successful for the first six months is 0.807 (80.7%). This is a conditional probability that takes into account different observation intervals and withdrawals during the interval. After one year of success in our study of 50 eyes, the chance of success during the remaining years of observation up to 15 years is 0.829 (82.9%). The closeness of these two percentages suggests that success of the procedure can be reliably assessed within the first postoperative six months.

Patients with onset of disease from birth to 2 months of age showed a success rate of 72%, although most of these patients were treated at an older age. When examining patients who were treated between birth and 2 months of age, we found a lower success rate of 59% with the initial procedure (Table 4). Although the data indicate a higher success rate of 80% for those patients older than 2 months at surgery, the difference between the two success rates does not attain statistical significance ($P > .1$). A lower success rate, however, might be anticipated in eyes with physical characteristics of being smaller, or with greater corneal cloudiness, each of which may increase the surgical difficulty of an intraocular procedure. Additionally, cases of congenital glaucoma that occur in the newborn period may have unrecognized associated anterior chamber angle abnormalities.[12]

We found no statistically significant association between the success rate of the first goniotomy and either duration of disease or level of intraocular pressure before treatment.

Another factor, previously stated to be associated with a poor result,[2,9] is a corneal diameter greater than 14 mm. Our study clearly demonstrated that an equal success rate should be anticipated regardless of corneal diameter. Morin, Merin, and Sheppard[13] showed that the corneal diameter is a direct function of intraocular pressure and duration of disease. They reported an increase of 0.5 mm in the corneal diameter for every 10 mm Hg above 20 mm Hg. They further reported a decrease of up to 2 mm in the corneal diameter after successful treatment. Sim-

ilarly, in this study a decrease in corneal diameter of 0.5 to 1.0 mm was observed postoperatively in six eyes, four of which had normal intraocular pressure by the age of 6 months, one at 12 months, and one at 15 months.

The refractive error in congenital glaucoma has not received adequate attention as an important clinical factor in this disease. Haas[8] reported anisometropia of at least 2.0 diopters of sphere or 1.5 diopters of cylinder in cases of unilateral glaucoma with the affected eye being more myopic or less hypermetropic than the normal fellow eye. Both Haas[8] and Douglas[7] suggested that in many cases the anisometropia was responsible for the extremely poor visual result despite surgical success. In this study we found that changes in the refractive error toward myopia may serve as the primary indicator of disease and that, after treatment, changes toward hypermetropia are likely to serve as the best indicator of surgical success of those factors that can be ascertained in the office. In the unilateral cases, the average amount of anisometropia was 4.93 diopters of myopia (that is, less hyperopia) on the affected side. This is consistent with other reports, although our visual results are much better in the unilateral cases than has been previously reported.[8] Eight patients had visual acuities of 6/12 (20/40) or better and two patients achieved bifixation with 40 seconds of stereoacuity. These results confirm the necessity of early surgical intervention and management of potential amblyopia by conventional occlusion therapy, which may be necessary to either reestablish a normal fixation response or to treat significant anisometropia of more than 2.0 diopters in cases of extreme myopia or hypermetropia. In bilateral cases, change in the refractive error toward hypermetropia was primarily achieved in the older age group. This again may support a tendency toward a higher surgical success rate with the initial operative procedure in older patients.

Although most cases of congenital glaucoma occur within the first year of life,[14] a varying degree of severity can be noted with this disease. The heterogeneity in the manifestation of this disease continues to warrant investigation in the infant patient with increasing anisometropia or bilateral progressive myopia.

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SCANNING AND TRANSMISSION ELECTRON MICROSCOPIC STUDIES OF TWO CASES OF PIGMENT DISPERSION SYNDROME

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We examined four eyes obtained post mortem from two patients who had increased intraocular pressure, normal visual fields, and pigment dispersion syndrome. Gross examination, light microscopy, scanning electron microscopy, and transmission electron microscopy disclosed elongated regions on the posterior iris surface of all the eyes. The cell membrane of the iris pigment epithelium was disrupted and there was extrusion of pigment granules at these locations. These areas of iris had a radial distribution (paralleling the course of the packets of zonular fibers) and corresponded to the peripheral iris transillumination defects. Pigment was dispersed in the anterior segment and on the pars plana. In the trabecular meshwork the pigment was free, within macrophages, and within endothelial cells. We observed giant vacuoles in the endothelium of Schlemm's canal.

These findings suggested that friction between the zonular fibers and peripheral iris led to focal disruption of iris pigment epithelium and release of pigment granules. The increased pigment within the trabecular meshwork and Schlemm's canal may indicate that the glaucoma sometimes associated with pigment dispersion is caused by pigmentary obstruction, although congenital imperfections of the outflow channels may also be a factor.

François[1] and Sugar and Barbour[2] were the first to describe pigmentary glaucoma. This rare, usually bilateral, disorder was originally believed to affect primarily young myopic men. Later reports[3-5] demonstrated that this disease also occurs in women and in elderly people. Myopia was found in almost every case. The syndrome is characterized by areas of depigmentation of the iris, pigment deposits on structures of the anterior segment of the eye, and increased intraocular pressure.[2,6] The presence of pigment within the corneal endothelium (Krükenberg spindle), anterior to the inferior Schwalbe line (Sampaolesi line), and in the trabecular meshwork produces a dense pigmented ring in the anterior chamber angle. Although not every patient with the pigment dispersion syndrome develops visual field loss,[5,7] many authors have reported open-angle glaucoma with this syndrome.[2,4,8-11] Several histopathologic studies have suggested that the pigment is released from the iris pigment epithelium.[11-17]

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Two different pathogenetic mechanisms have been proposed to account for the loss of pigment from the posterior iris: (1) an idiopathic atrophy or dysplasia of the iris pigment epithelium of unknown or congenital origin[3,4,15-17]; (2) the release of pigment may be caused by a rubbing of the peripheral (often concave) iris against the anterior packets of the lens zonular fibers in predisposed persons.[11]

Among the pathogenetic mechanisms suggested as causes of increased intraocular pressure in pigmentary glaucoma are obstruction of the intertrabecular spaces by pigment granules or by pigment phagocytosis in the trabecular meshwork endothelium,[4,6,8,9,18,19] damage to the trabecular endothelium by the phagocytosed pigment,[10] and a congenital defect, described as a "goniodysgenetic alteration."[20-22]

These theories concerning the mechanisms of release and dispersion of pigment and the associated glaucoma were primarily based on clinical observations and the histopathologic examination of surgical specimens. Few histologic studies have been done, however, on eyes obtained from patients with clinically documented glaucoma.[15,16]

We studied the gross, light microscopic, scanning microscopic, and transmission electron microscopic findings for four eyes (two patients) obtained at autopsy. These patients had had pigmentary dispersion with clinically documented increased intraocular pressure. We assessed the possibility of mechanical disruption of iris pigment epithelium by friction between the anterior zonular fibers of the lens and the peripheral iris, as suggested by Campbell.[11] Additionally, we examined the ultrastructure of the anterior chamber angle.

Case Reports

Case 1—A 51-year-old woman had had ocular hypertension for two years. Repeated ophthalmologic examinations had shown bilateral Krükenberg spindles and a considerable amount of pigment in the trabecular meshwork causing a 360-degree angle (Shaffer grade III) bilaterally. The cup-disk ratio was ≤0.3 in both eyes. There were no visual field defects. Intraocular pressure ranged from 18 to 26 mm Hg by applanation tonometry. She did not use any medication to decrease the intraocular pressure. She had mild myopia (R.E.: −1.25 and L.E.: −1.5). Before her death from cardiovascular disease, the patient's best corrected visual acuity was 6/6 (20/20) in both eyes.

Case 2—This man had had bilateral Krükenberg spindles and increased intraocular pressure for more than 26 years. Before his death at the age of 81 years, his chamber angles were deep and his intraocular pressure ranged from 20 to 32 mm Hg without therapy.

Fifteen years after his first examination, the patient began taking 1% pilocarpine drops every 12 hours; this was later increased to 4% pilocarpine. This therapy caused an increase in density of the Krükenberg spindle, but decreased his intraocular pressure to 20 to 23 mm Hg in both eyes. He had mild myopia (R.E.: -0.75 -0.5×90 and L.E.: -0.25 -0.25×120). His visual fields, regularly tested with the Goldmann perimeter, were always without defects. There was no abnormal cupping of the optic nerve head. In the years before his death, his visual acuity deteriorated slightly because of early senile cataracts.

Material and Methods

The eyes of both subjects were obtained within two hours of death. The right eye of Patient 1 and both eyes of Patient 2 were fixed immediately in 10% buffered formaldehyde. Patient 1's left eye was fixed in a buffered solution of 1% glutaraldehyde and 4% formaldehyde.

After gross examination and routine processing, we prepared 200 serial sections through the central pupillary optic nerve portion of the right eye in both cases. We prepared meridional segments of the anterior part of the left eye in both cases, with and without parts of the lens, for scanning electron microscopy by the critical-point drying method with liquid carbon dioxide and coating with gold-palladium alloy.

After examining the specimens by scanning electron microscopy (20 kV), we removed one piece of tissue (Case 1), showing a defect of the iris pigment epithelium, from the mount, placed it in 100% propylene oxide for 15 minutes,

embedded it in epoxy resin, and sectioned it through the iris defect for transmission electron microscopic examination.

For each eye, we postfixed samples of the chamber angle and two different areas of the iris and lens in 2% phosphate-buffered osmium tetroxide for two hours. Further processing of these samples included standard dehydration, embedding in epoxy resin, and preparation of semithin sections for orientation and thin sections for transmission electron microscopy.

Results

Gross examination disclosed pigment deposits in the trabecular meshwork and on the back of the cornea, where the deposit formed a Krükenberg spindle (Fig. 1). Dispersed pigment was also present on the zonular fibers, the equatorial region of the lens (Fig. 2) and on the pars plana (Fig. 3).

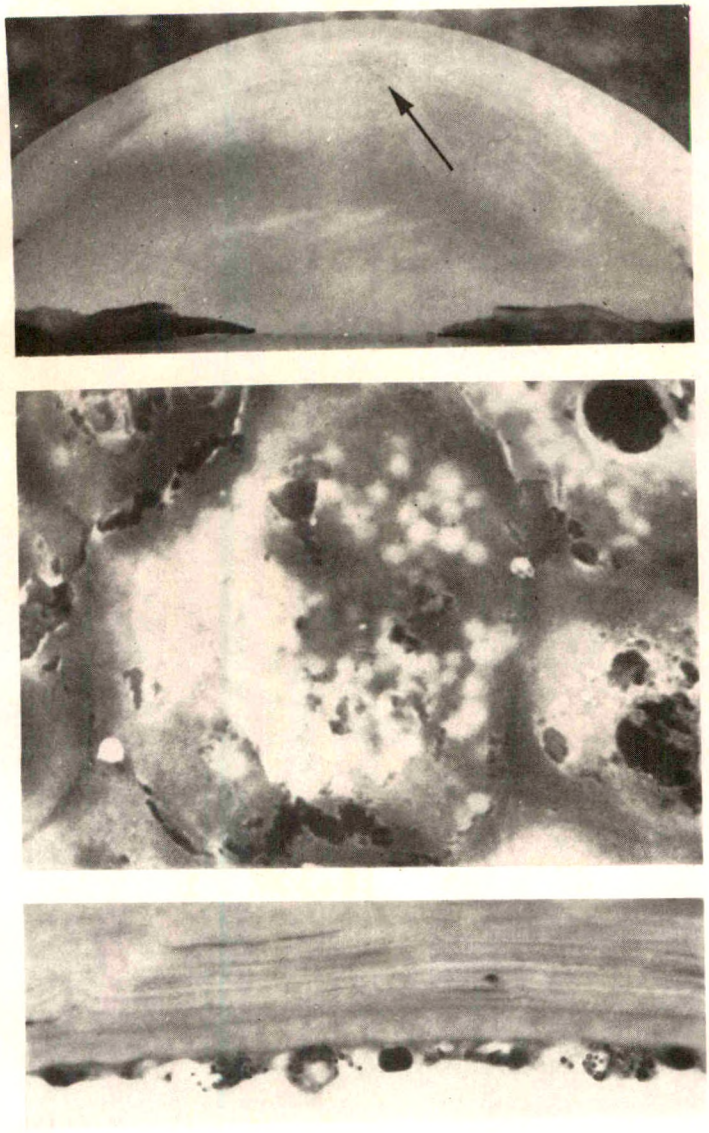


Fig. 1 (Kampik and associates). Case 1. Krükenberg spindle (top) appears as a vertical pigmented area (arrow) in center of cornea (×10). SEM (middle, ×3,000) and cross-sectional (bottom, hematoxylin and eosin, ×220) views show pigment granules within endothelial cells.

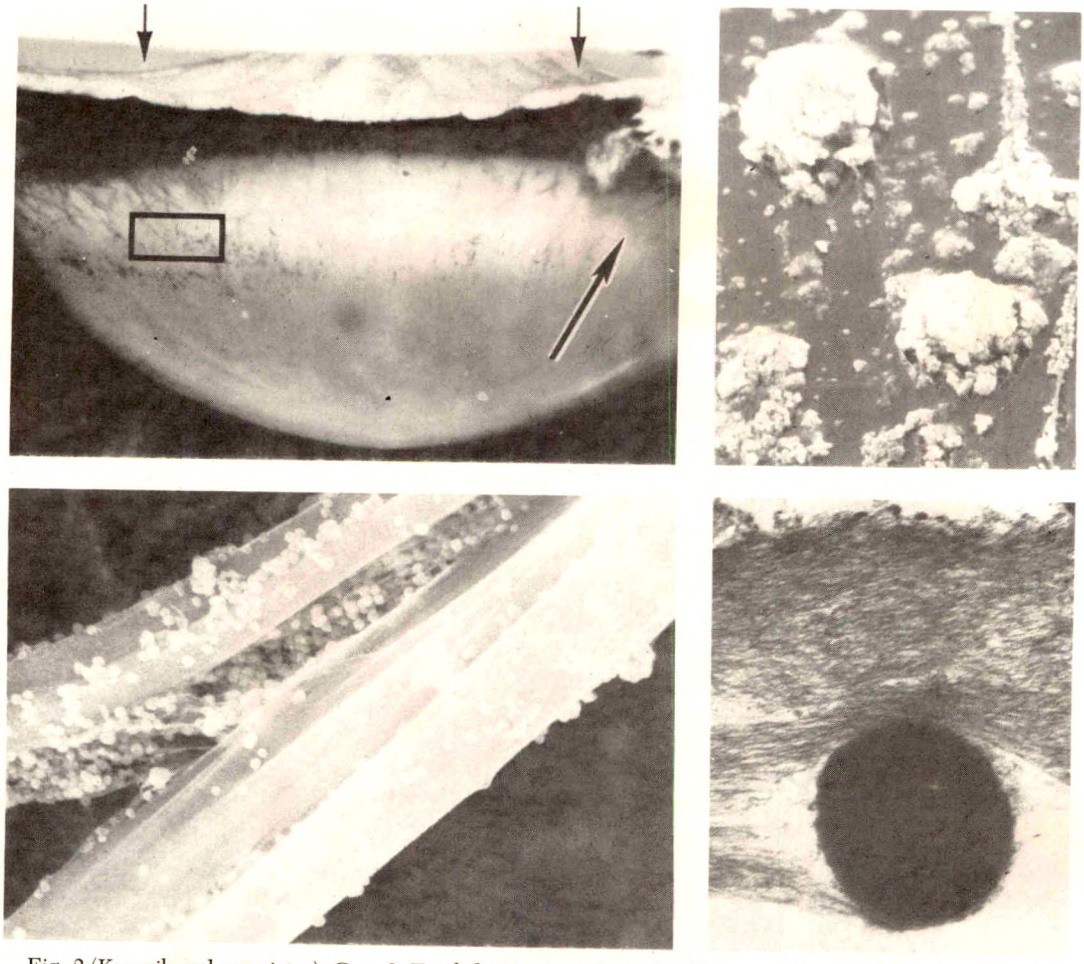


Fig. 2 (Kampik and associates). Case 2. Top left, Pigmented zonular fibers on lens capsule (long arrow) and midperipheral concavity on iris (short arrows) (×10). Top right, SEM view of pigment clumps within rectangle at top left (×800). Bottom left (SEM, ×1,500) and bottom right (TEM, ×30,000), Zonular fibers.

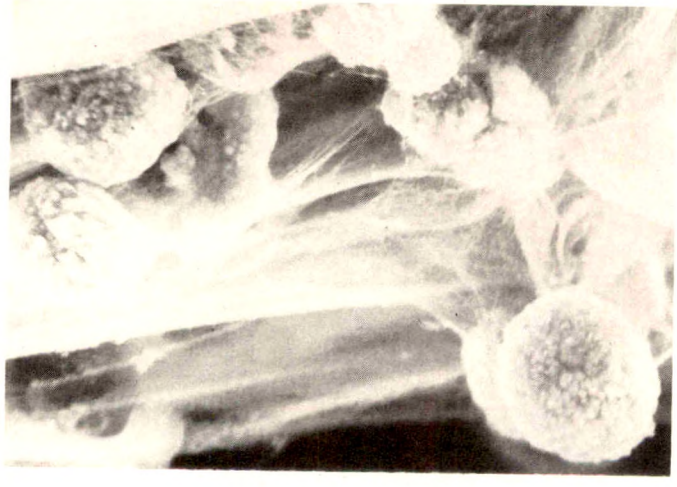


Fig. 3 (Kampik and associates). Case 1. Pigment-laden macrophages on pars plana (SEM, ×1,100).

We observed similar changes in the iris of each eye in both cases. Microscopically, there were many midperipheral radial irregularities on the posterior iris surface (Fig. 4). By scanning electron microscopy, those areas appeared as distortions in the sequence of circumferential ridges in an otherwise smooth posterior iris surface. These localized areas, measuring 20 to 200 μ wide and up to 600 μ long, paralleled the course of packets of the zonular fibers that followed the course of the valleys between the ciliary processes en route to the lens capsule.

At higher magnification, pigment granules extruded from several pigment epithelial cells within the local atrophic areas (Figs. 4 and 5). We noted occasional small membranous and filamentary structures in these areas (Fig. 5). In other atrophic areas, the iris pigment epithelium appeared thin, and bare muscle fibers of the underlying dilator pupillae muscle were visible (Fig. 5, bottom). The surface of the adjacent cells had microvillous processes surrounding some of these defects, giving them a rough appearance (Fig. 4).

Light microscopy showed localized atrophic areas of the iris pigment epithelium in the peripheral one third of the iris. The underlying dilator pupillae muscle showed areas of atrophy and hyperplasia, and the number of clump cells was above normal in the adjacent iris stroma (Fig. 5, inset).

A specimen from Case 1 that showed a linear defect of the iris pigment epithelium by scanning electron microscopy was sectioned through that defect and examined by transmission electron microscopy. In that area, which was about 25 μ wide, the posterior layer of iris pigment epithelium was partially absent and the underlying dilator pupillae muscle was discontinuous for a short distance. The adjacent cells lacked a basement membrane on their posterior surface, and only that region was covered by numerous microvilli (Fig. 6). The center of the defect was filled by a moderate amount of free pigment and cellular debris, including pyknotic nuclei. This eye had been fixed for electron microscopy 'and was well preserved. We saw similar defects in the posterior iris of Patient 2 with transmission electron microscopy.

Additionally, large intercellular and intracellular spaces were present within the atrophic areas of iris pigment epithelium (Fig. 7). We found redundancies of the posterior cell membrane and the basement membrane of the iris pigment epithelium in some areas (Fig. 7). Occasional pigment granules scattered throughout the iris pigment epithelium in both cases were slightly irregular in their electron density and showed some disintegration.

The pigmented and nonpigmented epithelia of the ciliary body were normal, and we observed no abnormalities of the pars plana except for pigment-laden macrophages in some areas.

There was a substantial amount of pigment in the anterior chamber angle (Fig. 8, top), although there were open channels in the trabecular meshwork. The cytoplasm of some of the trabecular endothelial cells was filled by pigment granules of the same diameter as those in the iris pigment epithelium (Fig. 8, bottom). Scattered pigment-laden macrophages were present on the surface of the trabecular pillars (Fig. 9).

We confirmed these findings by transmission electron and light microscopy (Figs. 9 and 10). We found that most of the trabecular pigmentation in these sections was in the uveal meshwork; the amount varied in different areas. We observed giant vacuoles in the endothelium lining Schlemm's canal (Fig. 10, top right) which was almost free of pigment granules. Although the trabecular endothelial cells appeared to be intact in cross

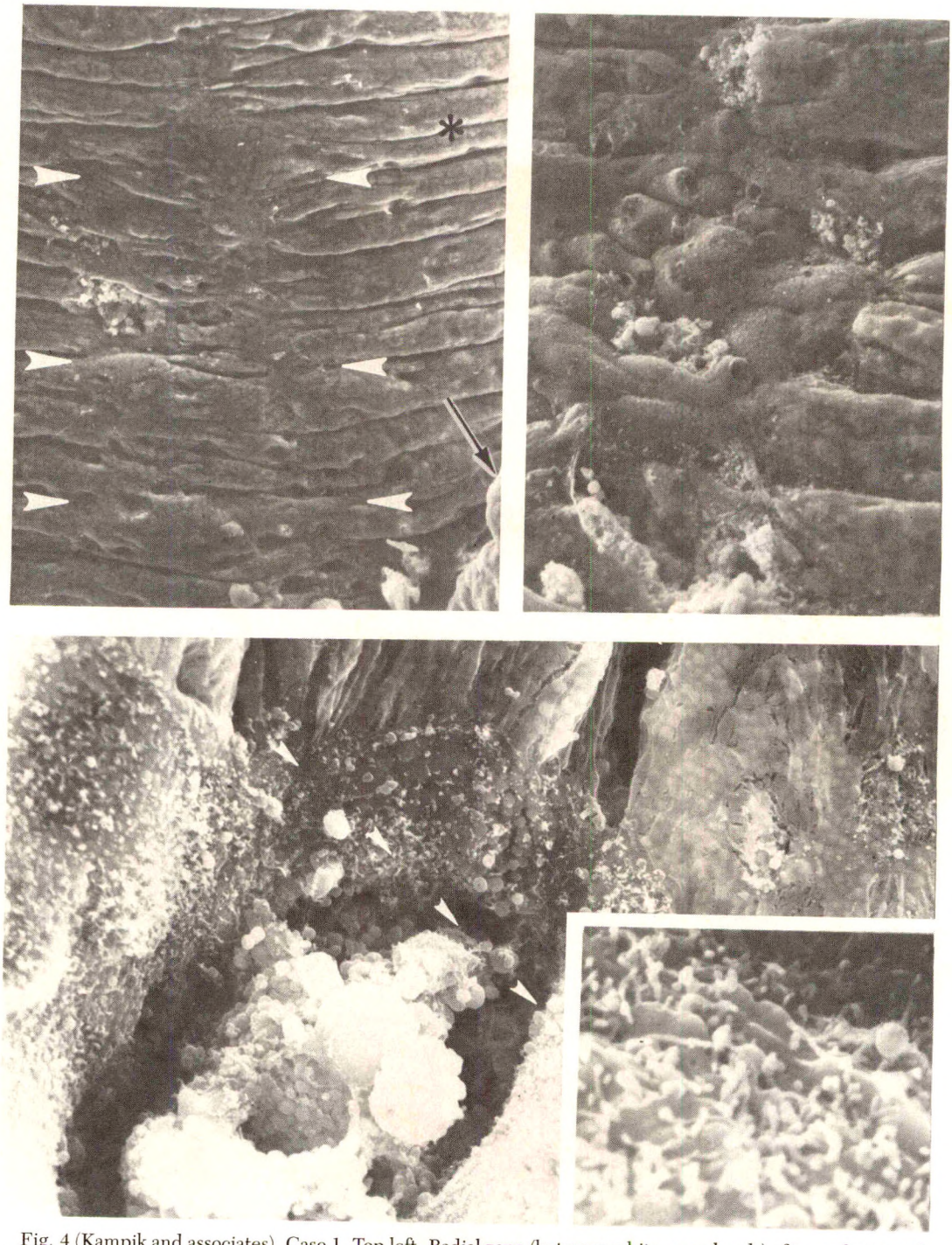


Fig. 4 (Kampik and associates). Case 1. Top left, Radial zone (between white arrowheads) of irregularities of the posterior iris surface runs perpendicular to the normal circumferential ridge of the iris (asterisk) and parallels the course of zonular fibers originating in the valleys between the ciliary processes (black arrow, tip of one ciliary process) (SEM, ×180). Top right, Irregular surface of posterior iris (SEM, ×400). Bottom, Broken pigment epithelial cells. White arrowheads indicate plane of section for TEM in Figure 6 (SEM, ×3,300). Inset, Microvilli partially cover surrounding cells (SEM, ×10,000).

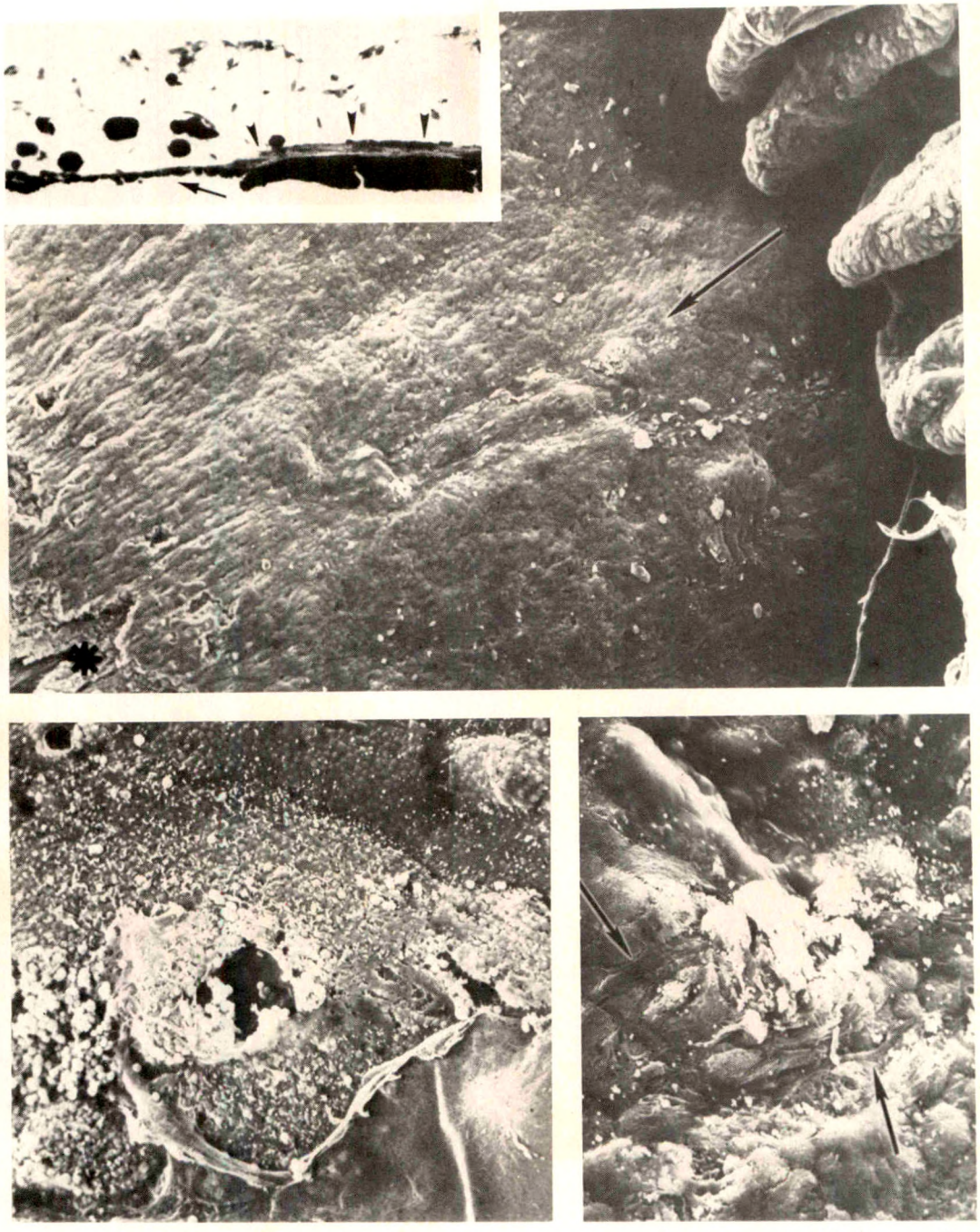


Fig. 5 (Kampik and associates). Case 2. Inset, Cross-sectional view showing atrophy of dilator pupillae muscle (long arrow) and hyperplasia (arrowheads) in midperipheral iris pigment epithelial defect (hematoxylin and eosin, ×250). Top, Two radial grooves parallel course of zonular iris pigment epithelium (SEM, ×80). Bottom left, A membranous structure covers parts of the cell (SEM, ×2,000). Bottom right, Possible bare fibers (arrows) of underlying dilator pupillae muscle (SEM, ×500).

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Fig. 6 (Kampik and associates). Case 1. Higher-power view of iris surface shown in Figure 4. Posterior layer of iris pigment epithelium is partially absent and the dilator pupillae muscle is discontinuous (long arrows). Cellular debris and free pigment granules partially fill the defect. Adjacent cells show microvilli (arrowheads) (TEM, ×27,000).

sections, by scanning electron microscopy some trabecular pillars seemed to lack endothelial cells (Fig. 11). However, we found this in only one eye (Case 1) and we could not identify definitely damaged or missing endothelial cells within the trabecular meshwork by transmission electron microscopy.

Pilocarpine therapy did not appear to have caused any detectable changes in the eyes in which it was used (Case 2).

Discussion

Histologic studies in two cases of clinically documented pigment dispersion syndrome showed that peripheral, localized, and radially oriented areas of the posterior iris surface were the sources of the released pigment granules. We found this pigment in the anterior segment of the eyes as free granules or within macrophages and within endothelial cells of the

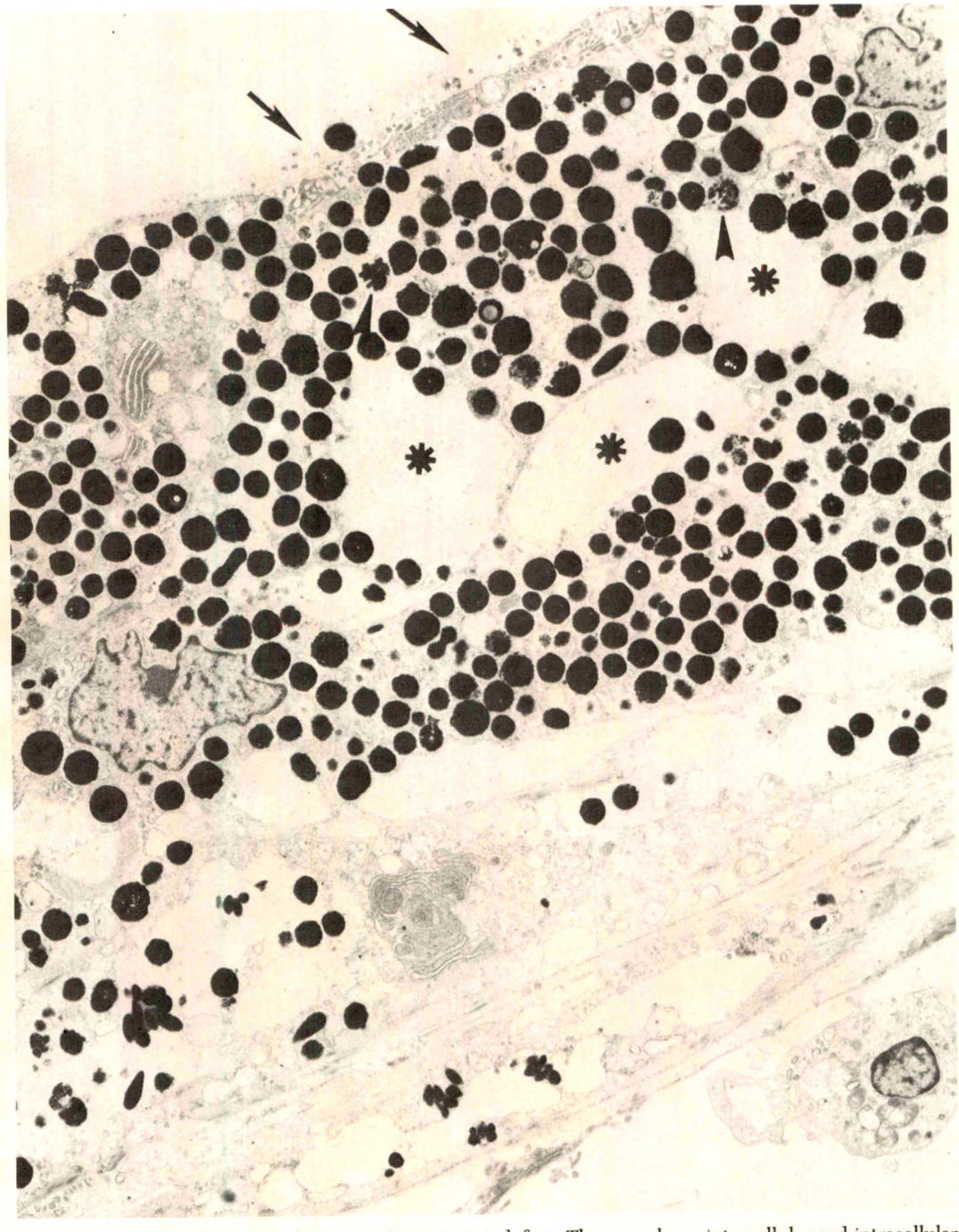


Fig. 7 (Kampik and associates). Case 2. Posterior iris defect. There are large intercellular and intracellular spaces within the epithelial layers (asterisks) and redundancies of the basal plasma and basement membranes (long arrows). Pigment granules are slightly irregular and show some disintegration (arrowheads) (TEM, ×5,000).

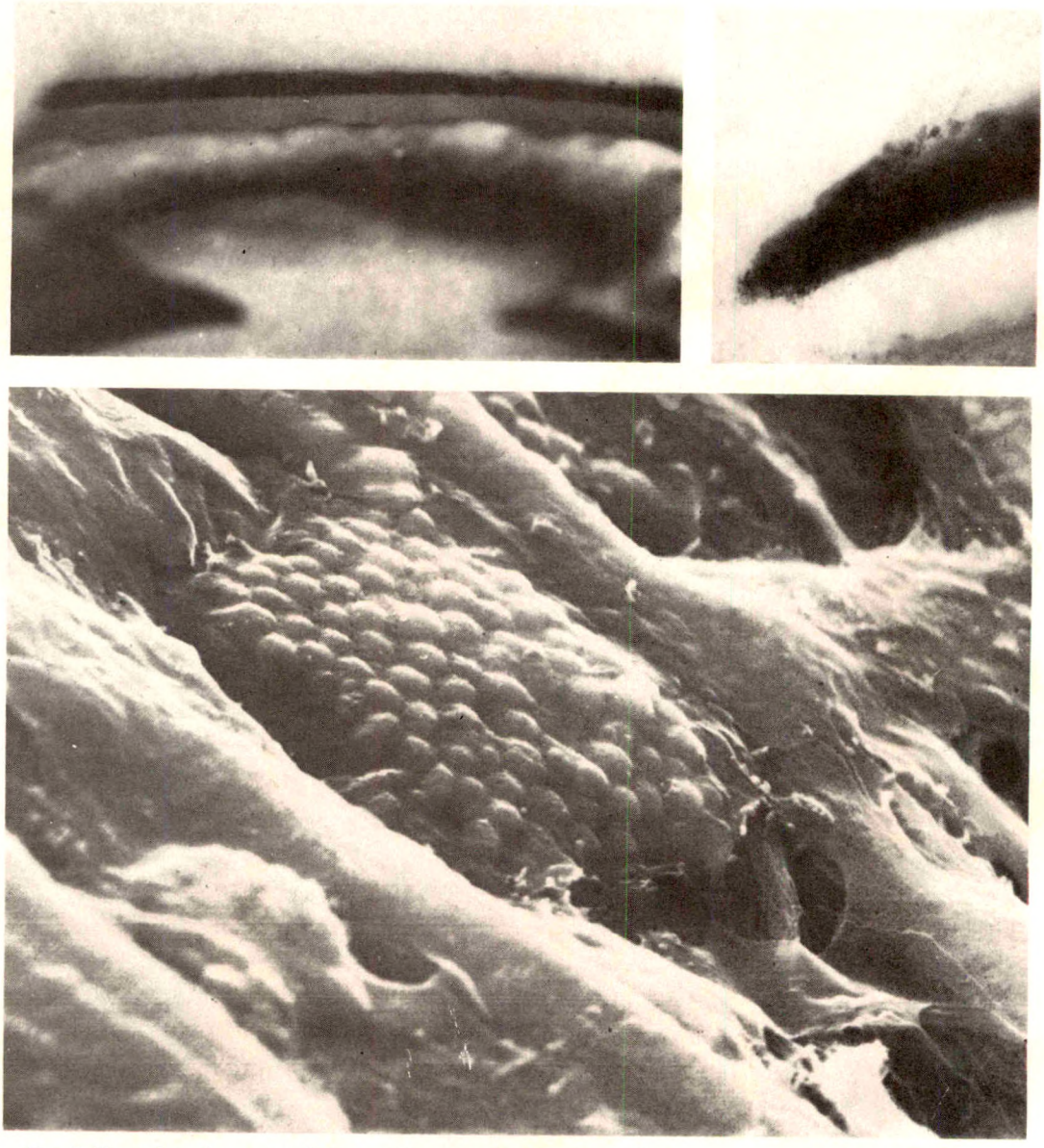


Fig. 8 (Kampik and associates). Case 1. Top left, Intense band-like pigmentation of trabecular meshwork (×8). Top right, Higher-power view shows full-thickness involvement (×50). Bottom, Trabecular endothelial cell has pigment granules with the same diameter as those in iris pigment epithelium (SEM, ×8,100).

cornea and trabecular meshwork. These findings were similar to those described in earlier clinicopathologic studies.[15,16] The scanning electron microscopic observations of the posterior surface of the iris in our cases, however, did not support the idea that these alterations were caused by a congenital or developmental abnormality.[15-17] Our material showed that the radially oriented defects in the posterior iris surface consistently followed the course of groups of zonular fibers of the lens. These findings are similar to those of Campbell,[11] who was able to correlate the number and positions of peripheral iris transillumination

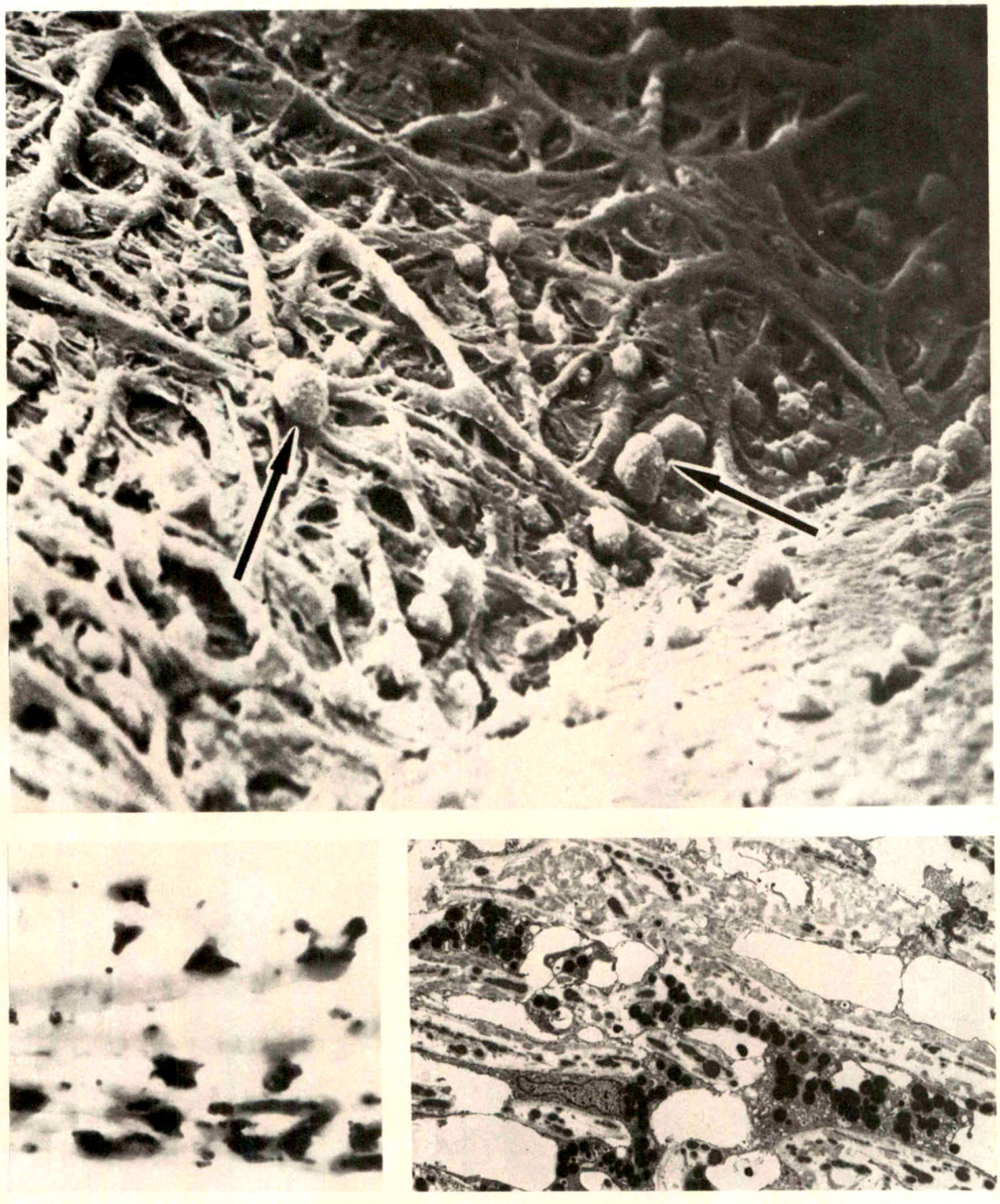


Fig. 9 (Kampik and associates). Case 2. Top, Trabecular meshwork with open channels and scattered pigment-laden macrophages (arrows) on surface of trabecular pillars (SEM, ×830). Bottom left (hematoxylin and eosin, ×900) and bottom right (TEM, ×1,800), Pigmented macrophages and pigmented endothelial cells within trabecular meshwork.

defects in eyes obtained at autopsy with the course of packets of zonular fibers of the lens. He postulated that mechanical rubbing of zonular fiber packets against the posterior surface of the iris leads to the changes observed in the pigment dispersion syndrome. According to this theory, the broken-up iris pigment epithelial cells, the localized atrophic areas, the focal redundancies of the posterior cellular membranes, and the mild disturbances of the iris dilator pupillae muscle

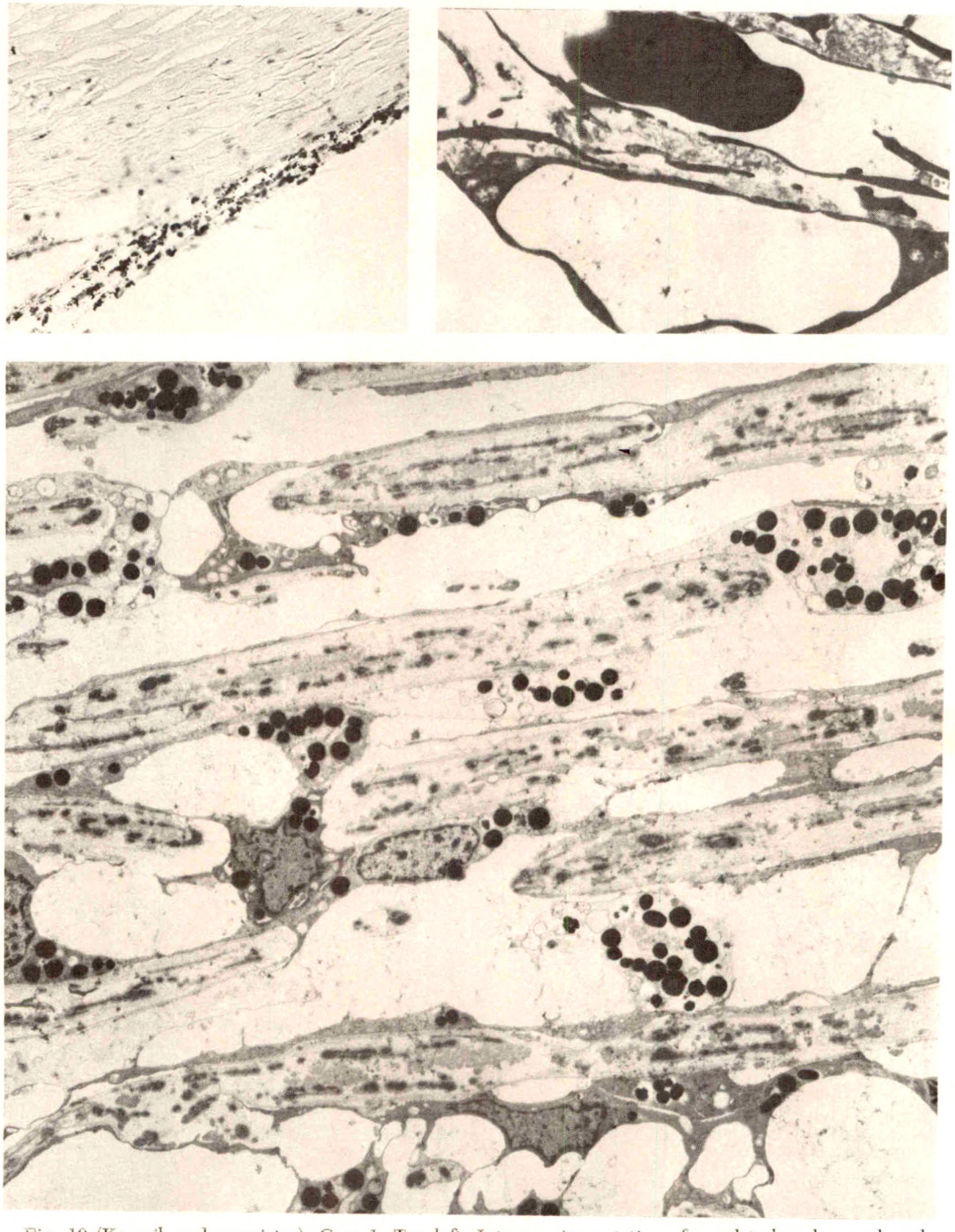


Fig. 10 (Kampik and associates). Case 1. Top left, Intense pigmentation of uveal trabecular meshwork (Fontana-Masson, ×180). Top right, Giant vacuoles in endothelium of Schlemm's canal (TEM, ×5,000). Bottom, Pigment-laden endothelial cells in trabecular meshwork (TEM, ×3,000).

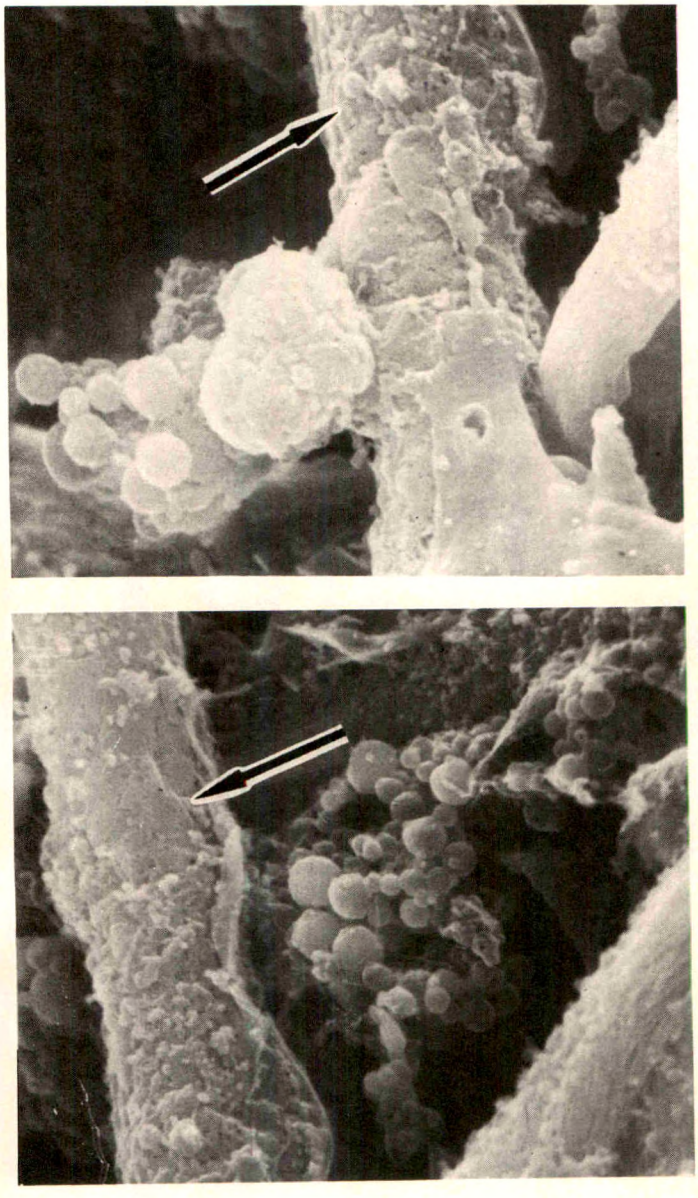


Fig. 11 (Kampik and associates). Case 1. Trabecular pillars partially lack endothelial cells (arrows). Intertrabecular space contains free pigment granules (SEM, ×8,000).

can be related to a mild but constant irritation by mechanical forces of the zonular fiber packets in those locations. This theory also explains the entirely normal structure of the other areas of the iris, since they do not touch the zonular fiber packets.

Sugar[6] noted that patients with pigment dispersion syndrome are usually myopic. Thus, the eyes have deep anterior chambers, allowing contact between zonular fiber packets and iris pigment epithelium. Since many myopic individuals do not have pigment dispersion, those who do may have an underlying anomaly, such as an anomalous insertion of the zonular lens fibers. However, we were not able to demonstrate this, since this

possibility occurred to us after removal and sectioning of the lens.

Krükenberg spindles, intense pigmentation of the trabecular meshwork, and pigment granules on the lens surface and the zonular fibers result from the release of pigment granules from the iris pigment epithelium. The dispersed pigment may be free, in macrophages, or phagocytosed by the endothelium. The Krükenberg spindle is caused by pigment uptake by the corneal endothelium. The heavy pigmentation of the trabecular meshwork is caused by pigment phagocytosis by the trabecular endothelium and by the presence of pigmented macrophages within and on the trabecular meshwork. Although previous studies[12,17] agree on these secondary changes, the relationship between pigment dispersion and an associated glaucoma is controversial.

Some observers described, on the basis of clinical features, the presence of an abnormal number of iris processes[5] or a membranous structure over the trabecular meshwork[22] as a sign of chamber-angle dysembryogenesis. They interpreted these abnormalities as a predisposing factor for glaucoma associated with pigment dispersion. However, we did not identify any anomalies of the anterior chamber angle by detailed histopathologic examination. We cannot, therefore, attribute the increased intraocular pressure in our patients to congenital chamber-angle anomalies.

Several investigators[10,19,23,24] have observed the absence of giant vacuoles of the endothelial inner wall of Schlemm's canal in pigment dispersion syndrome. Although the precise role of these vacuoles has not been completely defined,[25,26] we noted that giant vacuoles were present in our tissue specimens from patients with increased intraocular pressure. Since others have demonstrated that particulate matter and aqueous humor leaving the eye by bulk outflow do pass (at least in part) through such giant vacuolar structures,[27] the presence of vacuoles indicates that this mechanism of outflow was not completely absent.

Increased pigment within the trabecular meshwork may cause increased intraocular pressure by physically obstructing the trabecular outflow channels.[4,6,8,9,18] Richardson, Hutchinson, and Grant[10] found apparently damaged trabecular endothelial cells; they ascribed them to a possibly damaging effect of the released pigment granules. We did find abnormally large amounts of pigment within trabecular endothelial cells. Furthermore, some trabecular pillars lacked endothelial cells. Although this may have resulted from imperfect fixation, it is also consistent with damage to and loss of trabecular endothelial cells. Possibly it was only a sign of intense phagocytic activity. Rohen and van der Zypen,[28] in experimental animal studies, noted that intense phagocytosis can change the shape and appearance of the trabecular endothelial cells, often detaching them from the trabecular pillars and causing them to leave the meshwork via Schlemm's canal. Thus, pigment-laden endothelial cells and macrophages may create a partial obstruction of the intertrabecular spaces, augmented by detached trabecular endothelial cells, and leading to increased intraocular pressure.

Although there was considerable pigment within the trabeculum in our specimens, there still were patent openings connecting the anterior chamber and the juxtacanalicular area in each zone of the angle. Had the meshwork holes been completely obstructed by pigment, we would have concluded that pigment dispersion was solely responsible for the increased intraocular pressure. This was not the case, however, either in our material or in previously reported chamber-angle specimens from eyes with this syndrome. Also, we know that some

eyes have prominent pigment deposits in the angle without increased intraocular pressure. Thus, our data do not rule out the possibility that eyes developing glaucoma with pigment dispersion have underlying abnormal aqueous humor dynamics. In this case, increased intraocular pressure would result from the combination of the degree of pigment release, its duration, and the outflow capacity of the individual eye.

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THE NORMAL CUP-DISK RATIO

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We determined the cup-disk ratios of 580 individuals, ranging in age from 4 to 91 years, with a Hruby lens; we also determined the cup-disk ratios of 289 of the 580 with a direct ophthalmoscope. All had applanation pressures of 18 mm Hg or less. There was no linear relationship (covariation) between cup-disk ratio and refractive error or intraocular pressure, but there was a tendency toward increasing cup-disk ratio with increasing age. The mean cup-disk ratio in the group examined with the Hruby lens was 0.38 and that in the group examined with the direct ophthalmoscope was 0.25. With either method almost all eyes had cup-disk ratios of 0.7 or less. In 9% of the eyes the cup was ovoid. In only 10% of these cases was the vertical cup-disk ratio greater than the horizontal cup-disk ratio, and in only one case was this difference 0.2 or more.

The Hruby lens method consistently gave a slightly larger cup-disk ratio than the direct ophthalmoscope did. However, there was seldom a disparity of more than 0.2. The Hruby lens or biomicroscopic lens should be used to determine the fullest extent of the contour of the cup. Any cup-disk ratio of 0.7 or more, any vertical cup-disk ratio larger than the horizontal cup-disk ratio, and any disparity between the direct ophthalmoscope estimation and Hruby lens estimation of more than 0.2 should be viewed with suspicion.

During the last 60 years many investigators have described the normal cup-disk ratio and have suggested standard means for measuring it.[1-18] These investigators hoped that a better definition of normal cups would provide a better definition of abnormal cups. Some methods take into consideration the sloping edge of the cup, color change, and the area and depth of the cup.[7-9] One study used the biomicroscope,[16] but the studies have primarily used the direct ophthalmoscope with a monocular view.[1-5] Still, there is much disagreement about the relationships of age, sex, refractive error, and intraocular pressure to cup-disk ratios.

Photography is the best way to observe an optic disk over a period of time.[1,6,9-15] These previous studies used small samples and not all of them used stereoscopic photography. Although photographs are useful, they often lack the subtle details found with careful examination with a slit-lamp microscope and Hruby or biomicroscopic lens on any one visit.

In the usual clinical setting, the best way to examine an optic disk is stereoscopically with a Hruby or biomicroscopic lens.[7,8,19-21] We undertook a study

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with these tools to determine the cup-disk ratio in a normal population, using a fast, accurate, standardized method, and to discover the relationship of the cup-disk ratio to age, sex, refractive error, and intraocular pressure.

To determine the extent of the contour of the cup, we located the point at which the nerve fibers angled posteriorly away from the retinal surface. Enlargement begins at the surface of the optic disk[22] and can be determined with a large degree of accuracy by examining the break of a slit beam as it traverses the excavation of the optic disk.[8] Part of the study involved a comparison of the cup-disk ratios determined by the Hruby lens and the direct ophthalmoscope. We made no attempt to quantify the color or depth of the cup.

Subjects and Methods

We included 580 people (1,160 eyes), ranging in age from 4 to 91 years. These individuals had no history of optic nerve disease. All patients underwent a complete ocular examination and had applanation pressures of 18 mm Hg or less. We used tropicamide 0.5% and phenylephrine 5% to dilate the pupils. Media clarity was adequate in all eyes. The subjects cooperated in a careful examination of the optic disk with a Zeiss biomicroscope and a Hruby lens by the same observer (E. F. C.). For a large subgroup of the study (289 patients; 578 eyes) estimates of the cup-disk ratio were made with the direct ophthalmoscope as well. The data were retrospectively analyzed to determine the cup-disk ratios obtained with the slit-lamp and Hruby lens and with the direct ophthalmoscope and to correlate them with age, refractive error, intraocular pressure, and sex. We based the recorded cup-disk ratio on a grid system previously advocated by others.[17,19,22] The horizontal and vertical cup-disk ratios were estimated as decimals to the nearest tenth. Refractive errors were taken as the algebraic sum of spherical error and the spherical equivalent of the cylindric error. We used a computer to collate the data.

We used Pearson's product moment correlation (r) to analyze linear covariant relationships statistically and Student's *t*-test to compare groups of data. A probability value of .05 or less was used as the level of significance.

Results

Comparison of the direct ophthalmoscope and the Hruby lens—We examined 289 patients (578 eyes) with both a slit lamp with a Hruby lens and a direct ophthalmoscope to determine the cup-disk ratios. Figure 1 shows the cup-disk ratios by the two methods for the same 578 eyes. A shift to higher values is noticeable in the Hruby lens measurements. Of the direct ophthalmoscopic cup-disk ratio measurements, 100% were 0.6 or less but 1.9% of the Hruby lens measurements were more than 0.6.

The mean cup-disk ratio with a Hruby

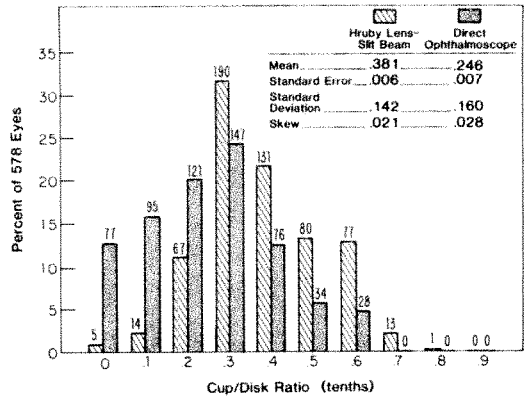


Fig. 1 (Carpel and Engstrom). Comparison of measurements made with the direct ophthalmoscope and the biomicroscope with Hruby lens. The frequencies of cup-disk ratio measurements with the biomicroscope with Hruby lens are shifted to higher values. This shift is reflected in the higher mean for the Hruby lens measurement. The frequency curve for the biomicroscope with Hruby lens is closer to a gaussian distribution, as reflected in the skew.

TABLE 1

DIFFERENCES BETWEEN THE CUP-DISK RATIOS MEASURED WITH THE HRUBY LENS AND THE DIRECT OPHTHALMOSCOPE IN THE SAME EYE

Difference Between Measurements	Eyes No.	Eyes %
Hruby < direct by 0.1	1	0.2
Hruby = direct	131	22.6
Hruby > direct by 0.1	199	34.4
Hruby > direct by 0.2	171	29.5
Hruby > direct by 0.3	70	12.1
Hruby > direct by 0.4	7	1.2

TABLE 2

OVOID CUPS

Difference Between Axial Cup-Disk Ratios*	Eyes No.	Eyes %
Vertical > horizontal by 0.2	1	1.0
Vertical > horizontal by 0.1	9	8.8
Horizontal > vertical by 0.1	73	71.6
Horizontal > vertical by 0.2	18	17.6
Horizontal > vertical by 0.3	1	1.0

*Average vertical cup-disk ratio was 0.40 ± 0.13; average horizontal cup-disk ratio was 0.49 ± 0.13.

lens in all 1,160 eyes was 0.35, whereas the mean ratio was 0.38 in the 578 eyes that also underwent a direct ophthalmoscopic evaluation. The mean cup-disk ratio in this same group with the direct ophthalmoscope was 0.25.

In 131 of the eyes, the cup-disk ratio was the same by the two methods (Table 1). The cup-disk ratio with the Hruby lens was smaller than that with the direct ophthalmoscope in only one eye. The slit lamp showed no cupping in this eye, whereas the direct ophthalmoscope measured a cup-disk ratio of 0.1. However, 248 eyes had a cup-disk ratio of 0.2 or more with the Hruby lens; 77 of these showed a difference of 0.3 or 0.4. In those cases where the difference between the Hruby lens and the direct ophthalmoscope was 0.2 or more, there was no significant correlation with intraocular pressure or age. When the *t*-test was used to differentiate between these sets of data, the *t* values were 20.9 in the right eye, 24.9 in the left eye, and 32.2 in both eyes, a highly significant difference ($P < .0005$). The two sets of data, however, had a high degree of linear covariance ($r = 0.78$).

Comparison of horizontal cup-disk ratio with vertical cup-disk ratio—Careful examination of these cups with the slit lamp disclosed that 102 of the 1,160 eyes were not round but ovoid (Table 2). We measured these cups both horizontally and vertically. The horizontal cup-disk ratios are included in the overall measurements for the rest of this study. The average horizontal measurement for ovoid cups was 0.49 (± 0.13). This was more than 0.1 disk diameter larger than the mean cup-disk ratio of 0.35 (± 0.14) in the nonovoid group. The average vertical cup-disk ratio was smaller than the horizontal by almost 0.1 disk diameter. Only ten eyes, or 10% of the ovoid cups, had a vertical axis larger than the horizontal axis, and only one cup had a vertical axis 0.2 larger than the horizontal.

Comparison of the right and left eyes—We found that 365 of the patients had the same cup-disk ratio in both eyes. Only 14 of the patients had a difference of 0.2 or more between the two eyes.

Cup-disk ratio and intraocular pressure—Neither the Hruby lens nor the direct ophthalmoscopic findings demonstrated linear covariance of cup-disk ratio with intraocular pressure when the two sets of data (cup-disk ratio and intraocular pressure) were cross-tabulated with Pearson's test. In individuals more than 60 years of age, there was a slight tendency for a linear covariation of increasing cup-disk ratio with increasing intraocular pressure by the direct ophthalmoscope ($r = 0.41$). We found no linear covariance

when evaluating patients older than 60 years with a Hruby lens.

We found a significant difference in the cup-disk ratios of eyes with higher and lower intraocular pressures. The group with intraocular pressures of 10 mm Hg or more included 1,088 eyes. Seventy-two eyes had intraocular pressures of less than 10 mm Hg. The mean cup-disk ratios were 0.35 ± 0.14 for the eyes with the higher intraocular pressures and 0.32 ± 0.15 for those with the lower intraocular pressures ($t = 2.14$; $P < .04$). Using intraocular pressures of 13 mm Hg or more or 12 mm Hg or less gave similar and statistically significant results ($P < .02$).

Dividing the eyes into two groups, one with a cup-disk ratio of 0.7 or more and one with a ratio of less than 0.7, produced a significant difference in the intraocular pressure. The large-cup group, consisting of 25 eyes, had a mean intraocular pressure of 14.7 ± 2.5 mm Hg, and the small-cup group had a mean intraocular pressure of 13.3 ± 2.6 mm Hg ($t = 2.89$; $P < .01$).

Age and cup size—Pearson's test disclosed a very low linear covariance ($r = 0.1729$) of increasing cup-disk ratio with increasing age. However, when the patients were grouped by age, the mean cup-disk ratio increased almost steadily from the first decade of life, with a mean ratio of 0.31, to the eighth decade, with a mean cup-disk ratio of 0.44 (Fig. 2). This relationship also appeared when the effects of intraocular pressure were taken into account. When we used the partial correlation to determine the effect of intraocular pressure on the relationship of cup-disk ratios and age, the coefficient was practically identical ($r = .1732$). Subgroups of the population divided by intraocular pressure level also showed a slight linear correlation between age and cup-disk ratio (Fig. 3). A group with intraocular pressures between 4 and 12 mm Hg (439 eyes) and a group with intraocular pressures between 15 and 18 mm Hg (413 eyes) had partial correla-

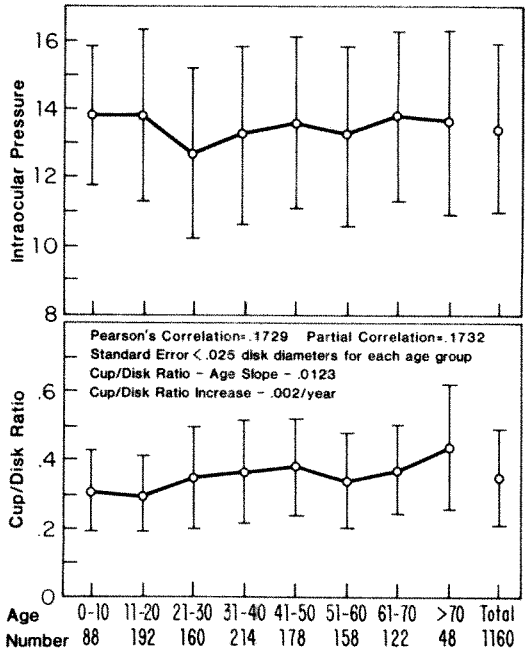


Fig. 2 (Carpel and Engstrom). Top, The means and standard deviations of intraocular pressure for each decade. Bottom, The means and standard deviations of cup-disk ratio for each decade show an almost constant increase in mean cup-disk ratio in older groups. The standard errors for all means are insignificant. Pearson's correlation and the partial correlation, controlling for intraocular pressure, reflect a slight linear covariation and are almost identical.

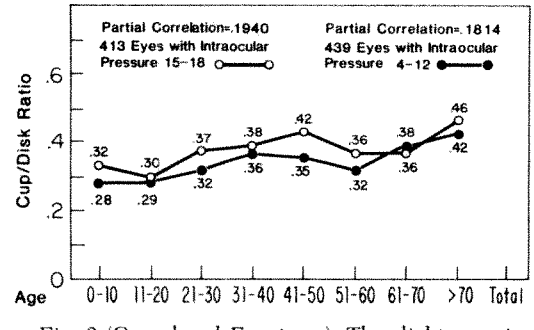


Fig. 3 (Carpel and Engstrom). The slight covariation between cup-disk ratio and age is identical in the eyes with lower (4 to 12 mm Hg) and higher (15 to 18 mm Hg) intraocular pressures. The calculated partial correlation coefficient, controlling for intraocular pressure for each group, reflects this covariation.

tion coefficients of 0.1814 and 0.1940, respectively.

Student's *t*-test disclosed highly significant differences between age groups. Patients less than 20 years old had a cup-disk ratio of 0.30 ± 0.11, whereas the remaining population had a cup-disk ratio of 0.37 ± 0.15 ($t = 7.66$; $P < .001$). Those less than 40 years old had a cup-disk ratio of 0.34 ± 0.14, whereas those older than 40 years had a cup-disk ratio of 0.37 ± 0.14 ($t = 3.82$; $P < .001$). Dividing the patients into two groups, those under 60 years of age and those over 60 years of age, again showed a significant difference (Fig. 4).

Sex and cup-disk ratio—The mean cup-disk ratio of all males, 0.36 ± 0.14, was slightly larger than the mean of 0.34 ± 0.14 for females ($t = 2.56$; $P < 0.02$) (Fig. 4). This difference in male-female cup-disk ratios was also significant in those less than 20 years old ($P < .015$) and in those more than 60 years old ($P < .03$). Females had consistently higher intraocular pressures in all age groups, but the difference was significant only in those less than 20 years of age.

Refractive error and cup-disk ratio—No linear covariance existed between refractive error and cup-disk ratio. However, when patients with moderate myopia were compared to the rest of the population, their cup-disk ratios were significantly larger. Those patients with a myopic refractive error of more than 4 diopters had a cup-disk ratio of 0.39 ± 0.16. The remainder of the population had a mean cup-disk ratio of 0.35 ± 0.14 ($t = 2.72$; $P < .01$) (Fig. 4). For the group aged less than 20 years, the difference in cup-disk ratio was reversed. Those with moderate myopia had a significantly smaller cup (0.20 ± 0.15) than the rest of the group (0.31 ± 0.11) ($t = 3.56$; $P < .001$).

Patients with hyperopia also had a significant difference in their mean cup-disk ratio. Patients with a hyperopic refractive error of 3 or more diopters had a cup-disk ratio of 0.28 ± 12. The rest of the population's mean cup-disk ratio was 0.35 ± 0.14 ($t = 3.68$; $P < .001$). This tendency was consistent in those less than 20 years old and in those more than 40 years old, although the difference for the latter group was not statistically significant.

General relationships—Patients with cup-disk ratios of 0.7 or more had a significantly higher intraocular pressure. These large cupped eyes had a mean intraocular pressure of 14.7 ± 2.5 mm Hg, whereas those with ratios of 0.6 or less had a mean intraocular pressure of 13.3 ± 2.6 mm Hg ($P < .01$). In Figure 2, the intraocular pressure and the cup-disk ratio are graphed for each ten-year age period. For the group aged 20 years or less, the intraocular pressure was 13.8 mm Hg. In the group aged 21 to 30 years, the average intraocular pressure decreased to 12.7 mm Hg. In the following decades, the intraocular pressure steadily increased to the 13.8 mm Hg recorded for the group aged 70 to 80

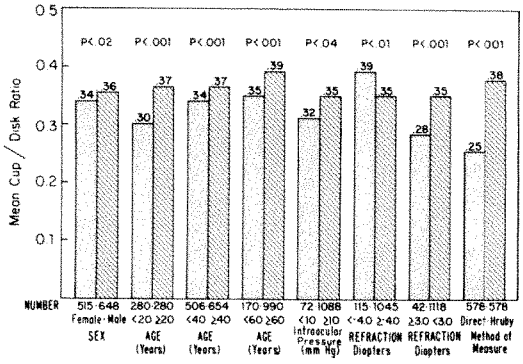


Fig. 4 (Carpel and Engstrom). The difference in mean cup-disk ratio for the factors compared in this study. Although the differences between groups may appear small, they are all statistically significant ($P < .05$). A covariant relationship between groups is not implied.

years. After dividing the population into groups aged 1 to 20 years, 21 to 59 years, and 60 years and over, we used the *t*-test to describe the differences among the groups. The young group (those aged 20 years or younger) had a mean intraocular pressure of 13.2 ± 2.6 mm Hg, whereas the group aged 21 to 59 years had a mean intraocular pressure of 13.8 ± 2.4 mm Hg ($t = 2.57$; $P < .001$). The mean intraocular pressure was significantly higher in those aged 60 years and older compared to those aged 21 to 59 years ($P < .015$). Patients with moderate myopia and hyperopia showed no significant difference in intraocular pressure when compared to the rest of the study population.

Discussion

Our results both confirmed and disagreed with many previously published reports on the cup-disk ratio. A large difference in cup-disk ratios between fellow eyes is rare. Of the 580 people in our study, only 14 had a cup-disk ratio difference of 0.2 or more (Table 3). This result agreed with those of previous studies,[3,16] which found similar cup-disk ratios between fellow eyes. Therefore, one should be suspicious of large differences between eyes of the same patient.

Our statistical analysis disclosed significant differences in cup-disk ratios in certain categories of patients. Eyes with lower intraocular pressures had smaller cups than eyes with higher intraocular pressures. The cup-disk ratio was smaller in younger people than it was in older people. Females had smaller cups than males. Patients with myopia had larger cups than any other group, but young patients with myopia had smaller cups. Previous reports, however, have been divided about these differences.

Figures 2 and 3 illustrate the slight linear covariation between cup-disk ratio and age. Pearson's coefficient and the partial correlation coefficient controlling the intraocular pressure reflected this slight covariation. Because these two coefficients were virtually identical, intraocular pressure appeared to have no effect on the relationship of cup-disk ratio and age. Our failure to observe this effect in our study does not mean that there is no relationship between intraocular pressure and age. Because our criterion for determining normal eyes (intraocular pressure ≤ 18 mm Hg) had a tendency to level the mean intraocular pressure for each decade (Fig. 2, top), there was some correction for intraocular pressure built into the data. The subgroups of eyes with lower (10 to 14 mm Hg) and higher (15 to 18 mm Hg) intraocular pressures showed that cup-disk ratios increased with age in both groups (Fig. 3). The partial correlation coefficients of 0.1814 and 0.1940, respectively, reflected these slight covariations.

Our results thus agreed with those of Pickard[4] who concluded that the physiologic cup increased with age. He repeatedly examined the same patients over a 15-year period, drew pictures of the cup, and used a grid to calculate the area of cupping. He concluded that there was a gradual increase in cupping as the patients aged. However, he also found that a large percentage of cups did not increase in size. The cups that did grow larger did

TABLE 3

Hruby lens cup-disk ratio difference between fellow eyes

	Patients	
Difference	No.	%
None	365	62.8
0.1	201	34.7
0.2	11	1.9
0.3	2	0.4
0.4	1	0.2

so sporadically, without any linear relationship. Because our study did not follow the same population over a period of time as Pickard's did, our results only imply that the cup increases with age.

Several other studies also indicated that cup-disk ratios increase with age,[5,14,16,18] but some investigators have found otherwise.[1-3] Snydacker[1] and Armaly[3] used the chi-square test after modifying the cup-disk ratio data into various ordinal categories. Armaly[3] used the chi-square test because his data were in a nongaussian distribution. Student's *t*-test, an appropriate test for ratio data, was used in our study as there was little kertosis or skew in the frequency curve of Hruby lens cup-disk ratio. Also, the sample size of 1,160 eyes made the *t*-test reliable despite the small deviation from a gaussian distribution.

Armaly[3] found no significant difference between the sexes with the chi-square test. Snydacker[1] stated that the distribution of cup-disk ratios was essentially the same in each sex. But Snydacker's distribution table showed a slight shift to larger cups in males. Ford and Sarwar[5] found physiologic cupping of more than 20% of the optic disk area more common in males than in females. One group also found larger cups (color cups) in white males.[12] Our study showed a larger cup-disk ratio in males. Even though this difference was small, the number of patients made it statistically significant. The difference was consistent in all age groups.

There have also been conflicting reports regarding the relationship of refractive error to cup-disk ratios. Snydacker,[1] using the chi-square test, found no significant variation in frequencies in the cross-tabulation of refractive error and cup-disk ratio, nor did Hollows and McGuinnes[14] using retinoscopic values. Two other studies found that patients with myopia had large cups, whereas those with hypermetropia had small cups.[5,18] Tomlinson and Phillips[15] correlated area cup-disk ratio, but not diameter cup-disk ratio, with refractive error. One study using the biomicroscope found no association of cup-disk ratio with refractive error in midrange.[16] Our study also indicated that overall, patients with moderate myopia (> 4 diopters) had significantly larger cups and those with moderate hyperopia (> 3 diopters) had significantly smaller cups. Young (≤ 20 years old) patients with moderate myopia, however, had significantly smaller cups than the remaining population. This group included only 14 eyes, but this is a large enough group to be significant ($P < .001$). Possibly accommodative control was incomplete, yielding greater myopic values in this group. This tendency toward smaller cups in young patients with moderate myopia needs further investigation. The tendency of patients with moderate hyperopia to have smaller cups was consistent in all age groups, including that aged 20 years and younger. This agrees with commonly held clinical impressions. It must be noted, however, that there was no covariance of cup-disk ratio and refractive error; that is, no increase in cup-disk ratio occurs on a linear scale from hyperopia to myopia.

We also explored the effects of intraocular pressure on cup-disk ratio. The analysis was slightly skewed by eliminating eyes with intraocular pressures of more than 18 mm Hg. Narrowing the range of normal intraocular pressure would most likely narrow the difference between groups, decreasing the significance.

The relationship between intraocular pressure and normal cups has been examined in previous studies, with conflicting results. Hollows and McGuinnes[14] found a tendency toward increased cupping with increasing intraocular pressure, but no statistically significant increase (chi-

square test) in cup size in the groups with higher intraocular pressures. Armaly and Sayegh[2] found that cup-disk ratios were significantly related to applanation pressure, in that the frequency of cup-disk ratios of 0.3 or more increased significantly with increasing applanation pressures. They did not suggest that a linear relationship exists. In our study, the group with very low intraocular pressure (< 10 mm Hg) had significantly smaller cups, and the group with higher intraocular pressures had significantly larger cups. Therefore, there is a relationship between intraocular pressure and cup-disk ratios but this relationship is not linear as expressed by Pearson's test. Because we chose an applanation pressure of 18 mm Hg as the cut-off point, our results do not definitively prove that there is no linear relationship.

Other observations provided clinically useful information. Approximately 9% of the time we found an ovoid cup, but in 90% of these cases the horizontal cup-disk ratio exceeded the vertical ratio. In only one case of vertical ovoid cupping was the difference more than 0.1. Therefore, ovoid cups with vertical diameters larger than horizontal ones are rare in normal eyes. This supports the observation of Kirsch and Anderson[21] that vertically ovoid cups may indicate glaucoma.

Selecting an applanation pressure of 18 mm Hg or less as normal is somewhat arbitrary. Only 19 people in the study had intraocular pressures as high as 18 mm Hg, and the likelihood of one of them having glaucoma was small and should not have affected the data in a sample of this size. Because visual fields were not performed, we relied on intraocular pressure and medical history as screening tools. Even if normal visual fields had been obtained, there would have been no guarantee that early glaucomatous cupping was not present.[23]

There were other subjective factors and possible errors of bias in this study. Previous studies have shown that errors are made in estimating the cup-disk ratio.[4,9,18,19,24,25] One study illustrated the inherent difficulty of diagnostic standardization by having five different examiners determine the horizontal cup-disk ratio by two different methods.[24] There was a wide variation in their findings. However, one protocol used the direct ophthalmoscope with its monocular limitations, and the second used the indirect ophthalmoscope with its magnification limitations. Although the second protocol used contour as the basis for the cup, both protocols chose as the margin of the cup a point midway in a slope or transition zone. In our study, a single observer, using strict definition of the cup with the slit beam and a magnified view of the optic disk with a Hruby lens, should have eliminated some of the major difficulties in cup-disk analysis.

Another study reported the majority of errors to be underestimates, 95% of the magnitude of 0.1, with none thought to be greater than 0.2.[19] Sommer, Pollack, and Maumenee[9] aptly pointed out that comparative stereoscopic photography is the best method of observing changes in an optic disk because sketching does not allow perfect reproducibility but does create a variability of up to 0.2 disk diameters. Of their 40 randomly reread stereoscopic slides, six were within 0.1 to 0.2 of the previous estimate of horizontal cup-disk ratio, and one was within 0.2 to 0.3. Thus, subjective judgment can be a source of error even when objective methods are used. Unless serial stereoscopic photographs are taken in an identical manner, and are superimposed and magnified during analysis, one can not eliminate observer error. At present this is not a common clinical practice. Although we did not redetermine a large number of cup-disk ratios for statistical analysis, 38 eyes randomly reevaluated in

a masked fashion by the same observer (E. F. C.) resulted in estimated cup-disk ratios that were identical 66% of the time and ± 0.1 in the remainder. Again, with a magnified stereoscopic image of the optic disk and a slit beam to assess the contour of the cup, large errors seem unlikely to occur.

Schwartz[25] pointed out that large interobserver differences exist in published studies of normal cup-disk ratios. He believed, therefore, that one should be wary of attaching clinical importance to specific values of cup-disk ratios reported as normal. Because we used one experienced examiner to perform all evaluations, any inaccuracies in estimating the cup-disk ratio should have been consistent. Possibly there were consistent overestimations or underestimations that affected the mean cup-disk ratio evaluation. However, the relationships between methods (direct ophthalmoscope vs Hruby lens), age, intraocular pressure, and sex should not have been greatly affected by these possible subjective errors.

Schwartz[8] emphasized that the size of the optic cup was generally grossly underestimated with a direct ophthalmoscope and that the cup appeared larger when observed with a Hruby lens, but he did not state the extent of this difference. In 447 of the 580 eyes, the slit lamp with Hruby lens gave a larger (≥ 0.1) estimated cup-disk ratio than the direct ophthalmoscope did. In only one instance was there a larger estimate with the direct ophthalmoscope than with the Hruby lens. With a Hruby lens, 1,135 of the 1,160 eyes had cup-disk ratios of 0.6 or less; with a direct ophthalmoscope 100% had cup-disk ratios of 0.6 or less. This supports the previous studies that suggested any cup-disk ratio of more than 0.66 should be viewed with suspicion.[1,4,10-12]

Our data confirmed the difference in the two methods and clearly documented that in normal eyes the cup-disk ratio is, on the average, underestimated by 0.13 with a direct ophthalmoscope. This was expected, because a slit lamp with Hruby lens or biomicroscopic lens provides a highly magnified stereoscopic view of the first change in contour of the nerve fibers, highlighted by bends in the slit lamp beam, whereas the direct ophthalmoscope depends largely on color in the absence of vessel deflection or obviously sharply demarcated cup edges, and could miss the first posterior deflection of nerve tissue at the surface of the optic disk. This difference is probably the difference between the true cup and the color cup.

Until stereoscopic photographs are routinely used, one should examine the optic disk at each office visit with a Hruby or biomicroscopic lens. It is useful to know the expected norms for Hruby or biomicroscopic lens evaluation. Because the average estimate of the cup-disk ratio with the Hruby lens is 0.13 more than it is with a direct ophthalmoscope, and because we found no more than a 0.2 disparity in 87% of the cases, one should not necessarily regard an apparent increase of 0.1 as a change when changing from the direct ophthalmoscope to the Hruby lens. However, any difference of more than 0.2 should be viewed with suspicion, and any patient with a cup-disk ratio of 0.7 or more should be considered at risk for glaucoma.

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RELATIVE BLUE-LIGHT EXTINCTION IN LENS NUCLEUS RELATED TO AGE AND CATARACT

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The extent of visual function loss from nuclear cataractous change can be estimated by observing the extinction of blue light compared to white light as a function of saggital lens depth in standard slit lamps. Apparent extinction (loss of transmissivity) of 50% or more of the depth of the lens in blue light of maximal intensity compared to white light at minimal intensity is associated with marked visual loss. However, this transmissivity ratio of 0.5 or less also applies if light intensity remains constant and the blue filter is simply moved in and out of the light beam. Therefore, if 50% or more of the lens appears dark to blue-filtered light, nuclear changes have significantly decreased effective visual acuity. Relative blue-light extinction associated with nuclear change that may be amenable to surgical correction can thus be distinguished from the extinction effect caused by age alone.

Blue-filtered light, unlike unfiltered white light, disappears in the depths of the slit-lamp of the lens in proportion to aging. If the anterior lens capsule is brought into focus, the whole optical section of the nucleus of a young lens appears uniformly visible in both unfiltered white light and blue-filtered light. In an older lens, however, and particularly in one with nuclear cataract, the posterior aspect of the optical section becomes invisible in blue light, although it remains clearly delineated in white light. The effect is best seen by rapid movement of the standard blue filter of any modern slit lamp in and out of the illumination path while fixation remains stable.

This blue-light extinction phenomenon is correlated clinically with "nuclear sclerosis," which is often consistent with retention of satisfactory visual acuity by conventional measurement. However, as the process advances, the level at which blue light disappears in the optical section gradually moves forward, in association with the classical signs of true nuclear cataract formation. As the rubescent nucleus darkens to brunescence, the level at which blue light is extinguished approaches the cortex, whereas the optical section of the nucleus in white light, although attenuated and altered in hue, remains visible to its posterior limit. The cortex is uninvolved in this process. This relative extinction of blue light has not been previously described as a clinical sign, although the physiochemical basis for it has been studied.

Loss of visual function from moderately

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advanced nuclear sclerosis may be difficult to distinguish from the frequently associated senile maculopathy. This study was undertaken to correlate blue-light extinction with nuclear cataract causing loss of visual acuity and to determine whether blue-light extinction in this condition can be separated from that caused by age alone.

SUBJECTS AND METHODS

Subjects—We examined a total of 173 eyes in 103 patients ranging in age from 18 to 97 years; all were free of corneal disease. The pupils were dilated with phenylephrine and tropicamide. We recorded fully corrected distance visual acuity, lenticular nuclear sclerosis, and nuclear or brunescent cataract. We excluded lenses showing cortical or subcapsular cataractous changes, unless such changes were confined to the far periphery of the lens. We also excluded eyes showing discernible or suspected central retinopathy. Three examiners graded the extent of nuclear sclerosis with white-light illumination on a scale of 1 to 4.

Instruments—We used a single-standard Zeiss slit lamp (116 SMM). A beam splitter (50%) interrupted one ocular, reflecting into a low-light-level television camera, the images from which were simultaneously displayed on a monitor and recorded. An audio-recorder, operating simultaneously, was used to identify patient, eye, and observational factors. The observer, who was using a full correction, focused a uniform slit beam, at a constant 40-degree angle from the axis of observation, on the anterior lens capsule. The visual axis and the axis of observation were kept parallel. Recordings were made in rapid succession with white light at the lowest rheostat setting and blue-filtered light at the maximum setting. Radiant flux density for these conditions, measured with an Epply thermopile, was greater for white light by a factor of 3.8. Slit-lamp magnification, camera settings, and fixation were maintained throughout the recordings. The cassette recording was subsequently replayed, stopped in the pause mode, and the intensity profile of the lens measured by a video-integrator, the signal from which actuated an X-Y recorder. We set the horizontal sweeps of the video-integrator across the lens image vertically to avoid "bloom" artifacts, but they were close to the central axis in all cases, and identical for both blue- and white-light conditions. Images in the two conditions were made comparable by standardization of sweep rate of the video-integrator with a function generator. The X-Y recorder thus graphed intensity as a function of semisaggital (40-degree) distance from the anterior lens capsule. Figure 1 shows typical recordings from a clear lens (left) and a lens with a nuclear cataract (right).

Measurements—We used a point on each X-Y tracing corresponding to the light intensity of the vitreous chamber as the baseline. The anterior capsule of the lens was identified as the beginning of the transient from the optically empty aqueous chamber (Fig. 1). We measured the distance (D for blue light and D′ for white light) across the tracing at which the Y value (relative intensity) decreased to 10% of its peak value (y/10 and y′/10). Apparent blue-light transmissivity compared to that of white light was determined by the fraction D (blue y/10)/D′ (white y′/10), or D/D′, and expressed as percentage of relative transmissivity. This latter is the inverse of relative extinction and is used to express results because it is the function actually measured. (Relative transmissivity is a convenient term describing the intensity of the lens image as a function of saggital depth as viewed in the slit lamp. It represents a complex inversely related to the several factors involved in extinction, including

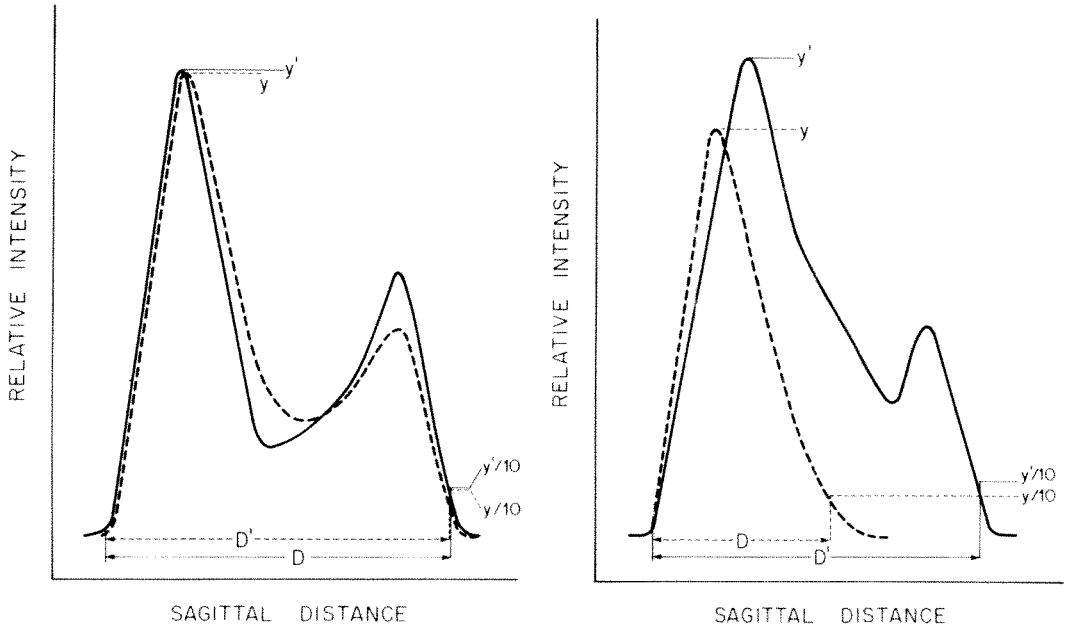


Fig. 1 (Krohn and associates). X-Y recordings of video-integrator intensity detection as a function of distance through 40-degree semisaggital optical cross-sections of two representational human lenses. Solid line, white light at lowest intensity setting; dashed line, blue light at highest intensity setting; y, peak intensity for blue light; y', peak intensity for white light; D, distance from anterior capsule to y/10; D', distance from anterior capsule to y'/10. Left, Clear lens of a 25-year-old individual with a corrected visual acuity of 6/6 (20/20). Right, 3+ nuclear sclerotic lens of a 63-year-old individual with a corrected visual acuity of 6/30 (20/100).

scatter and absorption, in particular, and fluorescence, phosphorescence, and heat production, for both entrance and exit beams as well.)

Results

With normal visual acuity, defined as 6/12 (20/40) or better, lens transmissivity for blue light compared to white light at these relative intensities remains constant to the age of about 50 years, after which data points significantly decrease and scatter with increasing age. The blue/white (D/D') ratio remains above 0.7 at all ages, however, in the presence of normal central visual acuity (Fig. 2).

Relative blue-light transmissivity decreases as a function of clinically graded nuclear sclerosis (Fig. 3). For lenses showing sclerosis of 3+ or more, the transmissivity ratio is consistently less than 0.7.

There is a similar relationship between relative blue-light transmissivity and decline in central acuity, presumably caused by nuclear lens change alone (Fig. 4). Transmissivity ratios of less than 0.7 appear to be associated with best corrected visual acuities of 6/24 (20/80) or less. Some overlap, however, is present between visual acuities of 6/12 (20/40) in normal lenses and 6/22 (20/70) in lenses showing significant nuclear opacification.

Discussion

We developed a method used to calculate relative extinction of blue light compared to white light in order to quantitate the observed clinical phenomenon. The point of extinction was designated as 10% of the peak apparent transmission for both cases because this is the point at which the light appears to fade completely during a clinical examination. This

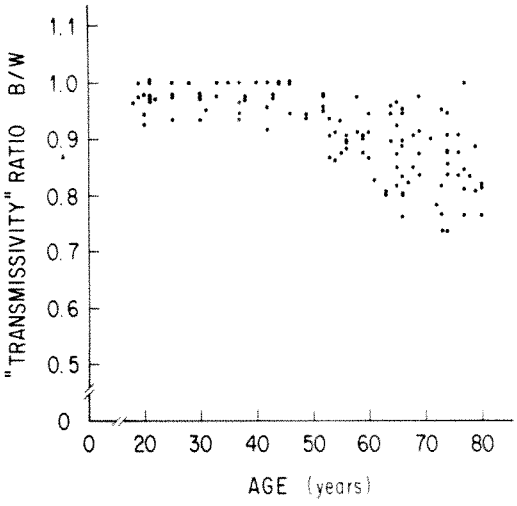


Fig. 2 (Krohn and associates). Plot of transmissivity ratio B/W (D/D′) as a function of age for eyes with corrected distance visual acuities of 6/12 (20/40) or better.

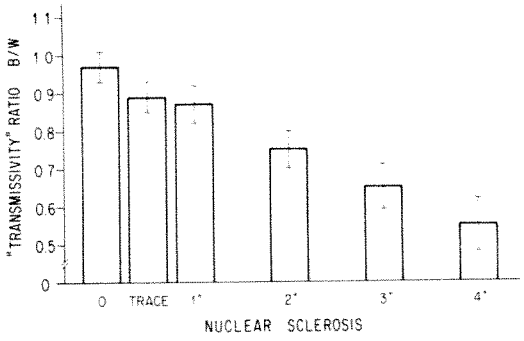


Fig. 3 (Krohn and associates). Mean transmissivity ratios B/W (D/D) ± S.D. related to clinically graded nuclear sclerosis. Estimate of relative nuclear change was made after three observers concurred.

eliminates the ambiguity of zero transmission, and normalizes the variation in peak intensities from one eye to another for both conditions.

Our findings indicated that extinction of 50% or more of the 40-degree semisaggital depth of the lens in blue light at maximal intensity, compared to white light at minimal intensity (transmissivity ratio of 0.5 or less), is associated with marked visual loss caused by nuclear sclerotic cataract. Therefore, if 50% or more of the lens appears dark to blue-filtered light, nuclear changes alone are responsible for significantly (and potentially correctable by surgery) decreased effective visual function. In the clinical situation, this estimate appears to be useful, although less precise, even if the light intensity remains constant and evaluation is made by simply moving the blue filter in and out of the slit-lamp beam. In this case, darkness of the posterior half of the lens in blue light with constant illumination would be even more significant for visual deterioration from nuclear change.

Relative extinction of blue light has not been previously described as a clinical sign, although the physiochemical basis for it has been extensively studied. These observations suggested that both scattering and absorption, the major components of extinction in this spectral range, are related to formation of high-molecular-weight protein aggregates, probably the derivative insoluble protein, and closely associated fluorogen accumulation.[1-8] Scattering may be related mainly to the change in molecular size.[9-12] In vivo measurements of lenticular backscattering as a function of age and nuclear cataract formation have shown a pattern quite similar to that for relative blue-light extinction found in this study.[10]

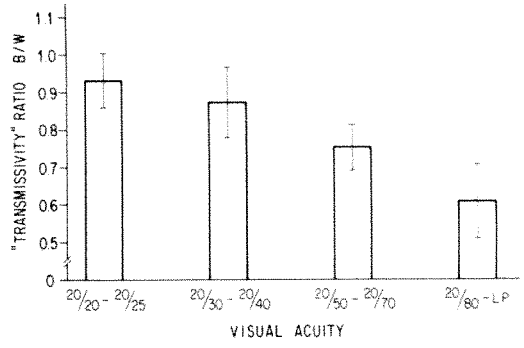


Fig. 4 (Krohn and associates). Mean transmissivity ratios B/W (D/D′) ± S.D., related to best corrected distance visual acuity in groups as shown.

The absorption component of the extinction phenomenon appears to be related to various fluorescent pigments linked to the insoluble protein and perhaps serving as cross-linking agents.[2,3,6,11] The nontryptophan fluorogens that have been most intensely studied are excited at 330 nm and fluoresce at 440 nm, a spectral range almost 100 nm shorter than that involved in our study. The blue filters of standard slit lamps (Wrattan 47 B or an equivalent, selected specifically for fluorescein excitation) transmit a broad band peaking at 430 nm. The fluorescent compounds responsible for relative blue-light absorption in slit-lamp illumination are thus still uncertain, although it appears likely that they are various oxidative derivatives of tryptophan.[9,10]

The relative contribution to the observed blue-light extinction of absorption and scatter as related to age is similarly in doubt.[9] The relative linearity in youthful lenses compared to the increased spread in older normal lenses as relative blue-light extinction increases suggested that several variables were involved (Fig. 2). Although this finding was consistent with the reported increasing spread in electrophoretic patterns of nuclear proteins in the same age group,[9] it also correlated with the increasing accumulation of various fluorogens found by many investigators.

Two further studies confirming the usefulness of this clinical test and enhancing its precision are in progress. In this study, we used maximal-intensity blue light and minimal-intensity white light. Measurements indicated that the total energy of the white light was almost four times that of the blue light. The first of these new studies has shown that this ratio can be reversed with sufficient illumination and appropriate neutral density filtration, confirming that the extinction phenomenon was not confused with an apparent extinction caused by relative intensity relationships. Preliminary results indicated that extinction remains a linear function of age in normal lenses despite the reversal of the relative intensity ratio. In the second study, we are comparing the presumptive loss of visual acuity from nuclear cataract, based on blue-light extinction determined before cataract extraction, to the difference between preoperative and postoperative best corrected visual acuities in a group of patients with no other abnormalities.

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INTRAOCULAR ENDOSCOPIC SURGERY

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We have successfully used a new small endoscope in posterior segment surgery in situations where the operating microscope or binocular indirect ophthalmoscope provided an inadequate view. In this series of 18 procedures, we performed posterior vitrectomies, removed retained foreign bodies, and reattached persistent retinal detachments. The only major complication was a retinal detachment. Learning to use a monocular system requires practice and preparation with experimental animals.

Pars plana endoscopy, using a new generation of small endoscopes,[1-3] has proven useful in cases where opacity in the anterior segment threatens to compromise or delay needed vitreoretinal surgery. This technique allows the surgeon to bypass the opacity, to observe details, and to operate in the posterior eye.[4] We used pars plana endoscopy to guide our procedures during observation of posterior segment abnormalities, removal of retained intraocular foreign bodies, and posterior vitrectomy.

MATERIAL AND METHODS

The endoscope has an intraocular shaft, 1.7 mm in diameter, containing a viewing rod lens and illuminating fibers (Fig. 1). The field view is 75 degrees, an area adequate for surgery. As the endoscope is brought closer to vitreoretinal structures and the total area under view becomes smaller, the structures are magnified; the magnification is limited to about ×10. Light is supplied by a fiberoptic light box with an adapter on the light cord to adjust to the endoscope fitting.

Because the endoscope must be completely stable within the eye, it is mounted on a stereotaxic positioner, modified from previously described systems,[5,6] which allows scanning of the retina (Fig. 2). The base plate of the positioner holding the endoscope also supports the patient's head when the plate is clamped to the end of the operating table.

All operations were performed with the patients under general anesthesia. We used curare to prevent any inadvertent head movements during surgery. All patients had undergone appropriate testing preoperatively, including ophthalmic ultrasonography and computed axial tomographic scans where indicated.

The first step is selecting the sclerotomy site for the endoscope. This is usually through the pars plana at the 12 o'clock position, which enables the surgeon to sit at the head of the patient in a position already familiar from binocular microscopic surgery. However, since the best view for manipulating instruments is obtained from a position opposite the abnormality, a foreign body at the ora serrata at the 4 o'clock position would be

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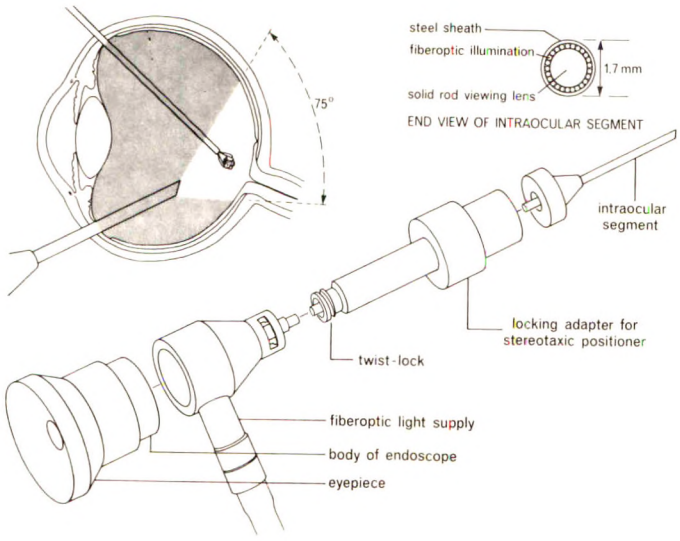


Fig. 1 (Norris and associates). Schematic side and end views of endoscope, light supply, and adapter for mounting in positioner. Schema at top left shows removal of intraocular foreign body.

best managed by inserting the endoscope at the 10 o'clock position. The space needed by the surgeon's hand and the operating instruments must also be considered. A second entry site for vitreous instruments closer than two clock hours does not allow sufficient space for manipulation. The instruments selected for the surgery, such as a foreign-body forceps or vitreous cutters, are maneuvered separately from the endoscope. We often used a separate infusion port to maintain the intraocular pressure.

We used pars plana endoscopy in 16 eyes of 15 patients. (These patients have been described in an earlier article.[4]) Two patients underwent a second endoscopy, for a total of 18 procedures. Of the 18, 13 involved posterior vitrectomies, seven were for vitreous hemorrhages, and six for complicated retinal detachments or retained intraocular foreign bodies. The five remaining procedures included three for evaluation of the posterior segment in eyes being considered for anterior segment reconstruction, one for endophthalmitis, and one to locate a retinal break in a detached retina behind a dense cataract the patient refused to have removed.

The following case reports are representative of the types of problems in this first series in which pars plana endoscopy was used.

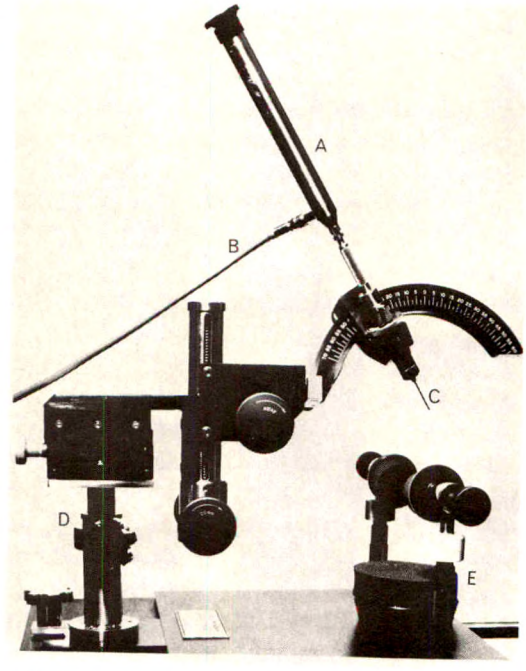


Fig. 2 (Norris and associates). Endoscope and stereotaxic positioner. A, endoscope; B, light supply cord; C, intraocular endoscope shaft; D, endoscope positioning controls separate from baseplate; E, head support and fixation.

CASE REPORTS

Case 3—A 56-year-old man with chronic uveitis and bilateral aphakia had had a visual acuity of light perception in his left eye for many years after the development of an occluded pupil which occurred after cataract extraction. The eye had also been treated for increased intraocular pressure. When vision began to fail in the right eye because of cystoid macular edema, retinal hemorrhages, and pupillary membrane formation, the patient requested an examination of his left eye.

Entoptic phenomena could not be elicited. There was no color perception. An electroretinogram showed a reduced response. Ultrasonographic tests demonstrated no evidence of retinal detachment, but showed vitreous debris and suggested the presence of a large optic nerve head cup.

We used pars plana endoscopy to evaluate the posterior segment before considering anterior chamber reconstruction in the left eye. The endoscopy showed cystoid macular edema, an enlarged optic nerve head cup, with a good rim of tissue, and an attached retina. We removed the endoscope and closed the sclerotomy. We opened the pupillary membrane and removed the vitreous debris with a vitreous stripper, using a surgical microscope. The left eye attained a visual acuity of 6/60 (20/200) and subsequently became the better eye.

Case 13—A 65-year-old woman, blind in her left eye from unknown causes, had had two unsuccessful retinal reattachment operations on the right eye. We suspected that a retinal break in an area of chorioretinal scarring with fixed folds inferotemporally was the cause of the persistent detachment, but could not see it. We performed a pars plana endoscopic examination at the time of a third attempt at reattachment. The endoscope was directed toward the fixed folds and chorioretinal scars. Indentation of the sclera over the suspect site pressed subretinal fluid containing pigment through the break and disclosed its location. We placed a scleral buckle over the defect and reattached the retina.

Case 15—A 16-year-old boy had sustained a penetrating wound of the right eye while hammering cement, which resulted in a large corneal laceration, iris prolapse, cataract, and vitreous hemorrhage. After repair of the corneal laceration and removal of the cataract, the view of the posterior segment through the surgical microscope was inadequate. Using the endoscope through sclerotomy at the 12 o'clock position, we located a metallic foreign body and removed it with foreign-body forceps. We also removed several small gray fragments that appeared to be cement with a vitrectomy instrument. The postoperative course was satisfactory until an unrelated episode of severe blunt trauma resulted in the loss of the eye.

RESULTS

Case 3 showed that endoscopically obtained information about the fundus can be useful in determining whether anterior segment reconstruction should be done. In Case 13, in which the patient had undergone two unsuccessful retinal reattachment procedures and several observers had failed to find the retinal defect, endoscopy was the only way of examining the area behind the retinal folds and vitreous scar tissue. Pressing subretinal fluid through the break conclusively proved the defect's existence and location. Endoscopy was the only means of observing the small flecks of pigment in the subretinal fluid, confirming its movement. In Case 15 the endoscope not only guided the removal of the retained metallic foreign body but also disclosed several small bits of cement in the bloody vitreous that would not have been noticed otherwise. This information demonstrated the need for a thorough removal of vitreous in order to minimize postoperative reaction.

The only complication in this series was a retinal detachment that appeared to be the result of the sclerotomy. One patient attained a visual acuity of 6/6 (20/20). In the others, cystoid macular edema, preretinal fibrosis, the sequelae of trauma, or anterior segment disease led to impaired visual acuities.

DISCUSSION

Since Thorpe[7] published a report concerning the extraction of intraocular foreign bodies by endoscopic techniques, smaller endoscopes more suited to surgery within the eye have been developed. One of us (J. L. N.) has adapted an endoscope to ophthalmic surgery.[3,4,6,8] The size of the endoscope is comparable to other instruments now used for pars plana vitreous surgery.

To date, pars plana endoscopy has been useful in two kinds of cases: (1) when additional information regarding the status of the retina and optic nerve is needed before deciding whether or not to perform anterior segment reconstruction

and (2) when anterior segment opacity obscures the view, making vitreoretinal surgery difficult. Standard binocular optical systems should be employed whenever possible and endoscopy used only when other methods are inadequate. Endoscopic procedures are more difficult because they are monocular and a period of training with animal surgery is necessary to transfer binocularly learned vitreoretinal surgery skills to endoscopy. The creation of one retinal detachment attributable to endoscopic technique in 18 procedures (6%) seems acceptable in view of the degree of difficulty and the complicated nature of these cases. The major difficulties we have experienced with endoscopic surgery have been the increase in operating time required and our limited experience with the procedure.

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COATS' DISEASE IN A PATIENT WITH CORNELIA DE LANGE SYNDROME

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A 16-month-old boy had the mental and physical retardation, low-pitched cry, phocomelia with syndactyly, hirsutism, low-set ears, bushy eyebrows, elongated eyelashes, blepharoptosis, and strabismus characteristic of Cornelia de Lange syndrome along with ophthalmoscopic findings characteristic of Coats' disease.

Cryotherapy made the abnormal telangiectatic vessels less prominent, but macular scarring produced poor visual acuity. Occlusion therapy was unsuccessful.

Cornelia de Lange syndrome is characterized by mental and growth retardation, multiple skeletal abnormalities, a feeble low-pitched cry, low-set ears, and hypertrophic eyebrows and eyelashes.[1] Ocular abnormalities have been associated with this syndrome.[1-7] We studied a patient with Cornelia de Lange syndrome and Coats' disease. To the best of our knowledge, this association has not been recorded previously.

CASE REPORT

We examined a 16-month-old boy in September 1977. The patient's mother observed that the child's upper eyelid was "droopy" and that the left pupil occasionally appeared bright yellow. The medical history included an uncomplicated pregnancy and delivery without illness or drug use. Maternal alcohol use had been limited to less than 8 oz of wine per week. The child's birthweight had been 2,097 g and supplemental oxygen had not been required. A diagnosis of Cornelia de Lange syndrome had been established elsewhere.

Physical examination disclosed mental and growth retardation, partial phocomelia with syndactyly (Fig. 1), low-set ears, hirsutism (Fig. 2), and a feeble low-pitched cry. Ocular examination disclosed that fixation was central and well maintained in the right eye but eccentric and unsteady in the left eye. The eyebrows were bushy, meeting in the middle of the forehead, and the eyelashes were elongated. The left upper eyelid had 4 mm of blepharoptosis with poor (3 to 4 mm) levator palpebrae superioris muscle function. The left pupil briskly reacted to consensual stimulation and was not obscured by the upper eyelid. Ocular rotations were normal, but there was a 15-diopter esotropia by the Krimsky method. Retinoscopy disclosed a −1.00 refractive error in both eyes. Ophthalmoscopic examination of the right eye showed nothing unusual, but examination of the left eye showed many telangiectasias and small aneurysms of the retinal vessels along the inferior temporal arcade just posterior to the equator. Yellow exudation was present in the macula and the inferior temporal quadrant (Fig. 3). The ophthalmoscopic findings were typical of early Coats' disease. The diagnoses of sickle-cell retinopathy, retinoblastoma, angiomatosis retinae, and retrolental fibroplasia were considered but excluded on the basis of the fundus findings, the noncontributory history, and the laboratory studies. Results of karyotyping were normal (Fig. 4).

In November 1977, we examined the patient while he was anesthetized. The diagnosis of Coats' disease was confirmed, and cryotherapy was applied over the telangiectatic vessels.

Reexamination of the fundus in April 1978 showed that the abnormal telangiectatic vessels were less prominent and were surrounded by cryotherapy scars. The exudates had decreased, but still involved the fovea. There were no new lesions.

We began cautious occlusion of the normal right eye in June 1978. Because there was a marked regression in the child's behavior during occlusion, however, we postponed amblyopia therapy until the macular exudates had resolved further. In January

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Fig. 1 (Folk, Genovese, and Biglan). Syndactyly and phocomelia of the arm.

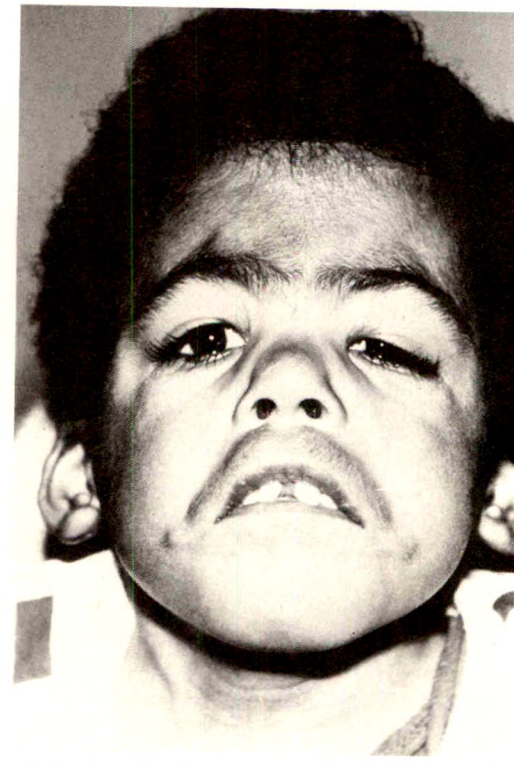


Fig. 2 (Folk, Genovese, and Biglan). The face shows low-set ears, blepharoptosis, elongated eyelashes, hirsutism, and bushy eyebrows meeting in the midline.

1979, we again tried occlusion. After five months of almost full-time occlusion, the fixation reflex of the left eye had not improved and we discontinued amblyopia treatment.

Examination in May 1980 showed that the fixation reflex of the right eye was central and steady, but the fixation in the left eye remained eccentric and wandering. The left eye remained esotropic and moderate blepharoptosis of the left upper eyelid persisted. Ophthalmoscopic examination of the right eye gave normal results. The left fundus showed a total resolution of the exudates, absence of telangiectatic vessels, and a residual flat macular scar.

Discussion

The external and anterior segment ocular features of the Cornelia de Lange syndrome include long bushy eyebrows meeting in the midline, long eyelashes, telecanthus, blepharoptosis, blue sclera, and microcornea.

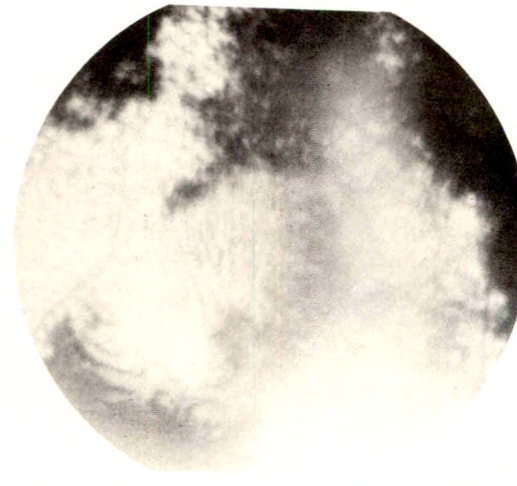


Fig. 3 (Folk, Genovese, and Biglan). The exudate present in the left eye before treatment of the telangiectatic vessels.



Fig. 4 (Folk, Genovese, and Biglan). The patient's karyotype, normal for a male infant.

Ophthalmoscopic abnormalities associated with this syndrome are infrequently reported. Richter[2] described two patients with pale optic disks, one of whom also had temporal hemorrhages in the right eye and a coloboma of the optic nerve in the left eye.

Ptacek and associates[4] reported nine cases of Cornelia de Lange syndrome. Four patients had severe myopia, two had optic nerve atrophy, and one patient had "extremely large veins on each nerve head." Milot and Demay[5] reported nine cases of Cornelia de Lange syndrome. Ophthalmoscopic examination of their second patient showed pallor of the optic disk and narrowed arterioles. The right eye of their Patient 3 had a large inferotemporal retinoschisis. Lim and Wong[6] reported a unilateral retinoblastoma in a Chinese girl with this syndrome.

Evens, Vinken, and Fryns[7] reported findings similar to those in our patient. One of their patients had severe myopia and "circumferential chorioretinitis" involving the right macula. The second patient in their report initially had microcorneas, deep anterior chambers, normal lenses, normal fundi, and bilateral severe myopia. Three years later, however, both eyes were hypotonic with atrophy of the iris, complete cataract, and a greenish hue. A similar brown or green coloration, caused by extensive subretinal hemorrhage and exudation, frequently appears in patients with end-stage Coats' disease. Coats' disease could have accounted for the rapid deterioration of this patient's eyes and the subsequent green discoloration.

Systemic features of Cornelia de Lange syndrome in our patient were the mental

and physical retardation, the low-pitched cry, phocomelia with syndactyly, the hirsutism, and the low-set ears. Ocular findings typical for this syndrome were the bushy eyebrows, the elongated eyelashes, blepharoptosis, and strabismus. Additionally, our patient had features in the fundus characteristic of Coats' disease.[8,9] Telangiectasis and aneurysm were present along the inferotemporal arcade of the left eye, causing massive exudation in the posterior pole. Although the vascular lesions responded promptly to cryotherapy and did not recur, the exudates took more than one year to resolve and left a macular scar which decreased visual acuity.

Although the occurrence of Cornelia de Lange syndrome and Coats' disease in our patient may have been purely coincidental, the infrequency of these conditions in the general population and the case reported by Evens, Vinken, and Fryns[7] suggests an association between these syndromes. This association should be considered when examining patients with either of these entities.

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THE OPTICS OF VITREOUS SURGERY

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We calculated the refractive power of the human eye in the various optical configurations that may occur during vitreous surgery. We found that a biconcave corneal contact lens with an anterior surface refractive power of −63 to −98 diopters allowed the fundus to be seen when the vitreous cavity was filled with air, as did a weak minus lens attached to the bottom of the operating microscope. Our tests showed that the unsymmetrical biconcave lenses were superior.

In modern vitreous surgery, the removal of the lens is common, as is changing the optical power by putting a flat-faced contact lens on the anterior surface of the cornea or by filling the vitreous cavity with gas. Some of these procedures have a profound effect on the total refractive power of the eye, and, therefore, affect the ability of the surgeon to view the operating field. This problem is particularly acute when air is introduced into the eye during surgery near the posterior retina. Most vitreous surgeons are reluctant to fill the phakic eye with air during vitrectomy surgery because of the poor view of the retina which this produces. An optical system that provided a clear view of the fundus under these conditions would be extremely helpful. It would allow surgeons to operate within an air-filled vitreous cavity in phakic eyes.

We have calculated the refractive power of the human eye in the various optical configurations that may occur during vitreous surgery. On the basis of these calculations, we propose that various types of corneal contact lenses be used in conjunction with an operating microscope to aid visualization of the posterior pole, particularly in the phakic eye where vitreous has been replaced with air.

MATERIAL AND METHODS

We are concerned only with some of the practical optical problems that face the vitreous surgeon. Only a superficial discussion of the theory of optics is needed. For a more rigorous explanation the reader can refer to standard texts on optics.

The refractive power of a spherical surface, D, is:

$$D = (n_2 - n_1)/R \qquad (1)$$

where n_1 and n_2 are the indices of refraction in front of and behind the refracting surface and R its radius of curvature. This is an approximation that holds for paraxial rays. If R is in meters, then D will be in diopters or reciprocal meters.

The eye has many refracting surfaces and these must be combined to obtain the total refractive power of the eye. Two optical systems with refractive power, D_1

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and D_2, have a refractive power, F, as follows:

$$F = D_1 + D_2 - D_1D_2\frac{d}{n} \quad (2)$$

where d is the distance between the two refracting elements (measured in meters), and n is the refractive index of the medium between. If the two surfaces are close together (that is, if d is very small), then the last term of equation 2 can be ignored, and the total refractive power is the sum of the components.

For three element optical systems the expression becomes:

$$F = D_1(1 - \frac{d_2}{n_2}D_3) + D_2(1 - \frac{d_1}{n_1}D_1)(1 - \frac{d_2}{n_2}D_3) + D_3(1 - \frac{d_1}{n_1}D_1) \quad (3)$$

where D_1, D_2, D_3, and F are the refractive powers of the individual components, the combination d_1 and d_2 the distances between the components, and n_1 and n_2 are the indices of refraction of the media in between. A reasonable simplification is to add the refractive powers of the two surfaces of the cornea, the anterior surfaces of the crystalline lens cortex and nucleus, and the posterior surfaces of the nucleus and cortex of the crystalline lens, using equation 2. Equation 3 can then be used to give the total power of all three systems.

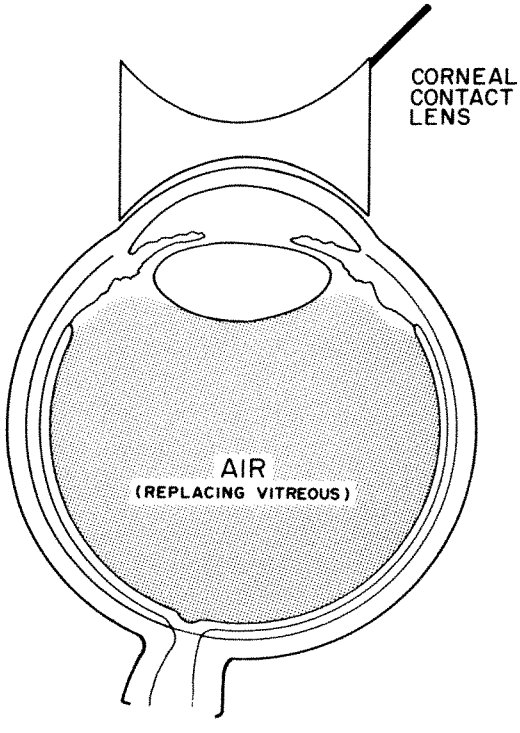


Fig. 1 (Landers, Stefánsson, and Wolbarsht). The biconcave corneal contact lens on the eye.

TABLE 1

GULLSTRAND'S SCHEMATIC EYE, RELAXED ACCOMMODATION*

Factor	Value
Index of refraction	
Cornea	1.376
Aqueous humor and vitreous body	1.336
Outer portion of lens	1.386
Core-lens	1.406
Air	1.000
Position of vertex of refracting surface (mm)	
Anterior surface of cornea	0.0
Posterior surface of cornea	0.5
Anterior surface of lens	3.6
Anterior surface of core-lens	4.1
Posterior surface of core-lens	6.6
Posterior surface of lens	7.2
Radius of curvature (mm)	
Anterior surface of cornea	7.7
Posterior surface of cornea	6.8
Anterior surface of lens	10.0
Posterior surface of lens	−6.0
Refractive power (diopters)	
Anterior surface of cornea	+48.83
Posterior surface of cornea	− 5.88
Anterior surface of lens	+ 5.00
Anterior surface of core-lens	+ 2.528
Posterior surface of core-lens	+ 3.472
Core-lens	+ 5.985
Posterior surface of lens	+ 8.33
Cornea	+43.05
Lens	+19.11
Whole eye	+58.64

*From Southall.[1]

RESULTS

The emmetropic eye has a refractive power of + 58.6 diopters when accommodation is relaxed. Most of this refraction takes place in the anterior cornea where the light passes from air that has a refractive index of 1.000 to the cornea that has

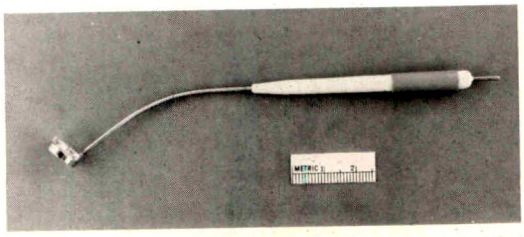


Fig. 2 (Landers, Stefánsson, and Wolbarsht). The biconcave operating lens.

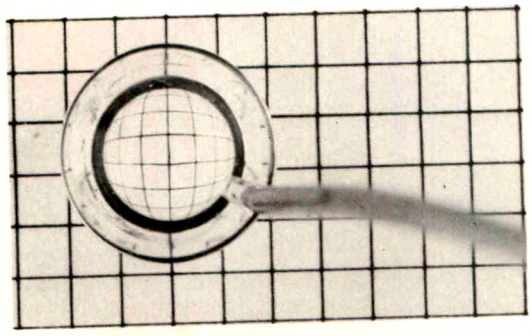


Fig. 3 (Landers, Stefánsson, and Wolbarsht). View through the biconcave operating lens.

a refractive index of 1.376. Inside the normal eye there are no interfaces with differences of refractive indices larger than 0.1 (Table 1).

A contact lens with a flat anterior surface is usually placed on the cornea during vitreous surgery. If a polymethylmethacrylate contact lens with a refractive index of 1.488 and a concave posterior surface radius of curvature of 7.7 mm is used, the refractive power of the anterior surface of the eye is changed from +48.8 diopters to −14.5 diopters (Table 2), and the fundus is easily seen. After lensectomy, when the lens has been replaced with fluid of the same refractive index as aqueous and vitreous, the refractive power of the eye is only that of the cornea, that is, +43.1 diopters. If a flat contact lens is placed on the eye after a lensectomy, the power of refraction of the entire eye becomes −20.4 diopters.

When an eye without a lens is filled with air, all the refraction occurs at the two surfaces of the cornea, which almost

TABLE 2

REFRACTIVE POWER OF THE HUMAN EYE AFTER VARIOUS SURGICAL PROCEDURES*

Cornea	Aqueous	Crystalline Lens	Vitreous	Refractive Power (Diopters)
Normal	Normal	Normal	Normal	+59
Flat-faced contact lens	Normal	Normal	Normal	0
Normal	Filled with air	Normal	Normal	+42
Flat-faced contact lens	Filled with air	Normal	Normal	−5
Normal	Normal	Replaced by aqueous	Normal	+43
Flat-faced contact lens	Normal	Replaced by aqueous	Normal	−20
Normal	Normal	Replaced by aqueous	Replaced by aqueous	+43
Flat-faced contact lens	Normal	Replaced by aqueous	Replaced by aqueous	−20
Normal	Filled with air	Filled with air	Filled with air	−6
Flat-faced contact lens	Filled with air	Filled with air	Filled with air	−70
Normal	Normal	Normal	Filled with air	+102
Flat-faced lens	Normal	Normal	Filled with air	+61
Symmetrical biconcave contact lens†	Normal	Normal	Filled with air	+22
Unsymmetrical biconcave contact lens (anterior surface, −93 diopters)†‡	Normal	Normal	Filled with air	0
Normal	Filled with air	Normal	Filled with air	+98
Flat-faced contact lens	Filled with air	Normal	Filled with air	+66
Unsymmetrical biconcave contact lens (anterior surface, −93 diopters)†	Filled with air	Normal	Filled with air	+17

*Based on Gullstrand's Schematic Eye, Accommodation Relaxed.[1]
†Refractive index, 1.488 (polymethylmethacrylate).
‡This lens gives the eye the same refractive power as that of a normal eye with a flat-faced contact lens.

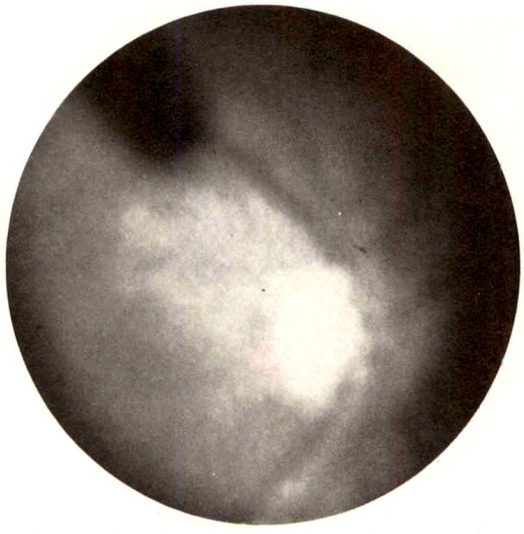


Fig. 4 (Landers, Stefánsson, and Wolbarsht). Human fundus seen through a symmetrical biconcave corneal contact lens. Air has replaced vitreous.

equal one another when air is on both sides. No optical aids are needed to see the fundus in this situation. If the lens is in place and the vitreous is replaced with air, the posterior surface of the lens becomes strongly refractive (+64 diopters) and the total refractive power of the eye increases from +59 to +102 diopters. A flat polymethylmethacrylate contact lens decreases this to +61 diopters but does not allow the fundus to be seen. Using an unsymmetrically biconcave corneal contact lens with an anterior surface radius of curvature of 5.25 mm and a posterior radius of curvature of 7.7 mm (Figs. 1 to 3) decreases the refractive power of the eye to that of a normal eye with a flat contact lens, allowing the surgeon to see the posterior pole (Fig. 4). A simple solution is a symmetrically biconcave contact lens in which both surfaces have a radius of curvature of 7.7 mm, reducing the refractive power of the eye to +22 diopters. The fundus can be seen almost as clearly through such a contact lens, which can be approximated by placing two ordinary flat-faced contact lenses back to back. A weak negative lens (approximately −0.87 diopter) adjacent to the 150-mm focal length objective lens of the operating microscope serves the same purpose (E. R. Noack, personal communications, 1980).

Putting air into the anterior part of the eye, as well as into the vitreous cavity, when the lens is in place makes the refractive power of the eye +99 diopters (Table 2). The unsymmetrical biconcave contact lens described above should permit good visualization of the fundus in this case.

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TEMPORARY KERATOPROSTHESIS FOR USE DURING PARS PLANA VITRECTOMY

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We have developed a series of temporary keratoprostheses that permit closed pars plana vitrectomy in eyes with abnormal corneas. These devices are placed into a trephined opening in the cornea, and provide a clear, stable view of the intraocular contents. Immediately after vitrectomy, the keratoprosthesis is replaced with a corneal graft. We have used this technique successfully in four eyes with opaque or severely traumatized corneas.

Closed pars plana vitrectomy is a useful technique for removing blood-stained vitreous, for preretinal membrane peeling, and for treating a variety of other anterior and posterior segment problems.[1] However, good visualization of the intraocular contents is necessary for vitrectomy surgery. Corneal opacities are often present in patients with recent trauma, blood-staining of the cornea, or corneal edema and scarring caused by disease or previous surgery. These corneal opacities have often made vision-saving pars plana vitrectomy impossible.

Although corneal transplantation can sometimes provide an optically clear view of the intraocular contents in eyes that had had opaque corneas, this is not always the case. Transient edema of the corneal graft often limits visualization of the posterior segment. Mechanical distortion present at the conclusion of the corneal graft procedure can limit visualization as well. Unfortunately, corneal transplants do not provide an excellent view of the fundus in some cases until several weeks after surgery. This delay is often undesirable, because the vitrectomy may be urgent. Also, the endothelial viability of a penetrating keratoplasty may be threatened by the large volume of irrigation fluid used and the manipulations of the globe performed during a complex vitrectomy.

To make closed pars plana vitrectomy possible in eyes with abnormal corneas, we have developed a series of temporary keratoprostheses to be used during operations in cases in which the view of the intraocular contents would be otherwise inadequate for vitreous surgery (D. Landers, unpublished data).

These keratoprostheses may be used to provide an optically clear view of the interior of the eye with minimal distortion. We have been successful with one such prosthesis, used in conjunction with closed pars plana vitrectomy in three eyes with chronic corneal edema and scarring, and in one eye with a penetrating injury.

MATERIAL AND METHODS

Principles of keratoprostheses—Cardona[2] has reviewed the development and principles of prosthokeratoplasty.

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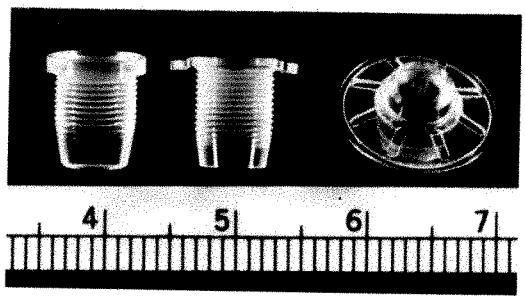


Fig. 1 (Landers and associates). The three types of temporary keratoprostheses. Type 1 is at left, type 2 in the middle, and type 3 at right.

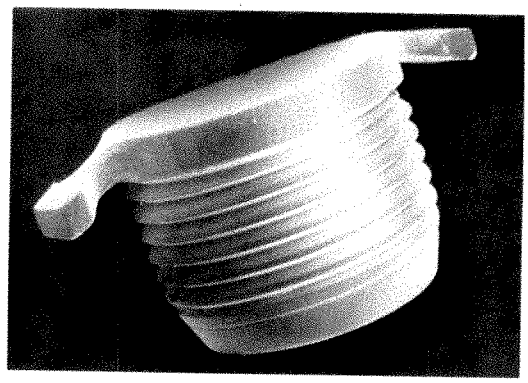


Fig. 2 (Landers and associates). In the type 2 keratoprosthesis, suture struts extend outward from the anterior edge; there is a threaded optical cylinder (×15).

However, the requirements for a keratoprosthesis to be used during surgery are somewhat different from those for one to be used on a permanent basis. A surgical keratoprosthesis remains in place for a few hours at most, and thus can be sutured to the cornea. A firm mechanical sealing of the corneal opening is sufficient, because a temporary keratoprosthesis will not melt the corneal stroma during the short period it is in position. A temporary keratoprosthesis should provide a wider-angled view of the fundus than a permanent keratoprosthesis as the surgeon needs to examine the peripheral retina during the vitrectomy. However, the diameter of the temporary keratoprosthesis should not be too large because this increases the chance of complications secondary to the subsequent corneal transplant.

The devices—We have designed three types of keratoprostheses (Fig. 1). All are fabricated of polymethylmethacrylate on a precision lathe, and have polished internal and external optical surfaces. Type 1 is a threaded optical cylinder 5.0 mm in length and 6.2 mm in diameter. The threaded shaft engages the corneal stroma to improve fixation. Type 2 is also a threaded optical cylinder 5.0 mm in length and 6.2 mm in diameter, but it also has struts at the anterior edge that allow it to be sutured to the cornea (Figs. 2 to 4). Type 3 is a smooth, beveled cylinder 5.0 mm in length and 6.2 mm in diameter at its outer extremity where a slotted circular flange allows interrupted- or continuous-suture fixation to the cornea. All three types of keratoprostheses have polished concave anterior and posterior surfaces with radii of 7.8 mm. All three have refractive powers of −135.6 diopters in air and −85.4 diopters in the aphakic, fluid-filled eye.

Type 1 can be used in opaque but structurally intact corneas. Type 2 is used in corneas with single lacerations or when manipulation or rotation of the eye is anticipated. This keratoprosthesis has withstood intraocular pressures of

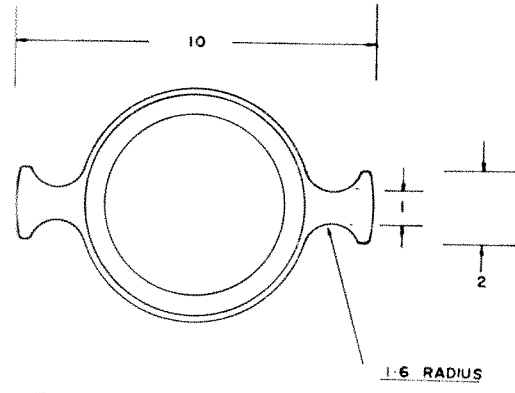


Fig. 3 (Landers and associates). Dimensions (in millimeters) of type 2 keratoprosthesis seen from the top.

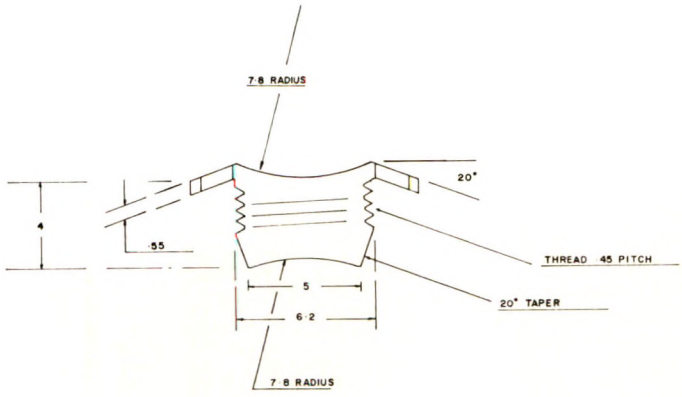


Fig. 4 (Landers and associates). Dimensions (in millimeters) of type 2 keratoprosthesis seen from the side.

70 mm Hg in animal and human eyes without allowing leakage of intraocular fluid or becoming dislodged. Type 3 is useful in corneas with many lacerations or deformities. All of the keratoprostheses have a length suitable for aphakic eyes. The concave optical surfaces provide a wider field for viewing the retina.

Case report

A 55-year-old man was referred here for a possible vitrectomy. The patient had a 20-year history of increased intraocular pressure that had required bilateral iridencleisis procedures and subsequent cataract extractions. The right eye did well except for late-onset cystoid macular edema that resulted in a visual acuity of 6/18 (20/60). The left eye required discission of a secondary membrane after an extracapsular cataract extraction. Subsequently, the secondary membrane recurred, along with opacification and condensation of the anterior vitreous and corneal edema. Visual acuity was 6/120 (20/400). The intraocular pressure was 35 mm Hg in both eyes. The left cornea had superficial peripheral vascularization

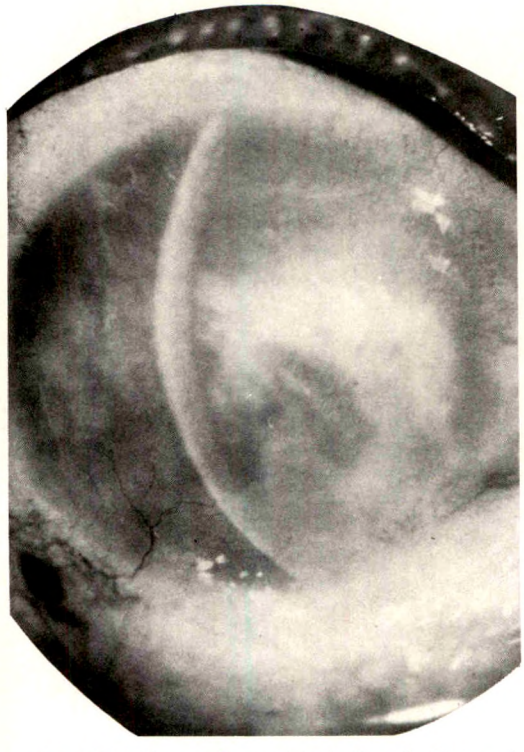


Fig. 5 (Landers and associates). Case 1. Cornea is diffusely hazy with edema and midperipheral vascularization.

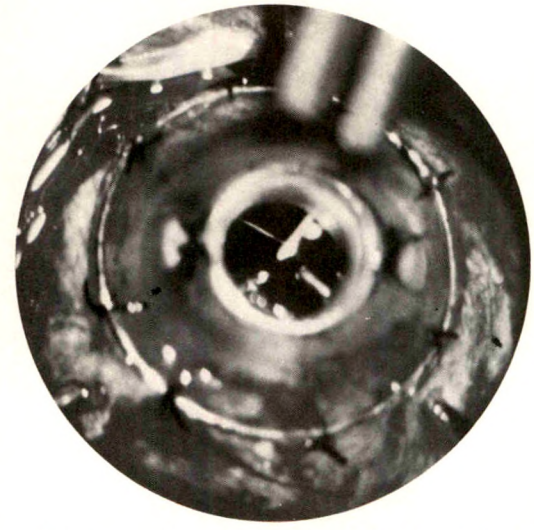


Fig. 6 (Landers and associates). Keratoprosthesis type 2, sutured in place. Fleiringa ring is visible. Vitrectomy instruments (including infusion needle, fiberoptic light pipe, and vitrectomy instrument) are visible through keratoprosthesis within vitreous cavity.

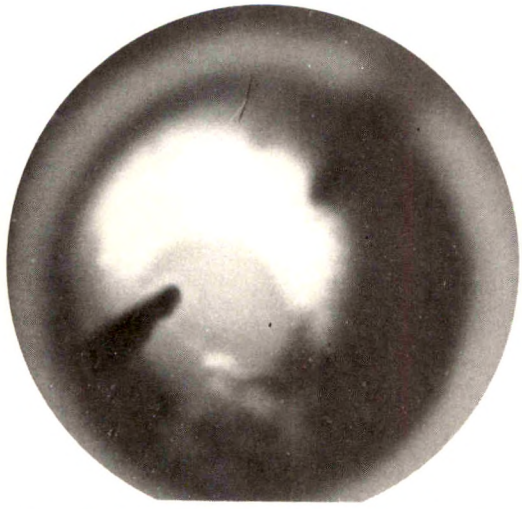


Fig. 7 (Landers and associates). Case 3. View through temporary keratoprosthesis shows fundus with retinal vessels and preretinal hemorrhage. Vitrectomy instruments can be seen through keratoprosthesis.

(Fig. 5). The anterior chamber was well-formed, but a dense secondary membrane and organized anterior vitreous obscured the view of the fundus. Ultrasonography of the left eye suggested an intact posterior segment without retinal detachment.

After the intraocular pressure was controlled with pilocarpine, the patient underwent surgery on the left eye. With the patient under general anesthesia, the surgeon prepared for a vitrectomy, and then attached a Fleiringa ring to the sclera with six interrupted silk sutures. Traction sutures were applied to the ring. He performed a corneal trephination with a 6-mm trephine and completed the incision with corneal scissors. He placed the 6.2-mm threaded keratoprosthesis with struts (type 2) into the cornea with a twisting motion and secured it with two 6-0 silk sutures about the struts (Fig. 6). The sclera was entered superonasally, superotemporally, and inferotemporally through the pars plana and a three-incision technique vitrectomy performed with the Ocutome system. After inspecting the peripheral retina and posterior pole with intraocular fiberoptic illumination (Fig. 7), he closed the scleral incisions. A 6.5-mm trephine was then used to cut the corneal donor button, from a cornea stored in McCarey-Kaufman medium, by the posterior punch technique against a Teflon block.[3] The surgeon removed the keratoprosthesis (Fig. 8) and secured the corneal graft in place with 16 interrupted 10-0 nylon sutures. The wound was watertight after the anterior chamber was reformed.

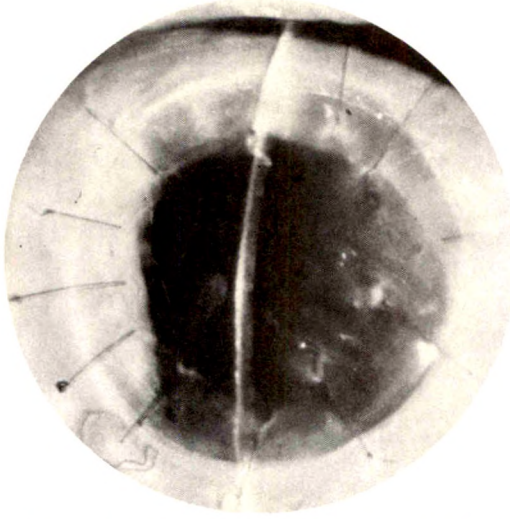


Fig. 9 (Landers and associates). Case 1. Clear, 6.5-mm corneal graft in place two weeks after vitrectomy carried out through 6.2-mm temporary keratoprosthesis type 2.

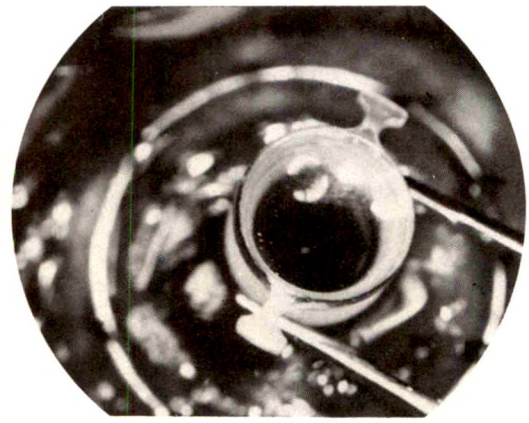


Fig. 8 (Landers and associates). Case 3. Keratoprosthesis type 2 being removed. Fleiringa ring is in place. Suture struts protrude from the anterior edge of the keratoprosthesis.

Postoperatively, there was mild edema of the transplant which cleared in the early postoperative period. The retina was normal. At the first postoperative visit, best corrected visual acuity in the operated upon eye was 6/30 (20/100). Results of an ophthalmoscopic examination were normal. There was moderate corneal astigmatism (Fig. 9). Six months postoperatively visual acuity improved to 6/18 (20/60). The fundus appeared normal through the grafted cornea.

The three other cases in which the temporary keratoprosthesis was used are summarized in the Table.

Discussion

Corneal opacities interfering with visualization of the intraocular structures can

TABLE

CLINICAL SUMMARY OF FOUR CASES IN WHICH TEMPORARY KERATOPROSTHESES WERE USED

Clinical Data	Patient			
	1	2	3	4
Age (yrs)	55	59	62	27
Cause of problem	Extracapsular cataract extraction	Battery explosion	Injury from nail	Metallic foreign body
Corneal abnormality	Vascularization	Scar	Scar	Laceration
Vitreal abnormality	Mid-vitreous membrane	Hemorrhage	Hemorrhage	Hemorrhage
Retinal abnormality	—	Chronic detachment	Chronic detachment	Foreign body
Final visual acuity	6/18 (20/60)	Late enucleation	6/240 (5/200)	6/9 (20/30)

be managed by removal of the cornea during surgery (open-sky technique), a prior penetrating keratoplasty (R. Castroviejo, unpublished data), or with a temporary surgical keratoprosthesis. One disadvantage of open-sky vitrectomy is that rotation or positioning of the eye is limited and may distort the ocular architecture and allow the intraocular contents to prolapse when pressure is applied to the eye or orbit.[4] A prior corneal transplantation may mean days or weeks of waiting for sufficient corneal clarity and wound strength to allow an adequate vitrectomy. Additionally, the corneal transplant may be rejected or become clouded after subsequent intraocular surgery.[5] The temporary surgical keratoprosthesis eliminates these problems and allows early surgery under the controlled situation of an essentially intact globe.

We recommend applying a Fleiringa ring during this procedure to provide anterior support and to prevent the globe from collapsing. Using an oversized donor graft is advisable because some dilation of the recipient bed can occur. Intraocular fiberoptic and other instruments may be used concurrently with the keratoprosthesis; the prosthesis has proven to be stable and secure in eyes from eye banks and in the several cases in which it has been used.

The concave anterior optical surface provides an excellent view of the posterior segment without distortion and with minimal annoying reflexes. The depth of field is excellent. The concave posterior optical surface minimizes the possibility of scratching the posterior surface with the vitrectomy instruments during surgery.

The anterior surface can be altered immediately by introducing a small drop of water onto the optical bowl, creating a convex anterior surface and allowing a shift of the focal plane to a more anterior position in the operative field without adjusting the microscope. We have not encountered some anticipated problems of limitation of the view of the peripheral fundus because the globe can be readily rotated.

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MEDICOLEGAL CLAIMS IN INTRAOCULAR LENS IMPLANT CASES

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In 11 of 22 medicolegal claims involving intraocular lens implants, the medical care given to the patients was judged to be substandard, whereas this was true in only 19 of 110 (17.3%) ophthalmic claims not involving intraocular lenses. The major reason for this difference was that the intraocular lenses were implanted in cases where they were not indicated. Also, many of these patients had relatively good vision before the surgery and were more likely to sue if the results were poor.

The number of medicolegal claims involving intraocular lens implants has increased in recent years. In the series reported here, for example, there were two claims in 1977, eight in 1978, 14 in 1979, and seven in the first six months of 1980. Obviously, one reason for this increase is that more intraocular lenses are being implanted.

I reviewed a series of 441 ophthalmic claims. Of these, 31 (34 eyes), or 7%, involved intraocular lens implants. It must be remembered that two years or more may elapse before a patient's claim is reviewed by an expert and five years before the case is adjudicated and judgment rendered.

Experienced attorneys estimate that 75% of the claims are rejected before medical advice is sought, and three out of four of the remaining 25% are rejected after medical advice is obtained.[1] Thus, the standard of medical care the patient received can be evaluated only in a small selected group of cases.

METHOD

From the series of 441 claims, I selected 157 for evaluation. Of these, 31 involved intraocular lens implants and 126 did not. Some cases were eliminated from the statistical evaluation because the standard of care could not be determined or was not relevant to the claim. This left 22 claims that involved intraocular lens implants and 110 that did not (Table 1).

RESULTS

In my judgment, 50% (11 of 22) of the cases involving intraocular lenses were associated with substandard care. This figure is considerably higher than 17.3% (19 of 110 claims) I found in the cases that did not involve intraocular lenses (Table 1).

Much of this difference can be explained by an examination of the reasons I judged the medical care to be substan-

TABLE 1

EVALUATION OF CLAIMS

Evaluation	With Implants	Without Implants
Total No.	31	126
No. analyzed	22	110
Substandard care	11	19
Standard care	11	91
Indefinite	8	8
Product liability cases	2	—
Faulty instruments	—	4
No injury	—	3
Fraud	—	1

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dard in 11 claims involving intraocular lens implants (Table 2).

Case 1—The patient had had preoperative myopia of 11 diopters. The visual result of the surgery was poor.

Case 2—The surgeon operated on the wrong eye. The visual result was poor.

Case 3—The patient had undergone penetrating keratoplasty, phacoemulsification, and an implant in one eye. Despite endothelial damage in this eye, an intraocular lens was implanted in the fellow eye which had glaucoma.

Case 4—After losing one eye which had undergone an implant, the patient underwent an implant in the fellow eye. The visual result was poor.

Case 5—The implanted intraocular lenses failed in both eyes.

Case 6—A one-eyed patient underwent keratoplasty, cataract extraction, and implantation in the remaining eye. The visual result was poor.

Case 7—A patient with glaucoma underwent a secondary implant after a previous filtering operation.

Case 8—A patient with glaucoma in his one remaining eye underwent an implant after three previous filtering operations.

TABLE 2

REASONS FOR POOR RESULTS

Reason	No. of Cases
Lens touching cornea, producing opacity	12
Lens luxated or subluxated	7
Significant inflammation	5
Retinal detachment	11
Secondary glaucoma	5
Endophthalmitis	1
Intraocular lens had to be removed	9
Total	50*

*The total is more than 22 because some cases had several complications.

Case 9—The surgeon was aware of a probable retrobulbar hemorrhage but proceeded with the cataract extraction and implant. There was vitreous loss and the visual result was poor.

Case 10—The suture became entangled in the foot plate of the intraocular lens. The surgeon cut the suture, but did not resuture the lens which became dislocated. This was the surgeon's first implant operation and no experienced surgeon was present.

Case 11—The surgeon admitted that the patient had not been informed that an intraocular lens was to be implanted. The visual result was poor.

DISCUSSION

I judged the medical care to be substandard primarily because intraocular lens implants were not indicated in these cases. This suggested that the surgeons involved were too bold and were not restrained by the generally accepted guidelines.

A contributing factor was that a number of the patients had relatively good vision before surgery. Preoperative visual acuities were recorded in 19 of the 22 cases involving implants. Only four of the 19 patients had visual acuities of 6/12 (20/40) or worse in the better eye. Four had visual acuities of 6/15 (20/50) in the worse eye. These patients have more to lose, and they are more inclined to sue if the surgical outcome is poor.[2]

Greater restraint on the part of this small minority of implant surgeons would reduce the incidence of claims.

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MICONAZOLE THERAPY FOR KERATOMYCOSIS

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I treated seven patients with keratomycosis (four with *Candida* infections and three with *Aspergillus* infections) with topical and subconjunctival miconazole. Progressive corneal ulceration stopped in each case, and clinical evidence of corneal infection disappeared. Posttreatment visual acuities were at least as good as (and usually better than) pretreatment visual acuities. Superficial punctate keratitis was associated with prolonged (one to two weeks) hourly instillation of miconazole, but there was no evidence of serious ocular toxicity.

During the past 30 years, fungal infections have been increasingly reported as a cause of keratitis.[1] The less-than-optimal resolution of such cases has been attributed to delayed diagnosis and to the lack of safe, effective antifungal agents that adequately penetrate ocular structures.[1-3] In 1975, pimaricin (Natamycin), a broad-spectrum antifungal agent with low toxicity, became the first antifungal agent approved for ocular use in the United States. It is effective for superficial corneal ulcers caused by fungi sensitive to it, but, because it does not penetrate the cornea or sclera well, it is not effective against deeper corneal ulcers.[1] Amphotericin B does not penetrate well either, and is extremely toxic.[4] Flucytosine is well tolerated,[4] but has a limited antifungal range.[1]

Miconazole is a broad-spectrum antifungal agent that has already proven to be safe and effective in human keratomycosis.[1] In animal experiments, this drug was well tolerated topically and subconjunctivally, and it penetrated the ocular coats readily.[4,5] I recently used miconazole to treat seven cases of keratomycosis in humans.

SUBJECTS AND METHODS

The seven patients were found to have keratomycosis after being referred here. Table 1 shows their clinical characteristics at my initial examination.

Scrapings from the edges and deep base of each corneal ulcer were obtained for Gram and Giemsa staining. They were also used for culture at 35 C on Sabouraud's medium and in cooked meat broth, and at 20 C on blood agar. I used the following criteria for diagnosis of fungal keratitis: (1) clinical features compatible with fungal keratitis; (2) fungal elements seen on stained smears of the corneal scrapings; and (3) growth of fungal organisms in the "C streaks" on at least two culture plates with scrapings from the involved cornea. Antifungal sensitivities were determined with tube and plate dilution methods.[6] All patients were then treated in the hospital with 0.3% topical

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TABLE 1

CLINICAL SUMMARY OF PATIENTS WITH KERATOMYCOSIS AT INITIAL EXAMINATION

Case, Sex, Age (yrs)	Systemic Disease	Corneal Status Before Keratomycosis	Ocular Drug Use	Visual Acuity of Affected Eye	Ulcer Characteristics
1, M, 36	None	L.E.: Exposure keratitis secondary to traumatic palsy of 7th cranial nerve (4 yrs)	Dexamethasone, chronic use	6/12 (20/40)	Peripheral ulcer, 2 × 3 mm; anterior 25% of stroma lost; dense white stromal infiltrate with feathery borders; endothelial plaque; small hypopyon.
2, F, 63	None	Normal	None	C.F. at 2 ft*	Large central ulcer with many dense white stromal infiltrates; endothelial plaque; small hypopyon.
3, F, 67	None	Normal	None	6/6 (20/20)	Peripheral ulcer with surrounding and underlying dense white infiltrate.
4, M, 45	None	Corneal transplant for aphakic bullous keratopathy (1 mo previously)	Dexamethasone and gentamicin	6/60 (20/200)	Paracentral superficial ulcer with underlying and surrounding dense white stromal infiltrate; two satellite superficial infiltrates.
5, F, 44	Chronic muco-cutaneous candidiasis	Bilateral recurrent corneal ulcers (10 yrs)	Artificial tears	H.M.*	Inferior, paracentral descemetocele, 4 × 3 mm; 2 fluffy white infiltrates on Descemet's membrane; diffuse stromal infiltrate extending from edges of descemetocele.
6, M, 43	Diabetic acidosis	No history of corneal disease	None	6/60 (20/200)	Central epithelial defect 6 mm in diameter with faint anterior stromal haze.
7, F, 38	None	Normal	None	6/60 (20/200)	Central corneal ulcer (3 mm in diameter; 25% of corneal thickness in depth) with fluffy white infiltrate in underlying and surrounding stroma; 2 satellite stromal lesions; endothelial plaque; small hypopyon.

*C.F., counting fingers; H.M., hand motions.

TABLE 2

TREATMENT AND RESULTS

Case	Organism	Sensitivity of Organism (μg/ml)			Treatment*†	Anatomic Result	Final Visual Acuity in Affected Eye
		Miconazole	Amphotericin B	Flucytosine			
1	*Aspergillus fumigatus*	0.2	0.7	0.4	Miconazole (1 drop/hr for 5 days; then 1 drop/waking hr for 2 wks; then 1 drop 6 times/day for 3 wks)	Healed ulcer with peripheral corneal stromal thinning and scarring.	6/12 (20/40)
2	*Aspergillus flavus*	0.4	>100	20.0	Miconazole (subconjunctival, 10 mg/day for 5 days; 1 drop/hr for 5 days; then 1 drop/waking hr for 4 wks; then 1 drop 6 times/day for 7 wks)	Healed ulcer with clearing of stromal infiltrates and hypopyon; corneal scar.	C.F. at 2 ft‡
3	*Aspergillus fumigatus*	0.4	0.8	40.0	Miconazole (subconjunctival, 10 mg/day for 3 days; 1 drop/hr for 5 days; then 1 drop/waking hr for 2 wks; then 1 drop 6 times/day for 3 wks)	Healed ulcer with clearing of stromal infiltrate; small peripheral anterior stromal scar.	6/6 (20/20)
4	*Candida albicans*	0.2	0.8	0.4	Miconazole (subconjunctival, 10 mg once; 1 drop/ hr for 4 days; then 1 drop/ waking hr for 3 wks; then 1 drop 6 times/day for 4 wks)	Healed ulcer with paracentral stromal scar.	6/18 (20/60)
5	*Candida albicans*	0.2	0.6	0.1	Miconazole (subconjunctival, 10 mg/day for 4 days; 1 drop/hr for 10 days; then 1 drop/waking hr for 4 wks; then 1 drop 6 times/day for 4 wks); gentamicin (subconjunctival, 40 mg/day for 4 days; 1 drop/hr for 10 days; then 4 times/day for 2 wks);	Clear lamellar graft; white infiltrates at host-graft interface disappeared; chronic *Candida* infection cleared up.	6/60 (20/200)
	Staphylococcus aureus	Sensitive to cefazolin and gentamicin					
	Proteus mirabilis	Sensitive to cefazolin and gentamicin					

					Cefazolin (subconjunctival, 133 mg/day for 4 days; 1 drop/hr for 10 days; then 4 times/day for 2 wks); Ketoconazole (oral, 200 mg/day beginning 14 days after surgery)		
6	*Candida albicans*	0.8	0.1	0.4	Miconazole (subconjunctival, 10 mg/day for 4 days; 1 drop/hr for 10 days; then 1 drop/waking hr for 3 wks; then 1 drop 6 times/day for 4 wks)	Healed ulcer; mild anterior paracentral stromal haze.	6/24 (20/80)
7	*Candida albicans*	0.4	0.4	0.2	Miconazole (subconjunctival, 10 mg/day for 5 days; 1 drop/hr for 5 days; then 1 drop/waking hr for 4 wks; then 1 drop 6 times/day for 4 wks)	Healed ulcer; stromal scar	6/15 (20/50)

*All patients also received 4% atropine. All except Patient 5 also received 0.3% topical gentamicin 4 times per day for broad-spectrum antibacterial protection because of ulcerating devitalized tissue.

†Concentrations of the topically administered drugs were as follows: miconazole (in the form of the intravenous preparation Monistat), 10 mg/ml; gentamicin, 14 mg/ml; and cefazolin, 133 mg/ml.

‡C.F., counting fingers.

gentamicin, 4% atropine, topical miconazole (10 mg/ml), and subconjunctival miconazole (10 mg).

I have selected three of the seven cases for discussion. The treatment and results for all seven cases are summarized in Table 2.

CASE REPORTS

Case 2—A 63-year-old woman was referred here for evaluation and treatment of a corneal ulcer. The ulcer had been treated at another hospital for 18 days with gentamicin (administered topically every hour and subconjunctivally once each day) and bacitracin (administered topically every hour). Several scrapings and cultures at the referring hospital failed to isolate any organisms. Five days before the patient was admitted to the referring hospital, her right eye had become red and painful. She treated the eye with teabag compresses.

Visual acuity at my first examination of the patient was R.E.: counting fingers at 2 ft and L.E.: 6/12 (20/40). The right cornea was studded with numerous stromal infiltrates. A large endothelial plaque and a small hypopyon with hyphema were present (Fig. 1, left). Corneal scrapings showed hyphal forms. Three days after the corneal scrapings were cultured, *Aspergillus flavus* was growing on the blood agar. Therapy with topical and subconjunctival miconazole was begun (Table 2). Over the next 12 weeks, the corneal infiltrates and hypopyon vanished. The patient was left with a scarred cornea and visual acuity of counting fingers at 2 ft in her right eye (Fig. 1, right).

Case 5—A 44-year-old woman was admitted here for treatment of a descemetocele in her left eye. Chronic mucocutaneous candidiasis had been diagnosed in this patient when she was 6 years old; she had developed idiopathic thrombocytopenic purpura at the age of 16 years. The subsequent appearance of arthritis and positive serologic findings resulted in the diagnosis of systemic lupus erythematosus. Her ten-year history of ocular involvement in candidiasis was marked by episodes of bilateral corneal ulcers and progressive corneal vascularization. The predominant form of antifungal therapy had been intravenous and topical amphotericin B and oral clotrimazole.

At my first examination of this patient, her visual acuity was hand motions in both eyes. The conjunctiva of each eye was injected, and the inferior fornix foreshortened. Extensive corneal scarring and vascularization were present in the right eye; there was a wedge-shaped superficial white plaque (Fig. 2, left). In the left eye there was a horizontally oriented inferior descemetocele, with two fluffy, white infiltrates on Descemet's membrane (Fig. 3, top left and top right). Corneal scrapings of the edges of the ulcer yielded gram-positive cocci, gram-negative rods, and yeast forms. Therapy was begun with topical and subconjunctival gentamicin, cefazolin, and miconazole (Table 2). The topical medications were instilled hourly, 15 minutes apart.

Two days after she was admitted to the hospital, the patient underwent lamellar keratoplasty carried down to Descemet's membrane in her left eye. The white infiltrates on Descemet's membrane were still present after the operation. Cultures of the infiltrates obtained during surgery grew *Candida* organisms. The infiltrates vanished during the 14 days of postoperative therapy (Fig. 3, bottom left). Systemic ketoconazole therapy (200 mg/day, taken orally) was then begun; during the next four weeks the patient's topical medication was gradually reduced and discontinued. The patient has continued to take systemic ketoconazole for six months, and has been free of *Candida* infections during that time. Visual acuity is now 6/60 (20/200) in both eyes. The right eye has improved: the white plaque has disappeared, and the corneal vessels have emptied (Fig. 2, right).

Case 6—A 43-year-old man was admitted here while in a state of diabetic acidosis. At the time of admission, the medical team noted conjunctival edema and injection of the right eye and candidal intertrigo and sought an ophthalmologic consultation. There was a central epithelial defect in the right eye. When the patient first regained consciousness, his visual acuity was 6/60 (20/200) in the right eye. A faint anterior stromal haze was present in the area of the epithelial defect. Candidal keratitis was suspected. Corneal scrapings confirmed this diagnosis; budding yeast forms and pseudohyphae were seen on smear, and *Candida albicans* grew on blood agar at room temperature. Therapy with subconjunctival and topical miconazole continued for the next nine weeks (Table 2). The corneal ulcer resolved, leaving a paracentral corneal stromal scar and a visual acuity of 6/24 (20/80) in the right eye.

RESULTS

In five of the seven patients, there was evidence of deep corneal foci of fungal invasion. In each case, after four to six weeks of miconazole therapy progressive corneal ulceration stopped and clinical evidence of corneal infection disappeared. In Case 5, I used lamellar keratoplasty as well as antifungal therapy with miconazole and ketoconazole. Topical miconazole therapy was routinely continued for six to 12 weeks. Ocular integrity was preserved in each instance; corneal scarring was left after the infections were resolved. Posttreatment visual acuities were at least as good as (and usually better than) pretreatment visual acuities (Tables 1 and 2). There was no significant

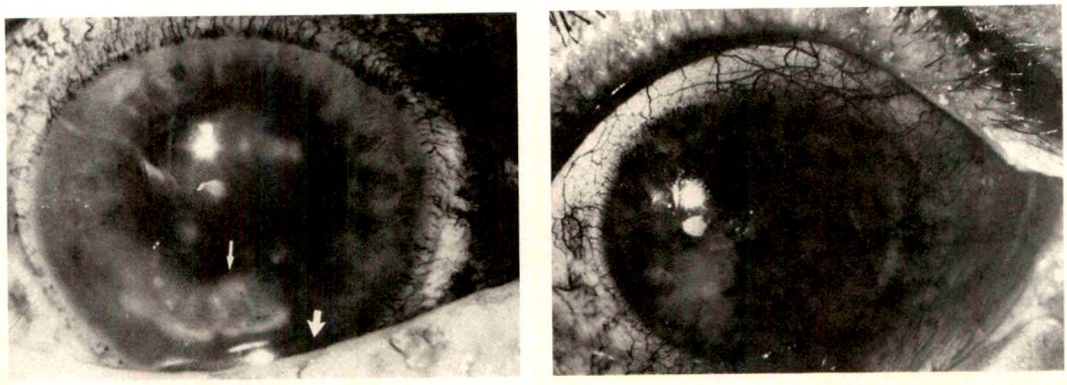


Fig. 1 (Foster). Case 2, right eye. Left, At first examination. Note the numerous white stromal infiltrates, endothelial fibrin plaque (thin arrow), and mixed hypopyon-hyphema (thick arrow). Right, After six weeks of therapy.

clinical evidence of ocular toxicity of miconazole, although hourly instillation of the drug appeared to contribute to superficial punctate keratitis. These changes resolved when the frequency of instillation of all ocular drugs was reduced. Subconjunctival miconazole (10 mg/day for up to five days) was well tolerated.

Discussion

In treating patients with corneal fungal infections, ophthalmologists are confronted with three major difficulties: the limited selection of antifungal agents available, the poor intraocular penetration of many of the available agents, and the toxicity of these agents. The need for safe, effective antifungal agents with broad ranges and good ocular penetration has been expressed many times since Leber[7] first described keratomycosis. Jones[1] emphasized the disastrous outcome of many cases of keratomycosis. He also stressed the poor ocular penetration and unacceptable toxicity of amphotericin B and the poor penetration of pimaricin. His early clinical experience with imidazole antifungal agents, including miconazole, suggested that these drugs have the characteristics necessary for use as ocular antifungal agents: high antifun-

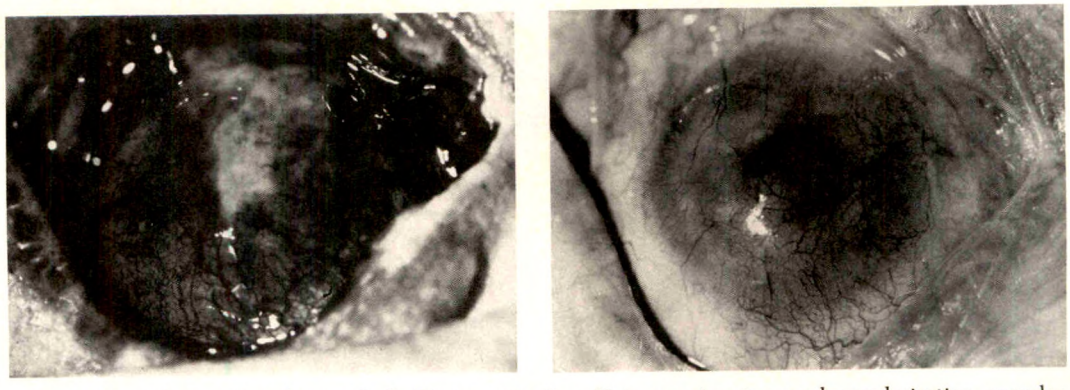


Fig. 2 (Foster). Case 5, right eye. Left, First examination. Note prominent corneal vascularization, vascular engorgement, and white, wedge-shaped superficial corneal infiltrate. Right, After 12 weeks of oral ketoconazole therapy.

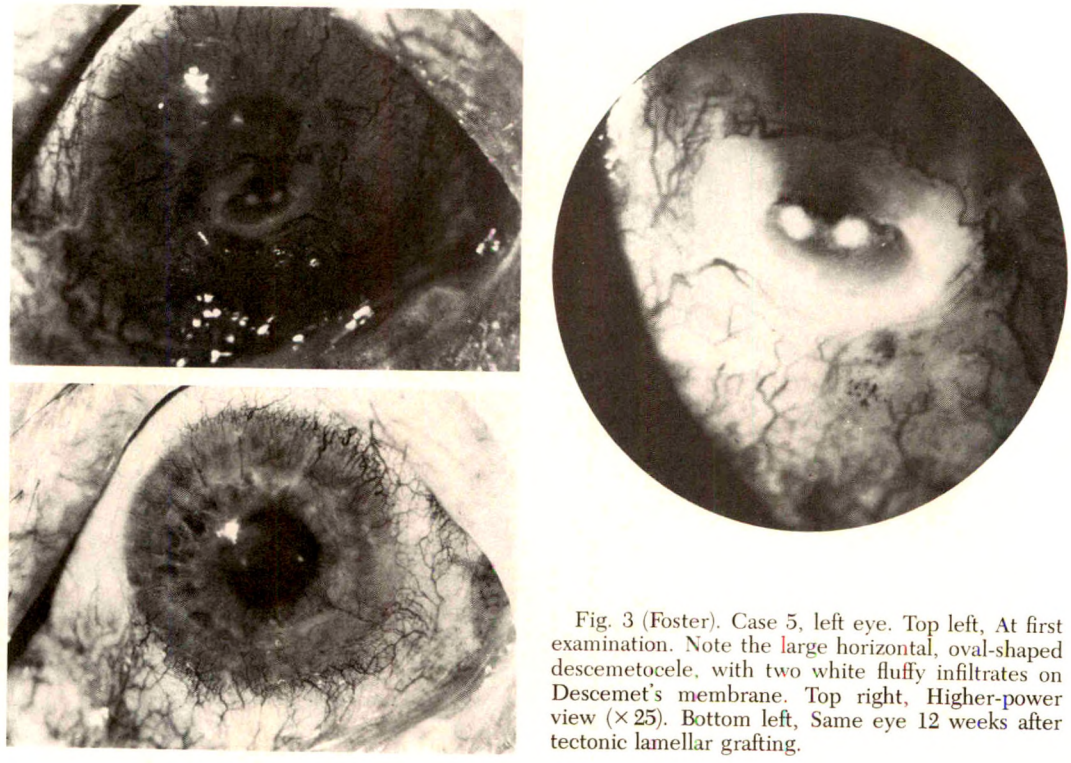


Fig. 3 (Foster). Case 5, left eye. Top left, At first examination. Note the large horizontal, oval-shaped descemetocele, with two white fluffy infiltrates on Descemet's membrane. Top right, Higher-power view (×25). Bottom left, Same eye 12 weeks after tectonic lamellar grafting.

gal activity, a broad range, the ability to penetrate the ocular coats, and nontoxicity. Other studies[4,5] defined more precisely the ocular penetration and toxicity of miconazole. This report confirmed Jones's[1] findings on the clinical efficacy of miconazole for keratomycosis caused by *Candida* and *Aspergillus* organisms. Miconazole, whether administered topically, subconjunctivally, or both, appears to be an alternate method of treating keratomycosis.

In Case 5, the role ketoconazole played in the outcome in the left eye and in the improvement in the right eye is unclear. In vivo data in animals[5] suggest that orally administered ketoconazole penetrates the cornea extremely well. For this reason (as well as my confidence in miconazole penetration into the cornea and aqueous humor after topical and subconjunctival administration), I believed lamellar keratoplasty was the most reasonable choice of surgical procedures for this patient. Lamellar keratoplasty is, in general, ill-advised in keratomycosis, because fungi remaining in the host tissue may proliferate and produce progressive fungal keratitis postoperatively. I was concerned about postoperative wound healing because of the extreme bilateral pathologic changes of the ocular surface and the conjunctival cicatrization in Case 5. I believed that lamellar grafting was safer than penetrating keratoplasty in this circumstance and that any remaining fungi could be eradicated by topical and subconjunctival miconazole and orally administered ketoconazole.

The significance of the white plaque in the right cornea of this patient is unclear. Cultures of the ocular surface epithelium

(cornea and conjunctiva) of the right eye were positive for *C. albicans*. This eye did not receive topical antifungal therapy; surface cultures done after six weeks of oral ketoconazole therapy were negative.

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Fifty years ago this month in THE JOURNAL

The scientific proceedings of the Amsterdam Congress of 1929 give concrete expression to the truth that all parts of the world can furnish observations which throw new light on our problems and furnish new inspiration and new methods for their solution. Different nations, like different individuals, may see things differently; and, reporting what they see, may bring individual contributions of high value to our swiftly developing stock of common knowledge. In Switzerland Gonin, influenced by the views of Leber of Heidelberg, and corroborated by those who first learned of his new treatment for detached retina, has almost reversed the prognosis and attitude of ophthalmologists toward that condition.

Jackson, E.: *World Ophthalmology*
Am. J. Ophthalmol. 14:456, 1931

FUNGAL GROWTH IN APHAKIC SOFT CONTACT LENSES

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Fungal growth was detected in two aphakic hydrophilic contact lenses. *Aspergillus* species was cultured from the extended-wear contact lens and *Dermatophilus congolensis* was identified by specific antibody staining of the daily-wear contact lens. As in the 16 previously described cases of fungal contact lens growth, our patients had no evidence of ocular infection but did experience irritation which cleared up when contact lens wear was discontinued.

Although only a small percentage of contact lenses are prescribed because of aphakia, these thicker contact lenses may be more susceptible to fungal growth and should be monitored carefully.

Fungal growth within hydrophilic contact lenses is uncommon, although in one series, 14% of soft contact lenses gave positive cultures for fungi after home sterilization.[1] We studied two cases of fungal growth in hydrophilic contact lenses, including what we believe to be the first known instance of *Dermatophilus congolensis* contamination. To the best of our knowledge, *D. congolensis* has not been previously reported in the ocular flora, but is recognized as a cause of pustular cutaneous streptothricosis in animals[2] and, on rare occasions, in animal handlers.[3]

Fungal growth has been previously reported in 16 other hydrophilic contact lenses. Six were Bionite-Griffin lenses (now known as Softcon),[4-7] seven were Bausch & Lomb lenses,[7-9] and three were unidentified lenses.[9] These apparent failures developed after both cold and heat sterilization and involved a spectrum of common skin and environmental contaminating fungi. Interestingly, both of our cases and one of those previously reported occurred in the thicker aphakic contact lenses.

CASE REPORTS

Case 1—A 58-year-old woman with insulin-dependent diabetes had had a cataract removed in March 1977. She was fitted by her optometrist with a daily-wear Bausch & Lomb soft contact lens eight weeks after surgery. On routine examination ten months later, the patient's eye and contact lens were found to be normal by one of us (R. O. B.). Fifteen months after surgery, she returned stating that she had stopped using the contact lens because it felt uncomfortable. The slit lamp showed that the contact had several areas of feathery, white opacification that appeared to be fungal growth. Upon questioning, the patient indicated that heat sterilization of the lens had been erratic. Her eye was otherwise normal and she was given a new contact lens. The patient has had no subsequent evidence of ocular or systemic infection.

Pathologic findings—Examination of the contact

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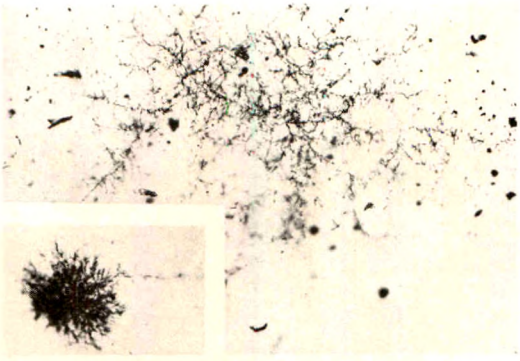


Fig. 1 (Berger and Streeten). Case 1. Filamentous network of *Actinomyces* organisms within the contact lens. Inset, A small dense colony of the organisms (×300).

lens under magnification showed several 2- to 3-mm stellate areas of a delicate filamentous network (Fig. 1) extending from the anterior surface deep into the contact lens, involving its central two-thirds. Bacterial and fungal cultures from one half of the contact lens disclosed no growth. Paraffin sections of the other half stained with hematoxylin and eosin showed thin, colorless threads with eosinophilic areas resembling nuclei. The Giemsa stain was best for demonstrating the organisms as blue filamentous strands interspersed with red beading and ending in occasional rounded blue bodies resembling spores (Fig. 2). They were similar to the *Streptothrix* organisms found in lacrimal canaliculitis.

Sections sent to the Communicable Disease Center in Atlanta showed an actinomycete with a morphologic resemblance to *D. congolensis*, and staining positively with fluorescent antibody to this organism.

Case 2—A 62-year-old man had been wearing CooperVision contact lenses continuously since a bilateral cataract extraction. He had undergone cataract surgery in the left eye in March 1980 and a CooperVision contact had been fitted in April. Six months later he returned, complaining that the left eye had been red and irritated for the previous four days. The contact lens had not been removed or cleaned in the interim. The conjunctiva was mildly injected without exudate or watering. Slit-lamp examination of the cornea and anterior segment disclosed no abnormalities except the aphakia. The contact lens, however, showed a white, feathery fungal patch at one edge. The patient suggested that this contamination might have occurred when some water from his dog's feeding dish splashed into his eye a week or so previously. After removal of the contact lens for culture, the irritation cleared up rapidly without treatment, and there were no symptoms from a new contact lens.

Pathologic findings—The specimen was a 10-mm soft contact lens which had a 1.75-mm round white spot near one edge. Magnification showed obvious coarse hyphae in a feathery radial growth pattern.



Fig. 2 (Berger and Streeten). Top, Case 1. The *Actinomyces* organisms appear as beaded filaments with occasional larger round forms (Giemsa, ×600). Bottom, Case 2. *Aspergillus* organisms permeating the contact lens (PAS, ×600).

Culture of one half of the contact lens and lesion for bacteria and fungi disclosed *Aspergillus* species.

Microscopic examination of paraffin sections showed large segmented and branching mycelia throughout the thickness of the contact lens (Fig. 2) that stained well with all fungal stains.

Discussion

Our cases of soft contact lens fungal contamination were similar to those previously reported (Table). One difference was that none of the previously described patients was symptomatic or had evidence of ocular infection during the observation period, whereas both of our patients had symptoms that cleared up rapidly after they discontinued wearing the contact lenses. In neither case, however, was there any evidence of a fungal infection of the eye, confirming the surprisingly small effect of a continuous fungal inoculum on the eye. Palmer, Ferry, and Safir[6] observed that the fungi

TABLE

REPORTED CASES OF FUNGAL CONTAMINATION IN HYDROPHILIC CONTACT LENSES

Study	Brand	No.	Type	Sterilization Protocol	Fungus
Bernstein[4]	Bionite	1	Cosmetic	3% hydrogen peroxide	*Rhodotorula* species
Shapiro[5]	Bionite	1	Aphakic, extended-wear	None	*Penicillium* species
Palmer, Ferry, and Safir[6]	Bionite	1	Cosmetic	Flexol	Ascomycetes
Morgan[7]	Bionite	3	Unknown	two, 2% hydrogen peroxide; one, Flexol	*Penicillium* species
	Bausch & Lomb	5	Unknown	Heat	Unknown
Sagan[8]	Bausch & Lomb	1	Unknown	None	*Aspergillus niger*
Gasset, Mattingly, and Hood[9]	Bausch & Lomb	1	Cosmetic	Unknown	*Candida albicans*
	Other	3	Cosmetic	Unknown	Unknown
Berger and Streeten	Bausch & Lomb	1	Aphakic	Heat	*Dermatophilus congolensis*
	CooperVision	1	Aphakic, extended-wear	None	*Aspergillus* species

were in the matrix of the contact lens and not on its surface, which might offer the eye a margin of safety. However, surface fungi were present on our extended-wear contact lens (Case 2), so that the possibility of infection after abrasion was real.

No conclusion can be drawn regarding the susceptibility of soft contact lenses to specific fungi because several different fungi have been found (Table). Wilson and associates[10] reported that four of the five fungi found, *Aspergillus*, *Penicillium*, *Candida*, and *Rhodotorula*, were chance contaminants of the external eye. Actinomycetes have not been reported as contaminants. They are difficult to culture but must occasionally be present, judging from their frequency in the lacrimal excretory system.

Dermatophilus congolensis is an actinomycete recognized as a cause of skin infection in animals, especially ungulates, and is sometimes referred to as cutaneous streptothricosis.[1] Infection in humans is rare and limited almost entirely to those working with infected animals. One example was the pustules reported on the extremities of four people who cleaned an infected deer in New York.[3] The organism *Actinomyces israelii*, commonly found in the lacrimal excretory system, is a distant cousin. Actinomycetes have many characteristics of bacteria but are still classified as fungi for most purposes. The source of the organism in our Patient 1 was unknown. Her elderly dog had died at approximately the same time she had the contact lens problem, but canine infection with *Dermatophilus* has been disputed.[1]

Two of the previously reported cases of fungal contamination involved unsterilized contact lenses.[5,8] The type of sterilization was unknown in four cases,[7] and in five others a cold chemical technique was used.[4,6,7] In Morgan's[7] five cases involving Bausch & Lomb contact lenses, heat sterilization was carried out irregularly. Compliance with these protocols is not always adequate.[2] Our Patient 1, for example, initially claimed that she followed instructions, but later indicated that the sterilization was erratic.

In the previously reported cases, six of the contact lenses were used for cosmetic purposes[4,6,9] and the purpose in nine instances was unspecified.[7,8] Shapiro's[5] patient and our two patients wore contact

lenses because of aphakia. Sources at Bausch & Lomb report unofficially that only 6% to 7% of contact lenses dispensed are in the aphakic range (+10.00 diopters or more). Although this series is small, the 16.6% incidence of aphakic lenses in the contaminated group suggests that these thicker contact lenses are more susceptible to fungal growth, especially when they are used for extended wear.

ACKNOWLEDGMENT

William Kaplan, D.V.M., and June M. Brown of the Communicable Disease Center, Atlanta, Georgia, performed the fluorescent antibody staining.

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OPHTHALMIC MINIATURE

I was not convinced that the publishers' view was commercially sound until I got a clue to my real condition from a friend of mine, a physician who had devoted himself specially to ophthalmic surgery. He tested my eyesight one evening, and informed me that it was quite uninteresting to him because it was normal. I naturally took this to mean that it was like everybody else's; but he rejected this construction as paradoxical, and hastened to explain to me that I was an exceptional and highly fortunate person optically, normal sight conferring the power of seeing things accurately, and being enjoyed by only about ten per cent of the population, the remaining ninety per cent being abnormal. I immediately perceived the explanation of my want of success in fiction. My mind's eye, like my body's, was "normal": it saw things differently from other people's eyes, and saw them better.

Bernard Shaw, *Plays Unpleasant*
"Preface: Mainly about myself"
London, Max Reinhardt, The Bodley Head,
Reprinted 1979

ACQUIRED BLEPHAROPTOSIS SECONDARY TO CONTACT-LENS WEAR

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Five patients (eight eyelids) had acquired blepharoptosis after using contact lenses. The ages (26 to 55 years) of these patients was not consistent with idiopathic senile blepharoptosis, and we excluded all other possible causes.

All patients had disinsertion and recession of the aponeurosis of the levator palpebrae superioris muscle, and the blepharoptosis improved in each case after reattachment of the aponeurosis to the superior tarsal border.

We believe there is a cause-and-effect relationship between the use of contact lenses and the acquisition of secondary blepharoptosis. The most likely explanation of this type of blepharoptosis is difficulty in inserting and removing the lenses. Thus, excessive eyelid manipulation may disinsert the aponeurosis of the levator palpebrae superioris muscle from the tarsus.

Acquired or secondary blepharoptosis is a common ophthalmologic condition that has many causes.[1] Dehiscence or disinsertion of the aponeurosis of the levator palpebrae superioris muscle from the tarsus often explains acquired blepharoptosis.[2] The causes of levator palpebrae superioris muscle disinsertion include previous eyelid trauma, previous ocular surgery (particularly cataract surgery), previous eyelid edema, and a family history of blepharoptosis.

To our knowledge, there are no published reports describing a cause-and-effect relationship between contact lenses and blepharoptosis. We studied five patients (eight eyelids) with acquired blepharoptosis and anatomically proven disinsertion of the aponeurosis of the levator palpebrae superioris muscle that we believed to be secondary to the use of contact lenses.

SUBJECTS AND METHODS

We retrospectively studied every case of acquired blepharoptosis in the practice of one of us (A. M. P.). We included patients who had used contact lenses (hard or soft) before the onset of blepharoptosis. We excluded patients with a familial tendency to blepharoptosis and those who had suffered previous ocular trauma or undergone ocular surgery and who were later fitted with contact lenses. We also excluded patients with previous eyelid edema or significant allergic or medical histories, and those who used ocular medications, particularly topical corticosteroids, on a regular basis. Patients with neurogenic and myogenic blepharoptosis were omitted as well.

We compiled a comprehensive history for each patient. Each patient underwent several ocular examinations, including

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the evaluation of eyelid status with and without contact lenses. The essential measurements were: (1) palpebral fissure width on primary gaze (in millimeters); (2) margin-reflex distance-1 (the distance in millimeters from an ocular light reflex to the upper eyelid on primary gaze)[3]; (3) function of the levator palpebrae superioris muscle[4]; and (4) margin-crease distance and margin-fold distance.[5] The margin-crease distance is the distance in millimeters from the upper eyelid crease to the anterior eyelid margin on downgaze. The margin-fold distance is the distance in millimeters from the inferior edge of the upper eyelid skin fold to the eyelid margin on primary gaze.

Preoperatively, 2 drops of 10% phenylephrine HCl ophthalmic solution were instilled in the superior fornix of each patient. We then re-examined the status of each eyelid. If this test produced a cosmetically acceptable elevation of the upper eyelid, we chose a Müller's muscle-conjunctival resection.[6] If the test did not elevate the upper eyelid, we recommended a levator palpebrae superioris muscle aponeurosis advancement-and-tuck procedure. Four of five patients with possible myasthenia gravis underwent an edrophonium chloride test. All the patients included in this study ultimately underwent blepharoptosis surgery.

Case reports

Case 1—A 26-year-old woman had had blepharoptosis in the left upper eyelid for seven months (Fig. 1, left). She had used hard contact lenses for 15 years without unusual difficulty. She had not suffered from any ocular or medical diseases, ocular trauma, or allergies, and had not used ocular medications. She denied having diplopia, general weakness, or other ocular symptoms. She had never been pregnant and the family history was noncontributory for blepharoptosis.

The ocular examination disclosed a visual acuity of 6/7.5 (20/25) in both eyes with hard contact lenses. The palpebral fissure width was R.E.: 11 mm and L.E.: 9 mm. The margin-reflex distance-1 was R.E.: 4.5 mm and L.E.: 3.5 mm; function of the levator palpebrae superioris muscle was 15 mm in both eyes. The margin-crease distance was 10 mm in both eyes but the margin-fold distance was R.E.: 2 mm and L.E.: 6 mm. After instillation of 10% phenylephrine, the margin-reflex distance-1 was unchanged in the left eye. Results of the remainder of the ocular examination were unremarkable. An edrophonium chloride test gave negative results.

The patient underwent exploration of the left levator palpebrae superioris muscle via the external (skin) approach. The aponeurosis of the levator palpebrae superioris muscle was disinserted 10 mm superior to the superior tarsal border. The aponeurosis was then advanced and reattached to the border. This elevated the eyelid to a satisfactory level at the superior corneoscleral limbus (margin-reflex distance-1, 6 mm).

One month after surgery the palpebral fissure width on primary gaze was 11.5 mm in both eyes. The margin-reflex distance-1 was R.E.: 4.5 mm and L.E.: 5.5 mm (Fig. 1, right).

Case 2—A 55-year-old woman noted the onset of bilateral blepharoptosis after being fitted with hard contact lenses. She had not had any ocular or medical problems, diplopia, weakness, previous eyelid trauma, or allergies. She had not used ocular medications. There was no family history of blepharoptosis.

Visual acuity was 6/7.5 (20/25) in both eyes with contact lenses. The eyelids had some excessive skin. The palpebral fissure width was R.E.: 7.5 and L.E.: 9 mm. The margin-reflex distance-1 was R.E.: 2.5 mm and L.E.: 3.0 mm (Fig. 2, left). The function of the levator palpebrae superioris muscle was R.E.: 17 mm and L.E.: 18 mm. The margin-crease distance was R.E.: 16 mm and L.E.: 12 mm. Results of the remainder of the ocular examination were unre-

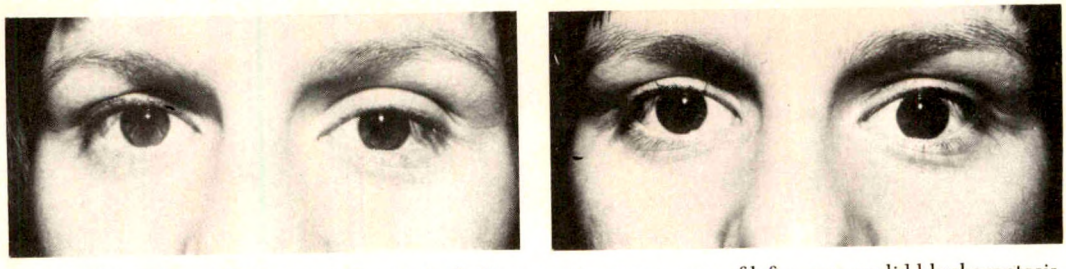


Fig. 1 (Epstein and Putterman). Case 1. Left, Preoperative appearance of left upper eyelid blepharoptosis. Right, Postoperative appearance after reattachment of disinserted left levator palpebrae superioris muscle to superior tarsal border.

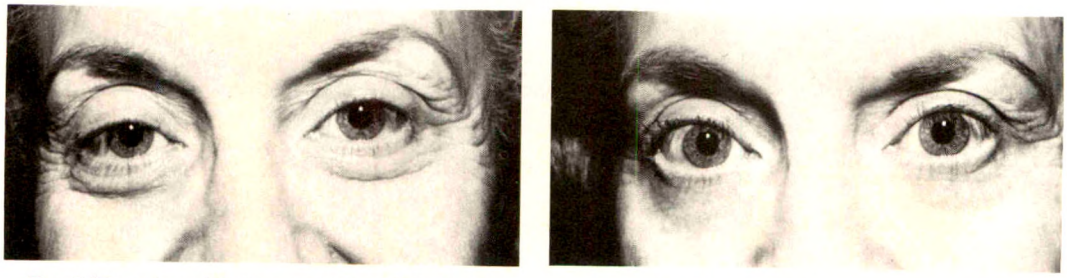


Fig. 2 (Epstein and Putterman). Case 2. Left, Preoperatively, bilateral blepharoptosis and excessive skin are present. Right, Postoperative appearance.

markable. After instillation of 10% phenylephrine, the upper eyelid became elevated and the margin-reflex distance-1 was 3.5 mm in both eyes.

Because of the slight residual blepharoptosis with phenylephrine and the thinness and high creases of both upper eyelids, both levator palpebrae superioris muscles were explored through the external approach. In each eyelid the aponeurosis of the levator palpebrae superioris muscle was disinserted approximately 10 mm above the superior tarsal border. Advancement and reattachment of the aponeurosis to the border was performed in each eyelid. A 10 × 50-mm ellipse of skin was excised from each upper eyelid.

Two months postoperatively, the palpebral fissure width on primary gaze was R.E.: 12 mm and L.E.: 11 mm. The margin-reflex distance-1 was R.E.: 6.5 mm and L.E.: 6 mm (Fig. 2, right).

Case 3—A 45-year-old woman had had blepharoptosis of the left upper eyelid for approximately five years. The onset of the blepharoptosis coincided with the use of hard contact lenses. One year later she had switched to soft contact lenses but the appearance of her eyelid had not improved. The patient did not have diplopia, weakness, other ocular symptoms or trauma, surgery, medical problems, or allergies. She denied having previous eyelid swelling or using ocular medications. A family history was noncontributory for blepharoptosis.

Visual acuity was R.E.: 6/9 (20/30) and L.E.: 6/7.5 (20/25) with spectacle correction. The palpebral fissure width was R.E.: 10 mm and L.E.: 7 mm (Fig. 3). The margin-reflex distance-1 was R.E.: 3.5 mm and L.E.: 0.5 mm. Function of the levator palpebrae superioris muscle was R.E.: 13 mm and L.E.: 12 mm. The margin-crease distance was R.E.: 6 mm and L.E.: 8.5 mm. There were bilateral epicanthal folds. On the left upper eyelid, a more prominent fold was present, and the margin-fold distance was R.E.: 2.5 mm and L.E.: 6.5 mm. Results of the remainder of the ocular examination were unremarkable. Instillation of 10% phenylephrine failed to elevate the left upper eyelid significantly. Results of an edrophonium chloride test were negative.

The patient underwent exploration of the left levator palpebrae superioris muscle. The aponeurosis was disinserted, and its inferior edge moved to 10 mm above the superior tarsal border. The aponeurosis was reattached to the border. This elevated the eyelid to the 12 o'clock position of the corneoscleral limbus, resulting in a margin-reflex distance-1 of 5.5 mm. Because the patient, who was Oriental, desired eyelids that appeared Occidental, she underwent bilateral reconstruction of the upper eyelid creases. The crease was placed 8 mm superior to the anterior eyelash line. The patient has been unavailable for postoperative follow-up.

Case 4—A 43-year-old woman noted the gradual onset of bilateral blepharoptosis that was worse in the left eye. The patient had been wearing hard contact lenses for eight years. She denied having any ocular symptoms, previous eyelid edema, ocular surgery or trauma, or using ocular medications. A family history for blepharoptosis was noncontributory.

Visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/9 (20/30) with contact lenses. The palpebral fissure width was R.E.: 6 mm and L.E.: 4 mm (Fig. 4, top left). The margin-reflex distance-1 was R.E.: 0.5 mm and L.E.: −2.0 mm. The function of the levator palpebrae superioris muscle was R.E.: 17 mm and L.E.: 12 mm. The eyelids were extremely thin and permitted the iris to be seen upon transillumination. The 10% phenylephrine test failed to elevate either eyelid. Results of an edrophonium chloride test were negative.

The levator palpebrae superioris muscles were explored bilaterally through an external approach. The aponeurosis was disinserted and moved to



Fig. 3 (Epstein and Putterman). Case 3. Preoperatively, left upper eyelid blepharoptosis is present. Note the asymmetry in margin-fold distance (R.E.: 2.5 mm and L.E.: 6.5 mm).



Fig. 4 (Epstein and Putterman). Case 4. Top left, Preoperative appearance. Top right, Photograph taken during surgery shows the disinsertion of the aponeurosis of the levator palpebrae superioris muscle from superior tarsal border. Small arrow, orbital septum; large arrow, aponeurosis of the levator palpebrae superioris muscle. Bottom left, Postoperative improvement.

20 mm above the superior tarsal border in each eyelid (Fig. 4, top right). The levator palpebrae superioris muscles were advanced and reattached to the borders bilaterally. Two months postoperatively, the palpebral fissure width was 10 mm in both eyes and the margin-reflex distance-1 was 5 mm in both eyes (Fig. 4, bottom left).

Case 5—A 50-year-old man had a one-year history of progressive bilateral blepharoptosis that limited his superior visual field. The patient had worn hard contact lenses for ten years without unusual difficulty. Medical, ocular, family, and allergic histories were noncontributory. He denied using ocular medications.

Visual acuity was R.E.: 6/6 (20/20) and L.E.: 6/7.5 (20/25) with contact lenses. The palpebral fissure width on primary gaze was R.E.: 6 mm and L.E.: 5 mm (Fig. 5, left). The margin-reflex distance-1 was R.E.: 0 mm and L.E.: −1 mm. Function of the levator palpebrae superioris muscle was 18 mm in both eyes. The margin-crease distance was R.E.: 13 mm and L.E.: 16 mm.

Both upper eyelids were thin. Results of the remainder of the ocular examination were unremarkable. Instillation of 10% phenylephrine failed to elevate the eyelids to a satisfactory level. Results of an edrophonium chloride test were negative. Superior visual field defects, probably secondary to the blepharoptosis, were found in both eyes.

The patient underwent exploration of both levator palpebrae superioris muscles. The aponeurosis was 10 mm above the superior tarsal border in both eyelids. A bilateral advancement-and-tuck procedure was performed, but the left upper eyelid was 2 mm lower than the right upper eyelid, which was placed at the 12 o'clock position of the corneoscleral limbus. Because of persistent blepharoptosis, the left upper eyelid underwent a second exploration two months later. A dehiscence was found in the left levator palpebrae superioris muscle. After this was repaired, the eyelid was placed at the superior corneoscleral limbus.

Six months after the initial surgical procedure, the palpebral fissure width was R.E.: 10 mm and L.E.: 8.5 mm (Fig. 5, right). The margin-reflex distance-1 was R.E.: 4.5 mm and L.E.: 3.5 mm.



Fig. 5 (Epstein and Putterman). Case 5. Left, Preoperative bilateral blepharoptosis; left eye is worse than the right eye. Right, Postoperative appearance after two surgical procedures (repair of disinsertion and repair of dehiscence).

Discussion

These patients ranged in age from 26 to 55 years, and were much younger than those with idiopathic senile blepharoptosis. Because medical histories and ocular examinations excluded a familial tendency and other causes of secondary blepharoptosis, we believed that contact-lens use was the primary causative factor in disinsertion of the aponeurosis of the levator palpebrae superioris muscles, leading to secondary blepharoptosis.

The time between the initial wearing of contact lenses and the development of blepharoptosis varied among the patients in this series. Three patients (five eyelids) had worn contact lenses for at least eight years before noting blepharoptosis. Two patients (three eyelids) recalled that the blepharoptosis had begun with the initial contact-lens fitting. All the patients in this study had worn hard contact lenses, although one patient (Case 3) had switched to soft lenses after discovering the blepharoptosis.

The findings for all patients were similar with and without contact lenses. When contact lenses are improperly fitted or worn too much, ocular irritation may cause a reflex blepharospasm, giving the patient a blepharoptotic appearance. It is important to examine the patient with blepharoptosis secondary to contact-lens wear with and without contact lenses.

All our patients underwent exploration of the levator palpebrae superioris muscle in the affected eyelid, and we found a true disinsertion of the aponeurosis in each case. The levator palpebrae superioris muscle was disinserted 10 to 20 mm above the superior tarsal border. In one patient (Case 5) persistent blepharoptosis was caused by an overlooked dehiscence in the aponeurosis of the levator palpebrae superioris muscle. When the aponeurosis was reattached or the dehiscence repaired, an immediate improvement in the eyelid level was apparent. The postoperative results confirmed that disinsertion or dehiscence of the levator palpebrae superioris muscle was the true anatomic explanation for the blepharoptosis in these cases.

The most likely explanation of blepharoptosis induced by contact lenses is difficulty in inserting and removing the lenses, which can cause excessive manipulation and rubbing of the eyelids. These mechanical factors may traumatize the eyelids, resulting in disinsertion of the aponeurosis of the levator palpebrae superioris muscle. Our patients had not used eyedrops, the instillation of which may cause excessive manipulation of the eyelids in some patients.

A less likely explanation is a toxic effect of the lens or lens solution, causing a hypersensitivity or allergic eyelid edema. This edema may then lead to secondary disinsertion of the aponeurosis of the levator palpebrae superioris muscle and subsequent blepharoptosis.[2] This was not true in our patients.

Acquired blepharoptosis is a frequent sequela of ocular surgery.[1] Most often this is caused by lens extraction; the current hypothesis is that the blepharoptosis is caused by postoperative eyelid edema, trauma to the levator palpebrae superioris muscle or nerve, or perhaps the opposing mechanical factors of the superior rectus muscle suture and the eyelid speculum.[2]

Cataract patients are often fitted with contact lenses after lens extraction. These patients may develop blepharoptosis months to years after surgery, and even long after the contact-lens fitting. We excluded these patients from this study to avoid confusion about the origin of the blepharoptosis. Contact-lens use may be the precipitating factor in blepharoptosis

in these patients, although this is difficult to prove.

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Ophthalmic miniature

Jadine nodded. It seemed like a perfect exit line to her, since she didn't know what he was talking about and didn't want to pursue his thoughts if they were anything like his eyes at this moment. Without melanin, they were all reflection, like mirrors, chamber after chamber, corridor after corridor of mirrors, each one taking its shape from the other and giving it back as its own until the final effect was color where no color existed at all.

Toni Morrison, *Tar Baby*
New York, Alfred A. Knopf, 1981

PLATELET-INDUCED RETINAL NEOVASCULARIZATION IN LEUKEMIA

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Retinal changes resembling those of background diabetic or hypertensive retinopathy commonly occur in leukemia, whereas retinal neovascularization is rare. When neovascularization does occur, it is usually the result of hyperviscosity caused by a greatly increased number of circulating leukocytes. A 42-year-old woman with diabetes mellitus developed chronic myelocytic leukemia and peripheral retinal neovascularization. The hyperviscosity leading to the neovascularization was probably caused by an increased number of circulating platelets.

Conjunctival and retinal hemorrhages unassociated with cellular infiltration are the most common ophthalmic findings in leukemia.[1,2] The ocular manifestations may be caused primarily by either hypercellularity in the blood or changes in the plasma proteins. Hemorrhage may also occur in the episclera, sclera, choroid, vitreous, and anterior and posterior chambers. Leukemic cellular infiltration generally appears near blood vessels but may occur in the conjunctiva, episclera, sclera, choroid, iris, ciliary body, optic nerve, and retina, and only rarely extends into the vitreous. Serous retinal detachment, papilledema, and retinal pigment epithelial hypopigmentation and hyperpigmentation have also been described.[3]

Leukemic retinopathy may resemble background diabetic or hypertensive retinopathy. Deep and superficial retinal hemorrhages and cotton-wool spots are often present. There may be venous and, less commonly, arteriolar dilation, vessel tortuosity, caliber irregularity, sheathing, and microaneurysm formation with surrounding retinal edema and hard yellow exudates.[1]

Proliferative leukemic retinopathy has been reported in three patients with chronic myelocytic leukemia.[4-6] The appearances of the new blood vessels in each case were similar to those seen in the sickle-cell hemoglobinopathies which have a seafan configuration. In each case, increased blood viscosity, caused by leukocyte counts between 250,000 and 600,000 cells/mm^3, resulted in vaso-occlusion that led to peripheral neovascularization. We report a fourth case of chronic myelocytic leukemia with peripheral fan-shaped retinal neovascularization in a patient who also had diabetes mellitus. However, the peripheral retinal vaso-occlusion and neovascularization were the result of a markedly increased number of blood platelets and not an increased leukocyte count.

CASE REPORT

A 42-year-old woman was first seen here in February 1977. At that time a diagnosis of chronic myelocytic leukemia with thrombocytosis was made. She had been treated with radioactive iodine (^{131}I) for

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Graves' disease in 1965 and she had required insulin for her diabetes since 1967.

Her initial complete blood cell count in February 1977 gave the following values: leukocytes, 33,700/mm^3 (normal, 3,500 to 11,000/mm^3); erythrocytes, 3.76 million/mm^3 (normal, 3.9 to 5.6 million/mm^3); and platelets, 988,000/mm^3 (normal, 150,000 to 450,000/mm^3). Monthly tests since that time have given leukocytic counts ranging from 10,000 to 37,600/mm^3 and platelet counts from 281,000 to 878,000 cells/mm^3, with platelet counts of more than 600,000 cells/mm^3 on 15 occasions. Her prothrombin time, and serum globulins, fibrinogin, hemoglobin electrophoresis, and serum viscosity values were within the normal range. Exacerbations of her leukemia, shown by increased platelet and leukocyte counts, were successfully treated with busulfan.

When she was first examined in our eye clinic in August 1977, her corrected visual acuity was R.E.: 6/7.5 (20/25) and L.E. 6/12 (20/40). She had leaking microaneurysms, retinal edema, and hard yellow exudates in both posterior poles. Tensions by applanation tonometry were 16 mm Hg bilaterally and Hertel exophthalmometry was 25 mm in both eyes with a base of 95 mm.

In September 1978 and March 1979 she received argon-laser photocoagulation to control the background diabetic retinopathy, which had reduced her visual acuity to R.E.: 6/12 (20/40) and L.E.: 6/30 (20/100). In December 1979, the left eye had superior perihperal fan-shaped areas of neovascularization with avascular zones anterior to these. Each area of neovascularization was from 1 to 3 disk diameters across, and was enmeshed in fibrous tissue (Figs. 1 and 2).

Discussion

New retinal blood vessels grow from existing retinal vessels, possibly stimulated by a vasogenic substance produced by adjacent areas of hypoxic retina.[7] In some cases of diabetes mellitus, platelet-fibrin thrombi may occlude retinal capillaries in the posterior pole or along the major vascular arcades, creating zones of capillary nonperfusion. Retiform networks of new vessels may then develop along the margins of the nonperfused retinal areas.

Neovascularization in peripheral retinal vaso-occlusive diseases differs from that in diabetes mellitus in location and configuration. Occlusion of a peripheral retinal blood vessel creates a zone of nonperfusion or avascularity distal to the site of the occlusion. Anastomoses develop between the occluded arteriole and

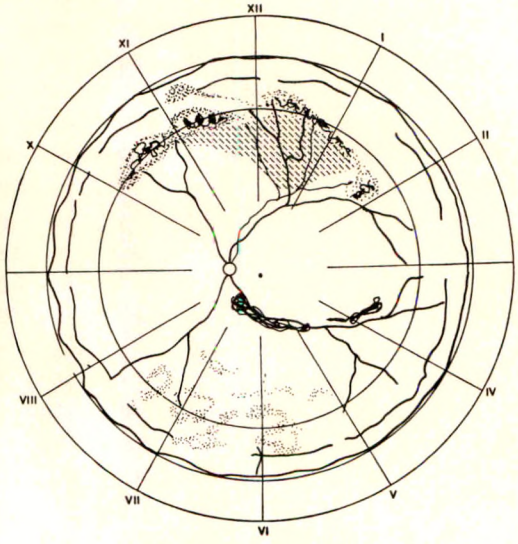


Fig. 1 (Leveille and Morse). Left eye shows many areas of fan-shaped fibrovascular tissue near the equator superiorly. Posterior to this there is a shallow tractional retinal detachment (broken lines). There is preretinal hemorrhage along the inferotemporal arcades and vitreous hemorrhage inferiorly (black dots).



Fig. 2 (Leveille and Morse). One area of superior peripheral neovascularization near the equator in the left eye. The new blood vessels are enmeshed in fibrous tissue.

nearby veins. The fan-shaped fibrovascular network grows along these shunts, sometimes extending toward the peripheral avascular zone. This seafan configuration of neovascularization is a tissue response of the peripheral retinal blood vessels to vaso-occlusion and hypoxia and is not pathognomonic of any one disease. Peripheral retinal neovascularization may be found in any disease causing occlusion of the peripheral retinal vessels, including the sickle-cell hemoglobinopathies,[8] Eales' disease,[6,9] sarcoidosis,[10] pars planitis,[11] and long-standing retinal detachment,[12] but, to our knowledge, has not been reported in diabetes mellitus.

Proliferative leukemic retinopathy resembles the peripheral fan-shaped neovascularization in the above-mentioned diseases rather than the more posterior retiform neovascularization of diabetes mellitus, although optic disk neovascularization has been reported in one patient with leukemia.[6] The three previously reported cases[4-6] of proliferative leukemic retinopathy were similar to our case in that the patients showed small and large areas of peripheral retinal fibrovascular tissue at or just posterior to the equator. This was bilateral in two cases; the fellow eye of the third case was lost to massive hemorrhage. The average size of the neovascular areas was 2 to 3 disk diameters, although some areas were as large as 5 disk diameters. Anterior to the new blood vessel formation, the terminal retinal vessels appeared to be sheathed and most were obliterated; this was confirmed by fluorescein angiography.

Although the hallmark of leukemia is a leukocytosis of malignant leukocytes, often with a decreased number of normal leukocytes, erythrocytes, and platelets, increased numbers of basophils, eosinophils, and platelets[13] may be present in the peripheral blood and contribute to increased blood viscosity. In addition to the number of cells, cell rigidity and serum protein also determine whole-blood viscosity.[6] Among the major plasma proteins, fibrinogin has the most pronounced effect on promoting the aggregation of cells, especially erythrocytes. Increased globulin levels may also promote cell aggregation. Decreased erythrocyte flexibility, as in the sickle-cell hemoglobinopathies, spherocytosis, pyruvate kinase deficiency, and burr cell formation in hepatic cirrhosis, causes abnormal blood flow in the microcirculation.[14] In leukemia, increased whole-blood viscosity and the associated reduced blood flow may be caused by increased globulin levels or increased levels of any cellular element, most commonly malignant leukocytes.[15] In our patient, plasma fibrinogin and globulin levels were normal, and the leukocyte count was never greater than 37,600 cells/mm^3, whereas the platelet count was between 281,000 and 988,000 cells/mm^3. Although leukocyte and platelet counts were often several times the normal values, the platelets contributed more to the absolute number of cells in the peripheral blood, and we believe that this was the major cause of the hyperviscosity that led to the peripheral retinal vaso-occlusion and subsequent neovascularization.

Although platelets may be increased in number in leukemia, qualitative defects usually do not occur. This is unlike diabetes mellitus, in which increased platelet adhesiveness and aggregation caused by a heat-soluble, nondialyzable plasma factor may occur.[7] Platelet abnormalities may lead to a microcirculatory sludging and focal ischemia with subsequent neovascularization.

Because peripheral retinal neovascularization of the type present in our patient and other patients with leukemia has not been reported in diabetes mellitus, we believe that in our patient the leukemia with the associated increased number of platelets was the cause of the

peripheral vaso-occlusion and neovascularization. However, it is also possible that qualitative platelet defects associated with diabetes mellitus may have aided in the development of the neovascularization.

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Ophthalmic Miniature

. . . in the Oriental countries . . . every separate and individual devil of them's got the ophthalmia! It's as natural to them as noses are, —and sin. It's born with them, it stays with them, it's all that some of them have left when they die.

Mark Twain, Gilded Age, Chapter VIII, Vol. 1

ORBITAL TUBERCULOSIS

THOMAS C. SPOOR, M.D., AND SALLY A. HARDING, M.D.
Columbia, South Carolina

A 37-year-old man had epistaxis, headache, intermittent swelling of the periorbital region, proptosis, epiphora, and chronic sinusitis. X-ray films showed marked erosion of the midline facial bones, total opacification of both maxillary sinuses, and clouding of the sphenoid and left ethmoid sinuses. He also had a cutaneous-maxillary sinus fistula and a purulent discharge. A histopathologic examination disclosed areas of acute and chronic inflammation with occasional noncaseating granulomas.

Biopsy specimens and postoperative sputum cultures grew *Mycobacterium tuberculosis*, although the patient had no obvious signs of systemic disease. Because tuberculosis of the orbit is rare in developed countries, it is often overlooked in the diagnosis of granulomatous inflammation of the orbit. This can lead to exacerbation of the disease by corticosteroid treatment.

Proven orbital tuberculosis is rare in developed countries.[1] The majority of cases are reported from underdeveloped nations with a high incidence of systemic tuberculosis.[2-4] Orbital involvement may result from hematogenous spread from distant foci[3] or may be secondary to extension from the paranasal sinuses.[4] We studied a case that demonstrated the difficulties encountered in diagnosing this rare entity in the United States.

CASE REPORT

A 37-year-old man came to the emergency room with epistaxis and headache. The medical history, although vague, disclosed intermittent swelling of the periorbital region for more than one year and two to three months of left-sided proptosis, epiphora, and chronic sinusitis.

The initial examination disclosed left-sided proptosis, midline facial deformities, a right-sided cutaneous-maxillary sinus fistula, purulent discharge, and congenital anomalies of both hands. X-ray films of the skull and sinuses disclosed marked erosion of the midline facial bones with total opacification of both maxillary sinuses and clouding of the sphenoid and left ethmoid sinuses. Exploration of the maxillary antra via bilateral Caldwell-Luc incisions showed abundant granulation tissue which was subtotally curetted. Histopathologic examination disclosed areas of acute and chronic inflammation with occasional noncaseating granulomas. Bacterial cultures were negative. Kinyoun's stain and Grocott's stain on the tissue sections were negative for mycobacteria and fungi, respectively. The patient was treated with a two-week course of systemic cephalosporins for suspected osteomyelitis of the maxillary sinus, and an ophthalmologic consultation was requested for evaluation of epiphora and proptosis.

The ocular examination disclosed a visual acuity of 6/6 (20/20) in both eyes; color vision was normal. There was 6 mm of proptosis (Hertel) on the left side and increased orbital resistance to retropulsation. Extraocular motility and intraocular pressure were normal, as were the results of slit-lamp and ophthalmoscopic examinations. The right lacrimal excretory system was totally obstructed. A right-sided cutaneous maxillary fistula was present.

A complete blood cell count was normal, the VDRL test unreactive, the sedimentation rate increased, and the chest X-ray film normal. A computed tomographic scan showed a large, homogenous mass extending into the left orbit from the ethmoid sinus with erosion of the medial orbital wall (Fig. 1). Exploration of the left orbit showed erosion of the

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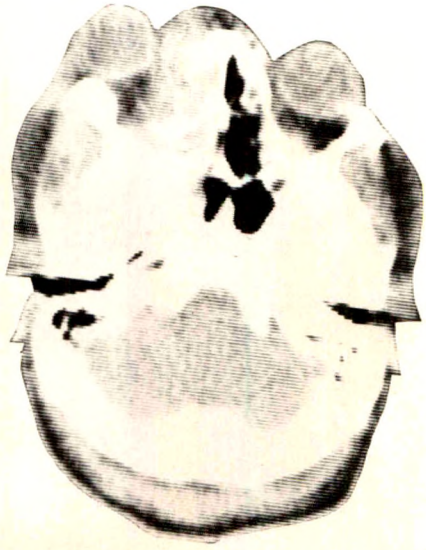


Fig. 1 (Spoor and Harding). Computed tomographic scan shows mass invading medial left orbit from adjacent paranasal sinus.

medial orbital wall by granulation tissue and necrotic material. Histopathologic examination (Figs. 2 and 3) disclosed prominent necrotizing granulomas with vascular involvement in some areas. There was no evidence of caseation. Large areas of necrotic debris were present. Kinyoun's stain for mycobacteria and Grocott's stain for fungi were negative. Histopathologic examination of the biopsy specimen from the lacrimal gland disclosed scattered areas of chronic inflammation with no granulomas present. Because of the clinical appearance of the mass at surgery and the presence of granulomatous inflammation, the chest X-ray films were reviewed and repeated. Re-examination disclosed subtle, diffuse micronodular calcifications and a left-sided apical lesion. Tomograms of the left upper lobe showed an apical density with questionable cavitation. An intermediate-strength purified protein derivative was applied and was markedly positive. Subsequently, cultures of the biopsy specimen and postoperative sputum cultures grew *Mycobacterium tuberculosis* on Lowenstein-Jensen and 7-H10 media. The patient had previously begun taking isoniazide and ethambutol and tolerated the two-drug regimen well.

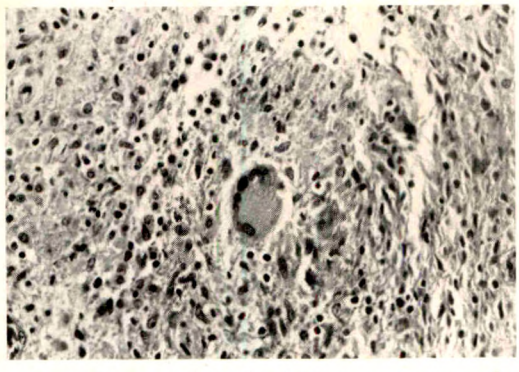


Fig. 3 (Spoor and Harding). Noncaseating granuloma with a typical Langhan's giant cell (hematoxylin and eosin, ×500).

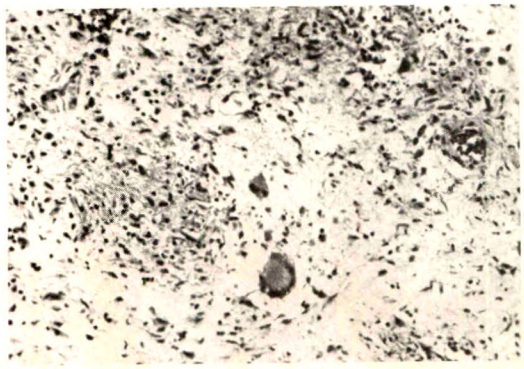


Fig. 2 (Spoor and Harding). Tissue removed from medial orbital wall shows necrotizing granulomatous inflammation within edematous soft tissue (hematoxylin and eosin, ×315).

Discussion

Our patient had painless proptosis, fistulas, and epistaxis with radiographic evidence of pansinusitis and extensive bony destruction. The referring physicians suspected a neoplasm and performed bilateral maxillary antrostomies to obtain a histopathologic diagnosis. Lymphomas, primary squamous cell carcinoma, and adenocarcinoma of the paranasal sinuses may be clinically similar. They found no evidence of malignancy, only acute and chronic inflammation with occasional giant cells. They diagnosed chronic maxillary osteomyelitis.

Maxillary osteomyelitis is primarily a disease of infancy; the symptoms include extensive suppuration, bony necrosis, and clinical evidence of acute inflammatory process.[5] In adults, maxillary osteomyelitis may be a sequela of acute sinusitis and may be accompanied by orbital cellulitis or cavernous sinus thrombosis.[6] A patient with this condition would be

severely ill and the diagnosis quite evident.

This prolonged course of painless proptosis accompanied the formation of fistulas and paranasal sinusitis with bony destruction is compatible with aspergillosis originating in the paranasal sinuses. Histopathologically, aspergillosis is manifested by a chronic granulomatous process with lymphocytes, epithelioid cells, histocytes, and foreign-body giant cells. Septate hyphae are recognizable in the cytoplasm of the giant cells and the organism can be grown on Sabouraud's agar. Cultures and stains from our patient were consistently negative for fungi.

Mucormycosis of the orbit has a much more dramatic clinical pattern, including painful proptosis, ophthalmolplegia, and visual loss in a patient with an underlying disease, usually diabetic ketoacidosis. Organisms are usually evident in biopsy specimens and grown easily on Sabouraud's agar. This diagnosis was not tenable in our patients.

Actinomycosis can also affect the orbit by spreading from adjacent tissues. This condition is manifested by gradually progressive, painless proptosis and bony erosion. Subcutaneous infiltration and nodules may eventually result in formation of fistulas. Vision is usually preserved and this can be helpful diagnostically. Chronic granulomatous inflammation with epithelioid cells and giant cells may be present. The organisms may be seen in clusters, as granules surrounded by an inflammatory infiltrate, or in the discharge.[5]

Rarely, the orbit can be affected by tertiary syphilis, either as a periostitis or secondary to a gummatous process in adjacent structures, most commonly the paranasal sinuses. Such infections are manifested by proptosis, ophthalmoplegia, and trigeminal neuralgia. This last symptom is an important means of distinguishing this condition from tuberculosis which is painless. The diagnosis can be confirmed serologically.

Lethal midline granuloma may produce radiographic evidence of pansinusitis and bony destruction with orbital involvement. Necrotizing granulomas and vasculitis may be prominent. Jakobiec and Jones[7] characterized four categories of lethal midline granuloma: (1) lymphomas of the central facial bones of a recognizable histologic type; (2) atypical lymphoma; (3) destructive inflammatory lesions such as syphilis and fungal infections; and (4) lesions such as Wegener's granulomatosis beginning in the sinuses and later generalizing to include characteristic pulmonary and renal involvement.

The chest X-ray film findings in Wegener's granulomatosis are characteristic; they consist of many bilateral descrete infiltrates, predominantly in the lower lobes of the lungs, with thin-walled cavities more than 1 cm in diameter.[7] These X-ray findings were lacking in our patient, as was renal involvement.

As the incidence of tuberculosis decreased, so did the general awareness of the disease.[8] A recent review indicated that the spectrum of this disease has changed, with extrapulmonary disease representing an increasing proportion of all cases.[9]

This case illustrated the difficulties encountered in diagnosing orbital tuberculosis in the United States. Infection of the orbit and paranasal sinuses by *M. tuberculosis* is rare and seldom reported in Western journals.[3-5] A recent textbook on orbital disease described orbital tuberculosis as a curiosity occurring in underdeveloped countries, usually without evidence of systemic disease (lymphadenopathy, pulmonary disease, or hepatosplenomegaly).[1]

Orbital involvement may be secondary to hematogenous spread from the primary complex with resultant periostitis of

the orbital margin or tuberculoma of the orbital tissue.[3] Periostitis is a more common manifestation[2,10] and in children usually shows the signs of a systemic tuberculosis infection. Orbital involvement may also be secondary to direct extension from adjacent structures (such as the lacrimal gland or paranasal sinuses). Tuberculous dacryoadenitis is also rare; it may occur by hematogenous spreading from other foci, often without signs of active systemic disease.[3,11] Tuberculosis of the paranasal sinuses is another rare manifestation not mentioned in the standard otolaryngologic textbooks. Jain, Chundawat, and Batra[4] described 43 cases of tuberculosis of the maxillary antrum; these consisted of two types of tuberculous lesions. Sometimes the infection is limited to the antral mucosa. More commonly, the lesion has a tendency to spread, causing bony erosion, formation of fistulas, and caseation, as it did in our patient. Both types of infection tend to be asymptomatic until the lesion is far advanced.

Agrawal, Jitendra, and Jaim[2] recently reviewed 14 cases of orbital tuberculosis. Children and young adults were primarily involved; all but two patients were less than 25 years of age (86%). Most patients had an active systemic disease or evidence of an inactive tuberculosis infection. A primary tuberculous complex was present on chest X-ray films in 75%. Intermediate tuberculin skin tests were positive in 85%. Nonhealing fistulas, either cutaneosinus or cutaneo-orbital, were present in 75%. X-ray evidence for erosion of periorbital bone was present in 78% of the patients.

The clinical signs of orbital tuberculosis described above were all present in our patient. We believe that proptosis, when accompanied by cutaneous fistulas and X-ray evidence of bony erosion, should prompt a review of the chest X-ray film, along with appropriate apical views and tomograms, and application of the tuberculin skin test. Special stains for acid-fast bacilli were negative despite the presence of extensive granulomatous inflammation in our patient. Cultures from the biopsy specimen were eventually positive for *M. tuberculosis*, as were sputum cultures obtained later. Failure to suspect tuberculosis as a causative agent of granulomatous inflammation of the orbit may lead to exacerbation of the infection by treatment with corticosteroids.

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OSTEOBLASTIC PROSTATE CARCINOMA METASTATIC TO THE ORBIT

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A 77-year-old man had subperiosteal osteoblastic adenocarcinoma of the orbit, metastatic from the prostate. The patient underwent orbital exploration and biopsy of the bony tumor. Despite incomplete removal of the tumor, visual acuity in the eye improved and he has remained in reasonably good health.

Formation of new bone in the orbit is rare in adults. Intraocular bone formation, however, is much more common and usually is the result of benign metaplasia of degenerating ocular tissues such as pigment epithelium, lens epithelium, and, on occasion, mesodermal fibrous tissue. Osseous choristomas[1,2] occur early in life and are usually located in the episcleral region or in the eyelids. Primary osteomas have been seen in the choroid.[3] Neoplastic bone formation may occur in the orbit in fibrous dysplasia,[4] in neurofibromatosis,[5] in the form of primary osteomas,[4,6,7] and in association with osteogenic or osteoblastic sarcomas.[4,6,7] Osteosarcoma with osteoid formation is seen as a primary tumor, but more often it is the result of an underlying bone disease like fibrous dysplasia or Paget's disease. It also occurs as a late effect of irradiation, occurring after retinoblastoma, for example.

The growth of new bone in an adult and the ability of epithelial tissues to cause bone formation through a metaplastic change are fascinating. We studied what we believe to be the first clinically and pathologically proven occurrence of unsuspected metastatic osteoblastic carcinoma of the prostate gland in the orbit.

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CASE REPORT

We first examined this 77-year-old man on Sept. 6, 1978. His family history was noncontributory. The patient's own medical history included extensive abdominal surgery for adenocarcinoma of the sigmoid or rectum in 1973. The patient had undergone a colostomy and appeared to be cured of his cancer. In 1973 he had had a transurethral resection of the prostate; all tumor tissue that had been removed was considered benign. In the summer of 1978 the patient had broken his spine during his work as a driver of a wrecker. This injury had healed by the time the patient came to us because of slowly increasing proptosis of his right eye associated with loss of vision.

At the first examination, the patient's best corrected visual acuity was R.E.: 6/60+1 (20/200+1) and J17 and L.E.: 6/7.5 (20/25) and J0. The examination disclosed distinct exophthalmos of the right eye. Exophthalmometer readings were R.E.: 23 mm and L.E.: 19 mm, with 102 mm as the basis. The right eye did not move up well and the patient suffered from constant diplopia. He kept his right eye closed. Swelling and redness of the right eyelids were associated with the exophthalmos. No tumor could be palpated in the right orbit or in the head region. The pupils reacted promptly, but there was a clinically obvious Marcus Gunn pupil in the right eye. Applanation tonometry values were R.E.: 14 mm Hg and L.E.: 16 mm Hg. Ophthalmoscopic examination of the right eye disclosed advanced papilledema with cotton-wool spots around the optic disk.

The patient thus exhibited evidence of a growing orbital tumor associated with papilledema in the right eye. His history of cancer of the colon and the more recent fracture of the spine led us to suspect a connection between these events and his orbital problem. We recommended that he be hospitalized and he was admitted on Oct. 5, 1978.

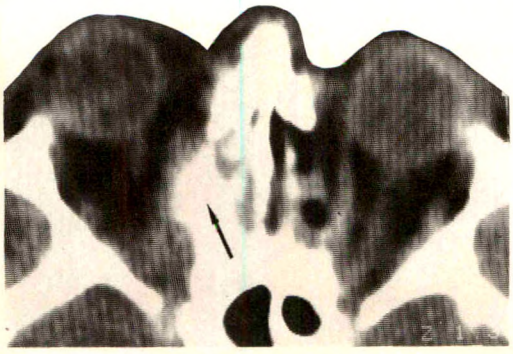


Fig. 1 (Wolter and Hendrix). Computed tomography at the level of the optic foramina shows a bony orbital tumor continuous with the right medial orbital wall (arrow).

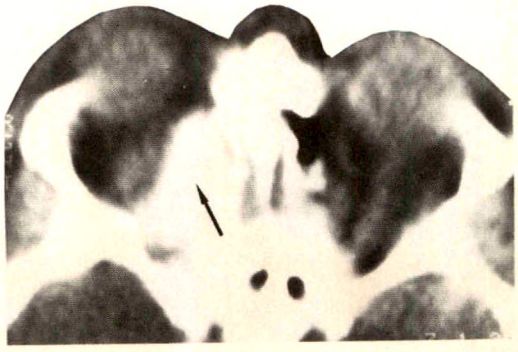


Fig. 2 (Wolter and Hendrix). Computed tomographic view superior to the optic foramina shows the massive bony tumor in the medial aspect of the right orbit (arrow).

Results of a general examination, including routine chest X-rays, were normal and there was no other obvious evidence of an active neoplasm or another serious disease. X-rays and computed tomography of the orbit, however, disclosed a sharply limited bony tumor on the inner aspect of the medial wall of the right orbit (Figs. 1 and 2). X-rays of the spine were not taken. We advised the patient to undergo surgical exploration of the right orbit so that we could make a diagnosis and relieve the choked optic disk.

The orbital exploration was done with the patient under general anesthesia on Oct. 6, 1978. We made an incision on the nasal aspect of the upper orbital rim, extending it down to the medial canthal ligament. The periosteum was cut and elevated. We found a tumor of solid bone protruding back into the orbit along the medial orbital wall in the subperiosteal space. This could not be cut with a knife. We removed three pieces of this bony mass, measuring $14 \times 8 \times 7$ mm, $12 \times 6 \times 4$ mm, and $11 \times 6 \times 5$ mm, with a hammer and chisel. These pieces formed most of the main tumor, but part of the tumor was left in place because it was too close to the optic canal to be removed safely. Frozen sections could not be done since the tumor was bony and required decalcification before it could be sectioned.

There were no complications postoperatively and visual acuity had improved to 6/22 (20/70) in the operated upon eye by Oct. 9. The papilledema was also much improved. The exophthalmos and diplopia had disappeared.

Histopathologic study, after decalcification, disclosed extensive osteoid formation of a regular pattern and arrangement all through the excised orbital tumor (Figs. 3 and 4). Many osteoblastic cells lined the surfaces of the bone (Fig. 4), but there were no multinucleated osteoclasts. Connective tissue, blood vessels, and islands of carcinoma that resembled glands filled the interstices (Fig. 4). There was some chronic inflammatory reaction in some areas near the islands of carcinoma.

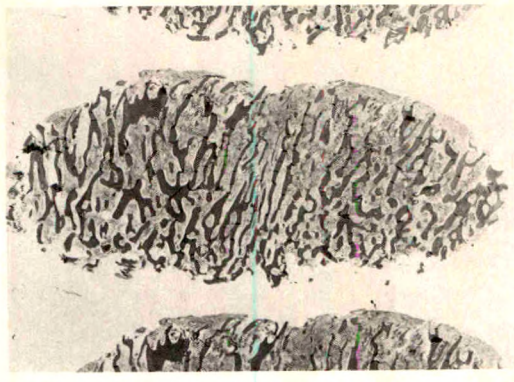


Fig. 3 (Wolter and Hendrix). Low-power view of one of the pieces of bony orbital tumor shows its regular structure (hematoxylin and eosin, ×30).

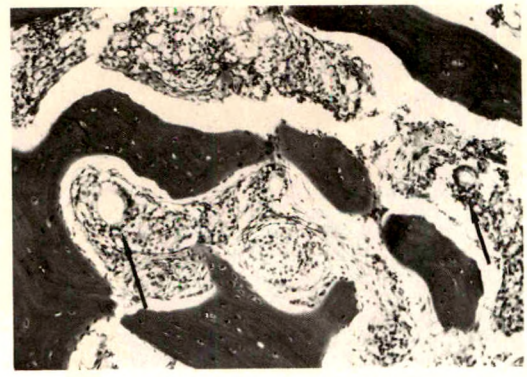


Fig. 4 (Wolter and Hendrix). High-power view of the epithelial formations (arrows) resembling glands and representing the metastatic carcinoma in the new-formed bone. Osteoblasts lining the bony structures indicate active bone formation, but no osteoclasts are present (hematoxylin and eosin, ×250).

Prostatic carcinoma is the most common cause of this type of osteoblastic metastatic involvement in general, but, to the best of our knowledge, it has not been reported previously in the orbit. It would have been even more unusual had this bony metastasis resulted from cancer of the colon. The patient underwent a second transurethral biopsy of the prostate on Nov. 15, 1978. The histopathologic diagnosis was infiltrating and moderately well differentiated adenocarcinoma of the prostate (Fig. 5). We considered this the source of the osteoblastic metastasis. Treatment with radiation and hormones was started. Further examination disclosed additional metastatic involvement of the spine, explaining the earlier fracture. However, the patient was still alive and doing well when he was last examined in July 1980. There was no obvious exophthalmos and visual acuity in the operated upon eye was unchanged.

Discussion

Metastatic neoplasms of the orbit are not as rare as we once believed,[8] but, if one excludes orbital involvement in generalized lymphomas and leukemias, they certainly are not common. The nonepithelial metastatic tumors found in the orbit include neuroblastomas, sarcomas, and melanomas. Adenocarcinomas are believed to be the most common epithelial tumors and they originate, in order of frequency, in the breast, lung, prostate, stomach, thyroid, kidney, and adrenal glands.[4] Ferry and Font[9,10] diagnosed metastatic carcinoma of the prostate in one of 28 cases of metastatic orbital carcinoma.

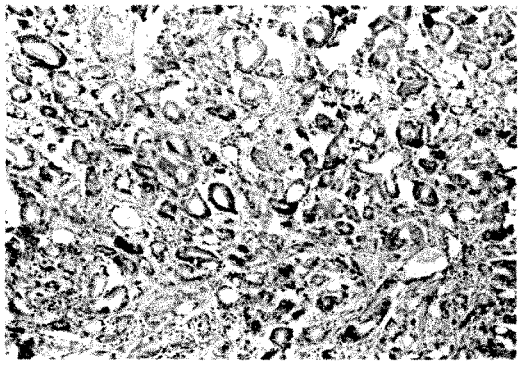


Fig. 5 (Wolter and Hendrix). Histologic section of the primary carcinoma of the prostate from tissue removed by transurethral resection on Nov. 15, 1978. Atypical gland-like formations resembling those of the orbital tumor are present (hematoxylin and eosin, ×250).

Rose[11] lists five of 51 cases of metastatic orbital tumors as prostate carcinoma. Bard and Schulze[12] in 1964 reported two cases and reviewed nine previous reports of metastatic prostate carcinoma in the orbit. All were located in or on the orbital bone, but the tumors were not osteoblastic. Attachment to the orbital bones is not limited to orbital carcinoma metastases alone, but has been observed in conditions such as orbital sarcoidosis.

Adenocarcinoma of the prostate gland typically is a primary neoplasm with a slow growth rate that initially produces no symptoms. This neoplasm has a tendency to spread to bones and often causes the first clear clinical symptoms by a spontaneous fracture of pelvis or vertebrae. About 70% of cases of prostate carcinoma result in osseous metastases; only 1% of these are subperiosteal and osteoblastic in nature.[13] Most general bony metastases of prostate carcinoma cause destruction of bone. The osteoblastic orbital metastasis in our case, however, developed on the surface of orbital bone and did not cause destruction or obvious weakening of the original bone. Thus, it did not fracture when the newformed bone was chiseled off.

The osteoblastic responses of tissues in general and of ocular and orbital tissues in particular are interesting. Bone formation in the eye most commonly occurs in the regions of the lens (cataracta ossea) and the pigment epithelium. There is no definite evidence that ossification can result from metaplasia of epithelial cells. Bone formation in degenerating epithelial tissues, as well as in carcinomas, is believed to be induced by epithelial elements, but it originates in primitive mesodermal cells of the stroma. These cells develop into osteoblasts and produce bone. Other carcinomas have the ability to induce the formation of multinucleated osteoclasts and thus cause osteolysis. Examples of neoplasms known to

have osteoblastic ability are prostate carcinoma, Hodgkin's disease, malignant carcinoid of the gut, and breast carcinoma. An osteolytic response is found most often in carcinomas of thyroid and lower bowel. However, our case showed once again that osteoblastic and osteolytic responses can be mixed and that both can appear in the same patient or, as shown by other cases, even in the same metastasis.[14]

The subperiosteal approach used in the surgical exploration of this case was ideal for removing as much of the neoplasm as possible while doing the least possible damage to the orbital bone and to the orbital contents. The bony nature of the orbital metastasis made it impossible to apply the two most helpful steps in the aggressive evaluation of an orbital tumor of unknown nature: a preoperative needle biopsy and a biopsy for frozen sections during surgery as soon as the most accessible part of the tumor was exposed. After the diagnosis was made postoperatively from decalcified histologic sections, radiation and hormonal therapy were begun. There was no need for additional surgery in the orbital region. Our patient had two types of cancer. After being cured of one that had led to a colostomy, he developed a second cancer that caused an osteoclastic metastasis in the spine and an osteoblastic mass in the orbit. The development of two different types of adenocarcinoma in this patient within the space of ten years may indicate some failure of the immune surveillance system.

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DYSKINETIC STRABISMUS AS A SIGN OF CEREBRAL PALSY

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We found dyskinetic strabismus in 66 patients with cerebral palsy. The most striking feature of dyskinetic strabismus is the fluctuation from esotropia to exotropia under the same accommodative conditions with a slow tonic deviation similar to a vergence movement. With increasing age, exodeviation becomes more prevalent. Dyskinetic strabismus is seen exclusively in cerebral palsy patients. Many of these patients have an athetoid component to their disorder. The association of dyskinetic strabismus with athetosis and upward gaze palsy suggests that the basal ganglia may be the site of the malfunction. The strabismus responds poorly to surgery and the associated athetosis is important in the diagnosis and treatment of cerebral palsy.

Strabismus patterns in patients with cerebral palsy are similar to those encountered in the general population but more common.[1-21] We studied a type of variable strabismus that occurs only in individuals with cerebral palsy. This form of strabismus, known as dyskinetic strabismus, requires treatment different from that used in other forms of strabismus. Although Seaber and Chandler[2] identified it as a significant component of cerebral palsy in 1975, it has not been studied in detail. This variable strabismus can actually precede other clinical signs of cerebral palsy and its identification can facilitate earlier referral and treatment.

SUBJECTS AND METHODS

During the past ten years, 732 children with cerebral palsy underwent neurologic, orthopedic, ophthalmologic, and orthoptic testing at the Lenox Baker Cerebral Palsy Hospital in Durham, North Carolina. The ophthalmologic tests were performed by an ophthalmologist and an orthoptist (J. H. S.). The patients underwent visual acuity testing, cycloplegic refraction (with cyclopentolate 1%, cyclopentolate 2%, or atropine 1%), ocular motility assessment, neuro-ophthalmologic, and external and ophthalmoscopic examinations whenever possible. Of the 732 children, 215 (30%) had strabismus (Table 1). Of those with strabismus, 66 (30%) had a type of strabismus that did not fit any of the conventional patterns (dyskinetic strabismus). In these patients the strabismus varied in amount and type, with the fluctuations from esotropia to exotropia apparently unrelated to accommodative effort or attention.

When a child with dyskinetic strabismus is asked to fix on a given accommodative target, the observer may see one or both eyes slowly converge. Then, with no alteration in attention or accommodation, the eyes may straighten or diverge (Figure). Various degrees of supravergence may occur concomitantly. This variable movement resembles a slow, tonic ver-

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TABLE 1

TYPE OF STRABISMUS

Strabismus	Patients (No. = 215)	
	No.	%
Esotropia	76	35
A-pattern	14	6
V-pattern	7	3
Exotropia	64	30
A-pattern	8	4
V-pattern	2	1
Hypertropia	8	4
Paralytic	6	3
Dyskinetic	66	30

gence. Alternation of fixation occurs freely whether the eye is esotropic or exotropic. Ductions are usually full. The deviation constantly changes and adequate prism cover measurements are impossible to obtain.

We matched these 66 patients (Group 1) with a computer-generated random set of 66 surveyed patients from the same hospital who had constant strabismus (Group 2). The two groups did not differ significantly in age, sex, or ability to cooperate. We compared the two groups to see if dyskinetic strabismus was associated with any specific ocular or neurologic disease patterns.

We used the χ^2 distribution and Student's *t*-test for statistical evaluations.

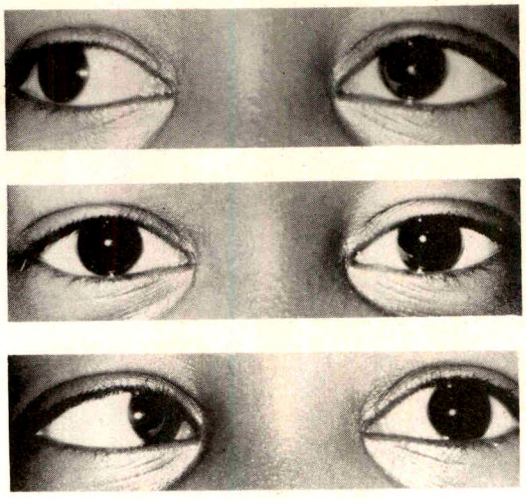


Figure (Buckley and Seaber). A patient with cerebral palsy fixing on an accommodative target. All three photographs were taken during a two-minute period, showing how variable the deviation can be in dyskinetic strabismus.

RESULTS

The patients ranged in age from 1 to 18 years (mean, 5 years). Sex and race were equally distributed. Dyskinetic strabismus was found in 66 (30%) of the patients with cerebral palsy who had strabismus. This equalled the 64 patients (30%) with exotropia and was only slightly less than the 76 patients (35%) with esotropia.

A classification of the patients by motor type of cerebral palsy disclosed a marked asymmetry between Groups 1 and 2. Of the 66 patients in Group 1, 37 (56%) had an athetoid component to their disorder. This differed significantly ($P<.0001$) from Group 2 which had seven patients (11%). The 37 patients in Group 1 represented 84% of the total number of those with an athetoid component (44 patients). The spastic type was distributed more equally with 17 patients (26%) in Group 1 and 30 patients (45%) in Group 2. The 17 patients in Group 1 represented 36% of the total number with the spastic type (47 patients).

We compared the associated ocular findings in Group 1 with those in Group 2 (Table 2). Refractive errors did not differ significantly, but amblyopia was much more common in Group 2. This amblyopia was not associated with anisometropia, refractive error, nystagmus, or any other ocular motility abnormality other than strabismus (Table 3). A high incidence ($P<.005$) of upgaze palsy was found in Group 1. Nine patients had upgaze palsy in Group 1, whereas only one in Group 2 did.

The fluctuating nature of this strabis-

TABLE 2

OCULAR FINDINGS

Findings	Dyskinetic Group (No. = 66)		Constant Group (No. = 66)	
	No.	%	No.	%
Refraction				
Emmetropia	25	38	26	40
Hyperopia (1 diopter of error)	25	38	27	40
Myopia (1 diopter of error)	16	24	13	20
Astigmatism	22	33	22	33
Anisometropia (difference > 1.5 diopter)	7	11	3	5
Strabismus				
Horizontal esotropia	—	—	36	56
Horizontal exotropia	—	—	30	44
Horizontal variable	66	100	—	—
Vertical hypertropia	8	12	5	11
Vertical variable hypertropia	7	10	—	—
Vertical DVD	8	12	4	6
A-pattern*	6	9	15	23
V-pattern*	8	12	3	5
Upgaze palsy†	9	13	1	2
Amblyopia‡	9	14	19	29
Optic nerve atrophy				
Monocular	1	1	3	5
Binocular	2	3	2	3
Hypoplasia	0	0	1	1
Nystagmus				
Horizontal	9	13	5	8
Vertical	3	5	2	3
Rotary	1	1	4	6
Latent	3	5	3	5
Coloboma	1	2	1	2
Retinitis pigmentosa	1	2	1	2
Dragged disk	0	0	1	2

*$P < .05$.
†$P < .005$.
‡$P < .03$.

mus can not be explained by an inability to control ocular examination factors. Variability might occur, for example, as a result of uncorrected accommodative esotropia or in patients with high AC/A ratios. The percentage of hyperopia in our Group 1 was actually less than that in Group 2, and 41 of the 66 patients (62%) had either myopia or emmetropia. We tried to control the accommodative effort and all measurements were made by at least two observers. Other factors in the examination technique that might have given a false impression of variability, such as the patients' attentiveness and cooperation, were similar in the two groups.

The strong association of athetosis with dyskinetic strabismus led to a search for additional ocular differences between athetosis and the other forms of cerebral palsy (Table 4). The only other significant finding was the absence of optic atrophy in the group with athetosis.

Our patient population did not differ significantly from those in previous reports[2,6,7,8,12,20] either in the incidence of visual defects or in the distribution of

TABLE 3

AMBLYOPIA IN STRABISMUS

Groups	No Amblyopia		Amblyopia	
	No.	%	No.	%
Constant (No. = 66)				
Esotropia	26	72	10	28
Exotropia	21	70	9	30
Dyskinetic (No. = 66)	57	86	9	14
Total (No. = 132)	104	79	28	21

cerebral palsy type. The only difference was the recognition of dyskinetic strabismus.

DISCUSSION

The term cerebral palsy covers a group of nonprogressive but changing disorders secondary to a malfunction of the motor centers of the immature brain. Cerebral palsy is characterized by paralysis, weakness, and uncoordination.[10] The most useful classification of cerebral palsy divides patients into groups according to the clinical and anatomic characteristics of spasticity, athetosis, and ataxia. The spastic type is caused by lesions in the cerebral cortex or pyramidal tract that affect upper motor nerves. It has the associated findings of increased tone of the involved musculature, exaggeration of the deep tendon reflexes, clonus, and a tendency to contractures. Athetosis is associated with lesions in the basal ganglia and is characterized by uncontrolled and purposeless movements that impair voluntary activity. The abnormal move-

TABLE 4

COMPARISON OF OCULAR FINDINGS BY MOTOR CLASSIFICATION

Ocular Findings	Athetoid (No. = 44)		Spastic (No. = 88)	
	No.	%	No.	%
Refraction				
Emmetropia	14	32	37	42
Hyperopia	18	41	34	39
Myopia	12	27	17	19
Astigmatism	16	36	28	31
Anisometropia	6	13	4	5
Strabismus				
Constant esotropia	4	9	32	36
Constant exotropia	3	7	27	31
Dyskinetic*	37	84	29	32
Vertical deviation	13	30	29	33
Upglaze palsy	6	14	4	5
Optic atrophy†	0	0	9	10
Nystagmus	10	23	20	23
Amblyopia	9	20	19	22

*$P < .001$.
†$P < .05$.

ments are less apparent when the patient is relaxed, but are increased under stress or tension.[7,11,19]

Dyskinetic strabismus can be the first clinical sign of cerebral palsy. We have seen two children in whom cerebral palsy was diagnosed because of the recognition of dyskinetic strabismus. The earliest indication is an esodeviation that is usually noticed by the parents shortly after birth. The parents are sometimes aware of the variation from straight to crossed, but most frequently comment on the appearance of varying degrees of esotropia. Only rarely have parents volunteered that they noticed the deviation changing from esotropia to exotropia early in the child's life. With prolonged observation, the fluctuation from esotropia to exotropia can be documented by a trained observer. We, along with many parents, have noticed that exodeviation occurs more frequently and with greater amplitude as time passes. We have observed several of these children for years and have noticed this in every case.

We found a much lower incidence of amblyopia in Group 1 (Table 3). The unstable angle of deviation may prevent deep sensory suppression from developing. A few of these patients reported fleeting fusion during their straight periods. Breakey, Wilson, and Wilson[1] noted a much lower incidence of amblyopia in patients with athetoid palsy compared to those with spastic palsy, and reasoned that it was caused by the location of the central nervous system damage, that is, the basal ganglia. Unfortunately, they did not say why this association occurs. Lesions in the basal ganglia may cause dyskinetic strabismus, which in turn makes amblyopia less likely to develop. Further evidence supporting the role of basal ganglia lesions in dyskinetic strabismus is the strong association of these lesions with upgaze palsy and athetosis. We know that damage to the basal ganglia in kernicterus can lead to athetosis and upgaze palsy.[11] The basal ganglia participate in the extrapyramidal system and help coordinate voluntary, involuntary, and reflex eye movements. Athetosis, upgaze palsy, and dyskinetic strabismus may represent differing clinical manifestations of the same central nervous system lesion.

Additional evidence for a common lesion is that dyskinetic strabismus can be the first indication of an athetoid component to the cerebral palsy. In 14 of our patients the diagnosis of athetosis was made months to years after the initial observation of dyskinetic strabismus.

Treatment—Unlike other forms of strabismus, dyskinetic strabismus does not respond to surgery. We have seen three patients with dyskinetic strabismus who underwent operation elsewhere. The surgery had little effect on the type or magnitude of their deviations. Others have reported excellent results after surgery on constant deviations in patients with cerebral palsy.[2,8] We generally agree with these findings. However, if variability is suspected we recommend treatment of any existing amblyopia and observation until a stable constant angle develops.

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Ophthalmic miniature

"Everyone's eyes are about the same size. You're disappointed. The 'windows of the soul' and all that? Where would romance be without eyes? The fact is, when we talk about the shape of a woman's eyes, we're really describing the shape of her eyelids. 'She deliberately shrouded the light in her eyes but in spite of herself it gleamed in the faintly perceptible smile.'"

"*Anna Karenina.*"

"A literate man! I suspected it all along. And it's eyelids, nothing but eyelids and muscle attachments."

Martin Cruz Smith, *Gorky Park*
New York, Random House, 1981

SELF-INFLICTED OCULAR MUTILATION

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Self-inflicted mutilation of the eyes is an uncommon but fairly distinct entity. Patients are typically young men; they often have criminal records and histories of drug abuse. Underlying schizophrenia is a common feature. The patients frequently suffer from castration fears, oedipal conflicts, repressed homosexual impulses, severe guilt, and a need for punishment.

We treated two patients with this disorder. The first, a 26-year-old prison inmate convicted of murdering his grandfather, cut his eyes with a razor blade. He had previously attempted to commit suicide, suffered from delusions, and experienced auditory hallucinations. Surgical repair of his injuries improved his visual acuity to R.E.: 6/15 (20/50) and L.E.: 6/30 (20/100). The second patient, a 24-year-old inmate of a mental institution, had previously undergone a lensectomy for a hypermature cataract induced by a self-inflicted lye burn in his left eye. One year later, he was treated for lacerations of his right eye and partial amputation of his tongue. In the year between admissions, he had enucleated his left eye. Visual acuity in his remaining eye was no light perception, so no surgery was performed.

Self-inflicted ocular mutilation is a bizarre form of psychotic behavior that is rarely encountered by ophthalmologists. Although some prefer to restrict the term "oedipism" to cases of self-inflicted enucleation (named for Oedipus who gouged out his eyes after learning he had committed incest),[1] many have applied it to all forms of self-inflicted mutilation of the eyes.

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Tapper, Bland, and Danyluk[2] related how Marco Polo, visiting Baghdad in the 13th century, was told the story of a cobbler who had destroyed his right eye with an awl after experiencing sinful desires when the leg of a young woman was exposed as he fitted her with slippers. MacLean and Robertson[3] stressed the following factors in cases of self-inflicted enucleation: (1) castration fears; (2) failure to resolve oedipal conflicts; (3) repressed homosexual impulses; (4) severe guilt; and (5) self-punishment.

Our two cases illustrate the salient features of self-inflicted mutilation of the eyes.

Case reports

Case 1—A 26-year-old man, a prison inmate, was brought here in May 1979 because of pain and decreased visual acuity in both eyes. When questioned, he calmly stated that he wished to leave prison and commit suicide, and so he had taken a razor blade and cut his eyes.

Visual acuity was light perception in the right eye and counting fingers in the left eye. There was a full-thickness corneal laceration in the right eye and many partial-thickness lacerations were present in the left eye (Fig. 1). A traumatic cataract and iris prolapse were also present in the right eye. A lensectomy and anterior segment reconstruction were performed in the right eye and the corneal lacerations were closed with 17 10-0 nylon sutures in the right eye and 28 sutures in the left.

The corneal edema gradually decreased and two months postoperatively visual acuity had improved to R.E.: 6/15 (20/50) and L.E.: 6/30 (20/100). Six months postoperatively, a total retinal detachment developed in the right eye; this was later repaired. The patient was transferred to another institution and has been lost to follow-up.

Psychiatric history—When he was 6 years old, the patient was raped by his alcoholic grandfather. In 1974, at 21 years of age, he was admitted to a psychiatric unit after taking 30 methaqualone pills (300 mg each) in a suicide attempt. He later developed viral meningitis, a urinary tract infection, and pneumonia. Four months before this suicide attempt, he had reported a homosexual assault that occurred while he was working in a restaurant kitchen. He admitted to occasional use of lysergic acid diethylamide.

The 1974 psychiatric profile quoted him as saying: "I'm down. I have nothing to look forward to. I don't have any confidence. I don't even care about girls anymore." He also complained of a disturbed sleep pattern, with early-morning awakening, difficulty in falling asleep, and persistent suicidal ideation. He experienced auditory hallucinations, hearing voices that called him a "faggot." His thought content was essentially depressive: "What's the use? I can't stop thinking of myself as a loser."

In 1976, the patient was admitted to a hospital on an emergency basis after being arrested for assaultive, homicidal behavior. He was undergoing outpatient psychotherapy at the time of admission. His condition had been stabilized by haloperidol and benztropine mesylate, but he had stopped taking them two weeks previously. His parents stated that he had become increasingly belligerent and had recently provoked fights with strangers whom he considered threatening.

The diagnosis was schizophrenia, paranoid type. He suffered from many delusions. The patient believed that he was working for the Central Intelligence Agency, and had recently run across an airfield, trying to catch a plane that was taking off because he was "to receive assignments." He also stated that, "I did a sex change on my own. I opened my head and started a boy from it. He is my father, but he is disguised. I changed from heterosexual to a trisexual. I have orders to Castro; I gave them." His affect was flat, he lacked insight, and his judgment was poor.

As an inpatient, he improved after reinstitution of haloperidol, benztropine mesylate, and diphenhydramine HCl. He was discharged and remained under observation as an outpatient.

In 1978, after a relapse, he was readmitted to the hospital; the diagnosis was chronic paranoid schizophrenia. In the emergency room, he shouted, became agitated, and threatened to kill the mayor and the grandfather who had sodomized him during his childhood. His thought content was characterized by paranoid delusions. The patient stated that the

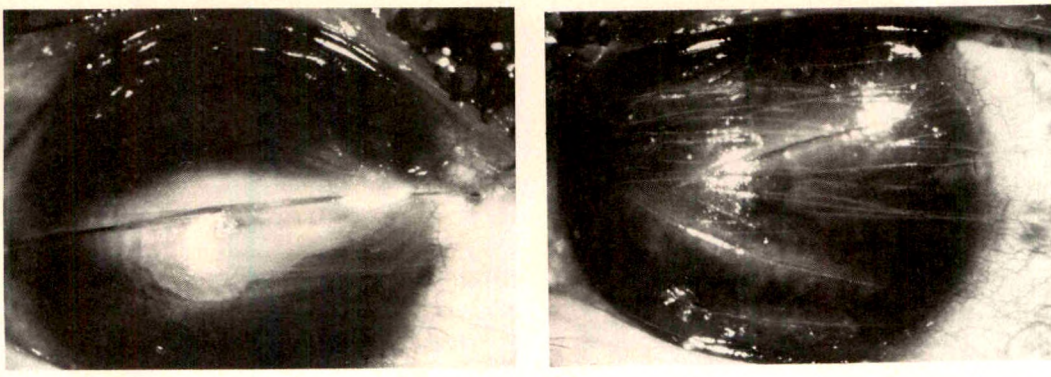


Fig. 1 (Yang, Brown, and Magargal). Case 1. Left, Right eye. A full-thickness, linear, horizontal corneal laceration is surrounded superiorly and inferiorly by marked corneal edema. A traumatic cataract was also present. Right, Left eye. Many partial-thickness corneal lacerations are apparent.

psychiatrist looked like a girl and was a "faggot." He also said, "I can feel your anus burning." The patient stated that he could control the minds of others and send them out to do his bidding. He added that he was an undercover agent with many lethal weapons inside his body. After his condition was stabilized with medication and psychotherapy, he was discharged for outpatient management. Several weeks later the patient was released from custody and subsequently murdered his grandfather, the crime for which he was incarcerated when we examined him.

Case 2—A 24-year-old man was admitted here in May 1978, and underwent a lensectomy for a hypermature cataract induced by a self-inflicted lye burn in his left eye. The right eye was normal, with a visual acuity of 6/7.5 (20/25).

Seven days postoperatively, a cicatricial entropion of the left upper eyelid was corrected with a surgical tarsectomy procedure and marginal rotation of the left upper eyelid. Eighteen days later, he was admitted again with an impending corneal perforation of the left eye and so a permanent tarsorrhaphy (intermarginal eyelid adhesion) was performed in an attempt to protect the cornea.

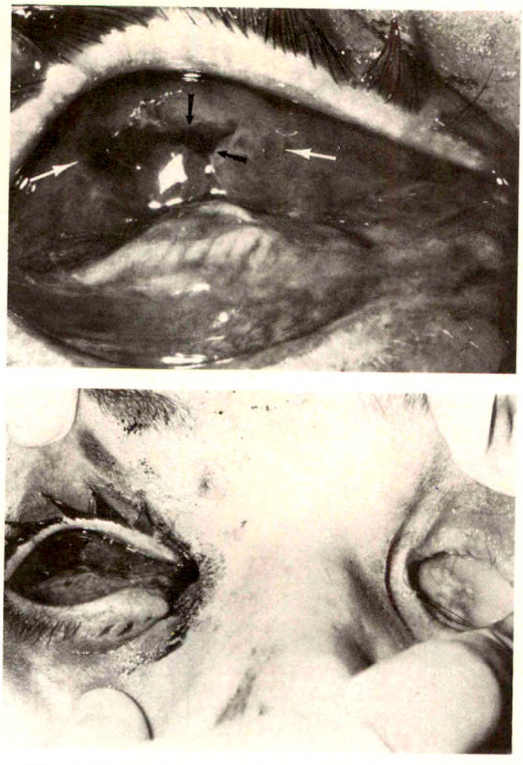


Fig. 2 (Yang, Brown, and Margargal). Case 2. Top, Right eye. A triradiate, full-thickness corneal laceration is present (black arrows). The corneoscleral limbus is indicated by the white arrows. Bottom, Both eyes. The right lower eyelid shows a laceration involving the lower canaliculus. The patient had previously enucleated his left eye and purulent material is evident in the socket.

Between May 1978 and June 1979, the patient was lost to follow-up. Although a reliable history could not be obtained from the patient, psychiatric records indicated that the patient had enucleated his left eye during this period.

In 1979 he was transferred here from a psychiatric institution after suffering lacerations of the right cornea and right lower eyelid in an abortive attempt at self-enucleation with the top of an aluminum can. An examination performed at the time of admission disclosed that there was no light perception in the right eye. The right globe was partially collapsed, and a triradiate corneal laceration was evident (Fig. 2). The cornea was diffusely hazy, and a hyphema was noted. There was vascularization of the cornea, probably secondary to a self-inflicted injury with a wooden stick several months previously. The conjunctiva was edematous with areas of chronic scarring. The patient was calm and did not appear to be distraught about his situation. Both the cornea and right lower eyelid laceration were repaired.

Postoperative ultrasonographic evaluation showed a diffuse hemorrhage in the vitreous cavity. Additionally, a pattern consistent with a total retinal detachment was present. Because the patient had no light perception, no further surgical procedures were attempted.

Just before admission, the patient had also attempted to amputate his tongue with the top of a soda can (Fig. 3). Although he had threatened to castrate himself, the patient had inflicted no injury on his genitals.

PSYCHIATRIC HISTORY—Psychiatric evaluation indicated that although the patient had mutilated himself at widely spaced, unpredictable intervals, he showed relatively normal behavior at other times. He had been considered a voluntary patient, residing in an open building. For several months before admission, he had been considered stable, until depression and anxiety increased during the week before admission. The diagnoses made were explosive personality, schizoaffective disorder, and depression.

DISCUSSION

Psychiatrists had established the diagnosis of schizophrenia in both of our patients, a common feature in other reported cases of self-inflicted ocular mutilation.[2,3] Macular lesions in cases of self-inflicted solar retinopathy have been reported in 27 patients in a psychiatric

hospital who were all schizophrenic.[4] However, Tapper, Bland, and Danyluk[2] cautioned that self-inflicted ocular injuries may occur in individuals with organic psychoses, as well as in those in acute and chronic drug-induced abnormal states. Depression, alcoholic intoxication, epilepsy, and diabetes with renal, vascular, and infective complications have all been associated with self-inflicted ocular injuries. However, chronic underlying schizophrenia is clearly a common finding.

We doubt that either of our patients injured himself for secondary gain, although injuring an eye to obtain monetary compensation has been reported.[5] Patient 1 said that he lacerated his corneas with a razor blade because he wanted to escape imprisonment. A literal interpretation suggests a simple motive of freedom from incarceration. It is not uncommon for prisoners to inflict minor injuries that are not life-threatening, such as swallowing foreign bodies, upon themselves in order to enter a hospital. However, an act serious enough to cause possible bilateral blindness is less likely to have been motivated by a desire to escape institutionalization. Instead, the patient may have been offering his self-inflicted blindness as penance to assuage his extreme guilt for murdering his grandfather. As the King James version of the Bible says, "If thy right eye offend thee, pluck it out and cast it from thee: for it is profitable for thee that one of thy members should perish, and not that thy whole body should be cast into hell" (Matthew 5:29). However, the previous incestuous relationship with his grandfather undoubtedly provided some rational basis for the slaying.

The eye may symbolize the penis, and injury to the eye may represent castration. Patient 2, who threatened to castrate himself, did partially amputate his tongue, which has possible phallic connotations. This was consistent with this motif of self-castration.

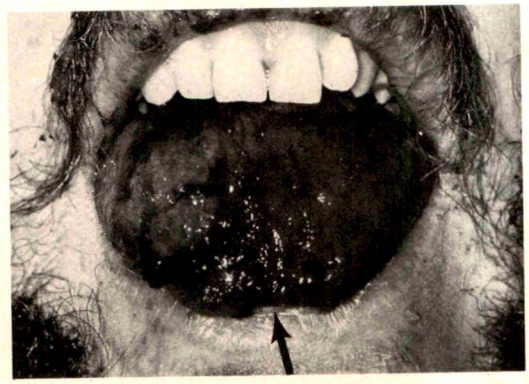


Fig. 3 (Yang, Brown, and Margargal). Case 2. The tongue is partially amputated (arrow) and there are several lacerations.

Because the ego and eye are intimately related, any act of violence directed toward the eye can be interpreted as a suicidal gesture. Patient 1 demonstrated suicidal tendencies when he nearly ended his life with an overdose of sedatives five years before assaulting his eyes.

Both of our patients were young men, and one of them possessed a criminal record. Self-inflicted ocular mutilation typically occurs in young men,[2,6,7] particularly in those who have been imprisoned.[6]

Drug abuse, particularly abuse of lysergic acid diethylamide, is frequently associated with self-inflicted mutilation.[7,8] This is not surprising because self-destruction while under the influence of hallucinatory agents is a well-recognized phenomenon.[7]

Tapper, Bland, and Danyluk[2] described a patient remarkably similar to ours. A young man with a long history of psychiatric treatment suffered from auditory hallucinations before his self-inflicated ocular trauma. Their patient, like both of ours, was not concerned about the exten-

sive damage to his eyes. On one occasion, he asserted that his eyes were healed. A period of such denial of reality is sometimes prominent in self-inflicted ocular mutilation.

Others have described self-inflicted enucleation, the most extreme form of ocular mutilation. Heller[9] wrote in 1912 of a mentally ill patient who tore free his optic nerve. In 1899, Axenfeld[10] demonstrated experimentally that he could perform an optic nerve avulsion on a cadaver in less than one minute. As the fingers forcibly enter the orbit, the insertions of the extraocular muscles are placed under tension severe enough to rupture these connections. This potent compressive force may also perforate the globe. The bony walls of the orbit offer sufficient leverage to make complete extraction of the globe possible. These findings supported Axenfeld's report of self-inflicted enucleation by insane patients.

Recent studies have indicated that prophylactic treatment with neurotransmitters may be effective in syndromes involving self-mutilation. Gilles de la Tourette syndrome is a neurologic disorder which may lead to grunting and self-mutilation. Van Woert, Yip, and Balis[11] stressed that Gilles de la Tourette syndrome is a genetically determined biochemical disorder, and not a psychological disorder as was once thought. In this condition, purine metabolism is abnormal, with red blood cell hypoxanthine-guanine phosphoribosyltransferase less stable than normal.[11] Abnormal enzyme peaks can be detected by isoelectric focusing.[11] In 1977, Van Woert, Yip, and Balis[11] found that treatment with L-5-hydroxytryptophan, the precursor of serotonin, was successful in arresting the self-mutilating behavior of a child with Gilles de la Tourette syndrome. On three occasions when they substituted a placebo, aggressive behavior, including biting and facial punching, returned. Additionally, they were able to reduce the dosage of the major and minor tranquilizers required by their patient, allowing him to become more alert and eliminating the undesirable sedative effects of the neuroleptic drugs.

Similarly, self-mutilation in Lesch-Nyhan syndrome has been biochemically treated with L-5-hydroxytryptophan by Mizuno and Yagari.[12] They reported that periodic administration of this drug prevented the self-mutilation completely in four cases. Enzyme studies suggested that individuals with Lesch-Nyhan syndrome and those with Gilles de la Tourette syndrome do not have the same biochemical de[illegible]ct.[11] Thus, the action of L-5-hydroxytryptophan may be nonspecific. Although neither of our patients underwent biochemical assays of red blood cell purine metabolism, perhaps such assays should be done in patients with histories of self-inflicted mutilation of the eyes to determine whether this precursor of serotonin might be effective preventive therapy.

ACKNOWLEDGMENT

James L. Chapel, M.D., Department of Psychiatry, University of Missouri, Columbia, Missouri, reviewed the cases.

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Ophthalmic miniature

Puta relicta nobis medicamenta, quibus sanarentur oculi. Non opus est mihi alia quaerere: haec tantum morbis et temporibus aptanda sunt. Hoc asperitas ocularum conlevatur. Hoc palpebrarum crossitudo tenuatur. Hoc vis subita et humor overtitur. Hoc acuitur visus. Teras ista oportet, et eligas tempus, adhibens singulis modum.

Translation: Let us consider the remedies handed down to us for healing the eyes. There is no need to look for others. All one has to do is to apply them to fit the disease and the time.

This one takes away soreness from the eyes.
That one attenuates thickened lids.
That one removes sudden irritation and tearing, and that other one sharpens the vision.

Use each one at the right time and in the right way.

Lucius Annaeus Seneca: Epistles (Epistles LXIV)
Translated by Emile D. Svarc, M.D., F.R.C.S. (C)

JULIUS HIRSCHBERG, THE NEGLECTED HISTORIAN OF OPHTHALMOLOGY

CHARLES SNYDER
Boston, Massachusetts

The history of ophthalmology up to the start of World War I is well documented, perhaps better documented than the history of any other medical speciality. We owe this fortunate state of affairs primarily to the efforts of one man—Julius Hirschberg, M.D. (Figure). An extraordinary person, certainly a genius, he devoted years to the task of recording the past of the medical speciality he loved and practiced. Circumstances prevented him from receiving during his life all the recognition and gratitude he deserved. Since his death in 1925, his life, accomplishments, and great gifts to ophthalmology have been treated at length only twice in the medical literature—once in English and once in German.[1,2] A strange fate indeed for one of the world's major medical historians.

EARLY YEARS

Julius Hirschberg was born Sept. 18, 1843, in Potsdam, Germany.[3] His father, a modest merchant, was ambitious for his children. His father recognized young Julius' superior intellect and made plans to give the boy the best education the family purse would allow. At the age of 9 years, he entered the Royal Victoria Gymnasium in Potsdam. A willing and excellent pupil, he usually led his class. The curriculum of the school emphasized the classical languages, Greek and Latin. These Hirschberg quickly mastered and he went on to read and enjoy the writings of the ancients. One biographer suggested that Hirschberg's lifelong love of the Graeco-Roman world began during his years at the gymnasium.[1] His father saw to his education in modern languages. At an early age the boy was reading English, French, and Italian. Later in life he would add Arabic and modern Greek to his list. As a man he was fluent in five modern languages and knowledgeable in



Figure (Snyder). Julius Hirschberg at the age of 70 years. (From "Julius Hirschberg's ausgewählte Abhandlungen (1868–1912) zu seinem 70. Gehurtstage ihm überreicht im Namen seiner Schüler von Oskar Fehr und Wihelm Münsam." Leipzig, Veit and Co. 1913.)

From the Lucien Howe Library of Ophthalmology, Harvard Medical School, and the Massachusetts Eye and Ear Infirmary, Boston, Massachusetts.

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half a dozen others. With such a capacity for learning, it is not surprising that he graduated first in his class.

He went to Berlin to study medicine. His family by this time had little money and he had to support himself by tutoring fellow students and acting as stenographer to a medical society. During his student days many of the titans of 19th century German medicine were in Berlin—Helmholtz, Brücke, and DuBois-Reymond in physiology, Virchow, Traube, and Frerichs in pathology, and Langenbeck and Jünkgen in surgery. When the time came for Hirschberg to have a practice of his own, he, a hero-worshipper, placed photographs of all of them on his office walls. It was probably under Virchow's direction that he wrote his doctoral thesis, "Symbolae ad therapiam partus cum placenta praevia complicata." He graduated and took his degree on March 21, 1866. A year later he passed his qualifying examination. He was ready to meet the man who would shape his life and career—Friedrich Wilhelm Ernst Albrecht von Graefe (1828–1870).

As Hirschberg told it, their meeting makes a fascinating story.[3,4] During the 1866 war between Prussia and Austria, an epidemic of cholera broke out in northern Germany. Von Graefe was made chief of one of the cholera hospitals in Berlin. His hospital was understaffed so he turned to Virchow, who had charge of another hospital, for an assistant. Hirschberg as a student had worked in Virchow's pathologic laboratory and was at this time working under him in his cholera hospital. Virchow transferred Hirschberg to von Graefe's service. Hirschberg and von Graefe had not been together long when Hirschberg, by following von Graefe's orders to the letter, quarrelled with a police surgeon. In Prussia, recently graduated young doctors did not quarrel with police surgeons. Hirschberg was dismissed. Von Graefe explained the actions of his young assistant to higher authorities and then resigned. Perhaps to make amends, von Graefe then offered Hirschberg an assistantship in his ocular clinic. Hirschberg was 23 years old when he accepted the appointment.

Von Graefe had the rare qualities of earning the devotion of his young associates and of drawing out the best that was in them. Like others before him, Hirschberg was happy and enthusiastic about working under von Graefe; he was willing, even eager, to do more than his assigned duties. Von Graefe's genius, his extraordinary acumen, his love of work, charitable nature, and complete personal and intellectual honesty all made their mark on Hirschberg's impressionable nature. As a young man and as a neophyte clinician he could not help but be enriched by the experience. In later years Hirschberg repaid the debt by writing and speaking of his beloved master and his work. Indeed, the last piece of writing to come from Hirschberg's pen was an edited volume of lectures that von Graefe had delivered in 1859–1860.[5]

THE "POLICLINIC"

Hirschberg stayed with von Graefe for two years, and then left to start his own private practice and policlinic. Whenever he could, he used von Graefe's clinic and von Graefe's lifestyle as patterns. In time, he chose a building at 36 Karlstrasse.[4] On the first floor he conducted his large policlinic and held his lectures. The second floor was occupied by his consulting room, his attractive apartment, study, and library. The remainder of the building was a hospital with wards and private rooms for 40 patients and a well-equipped operating room. He was especially proud of the operating room, declaring it to be the first aseptic operating room in the world. With his own hands he sterilized the instruments and prepared for the operations.

This building was his world.[1] Within its walls he did all of his work and lived his life. His unvarying schedule called for him to rise early and meet his assistants at 8 A.M. in the operating room because operations were the first order of the day. Next came rounds and dressings. The time that remained of the morning he spent in his study. He met with his students at 1 P.M., began his policlinic service at 2 P.M., and began to see his private patients at 4 P.M. Rounds were made again later in the evening. He spent a few of his evenings in social activities, but gave most of them to study and writing.

Success in his private practice was soon his. Patients came to him from Berlin, from other sections of Germany, and from foreign countries. His practice grew steadily until it was so large that he needed the services of two full-time assistants and a number of volunteer assistants. The Hirschberg Clinic became one of the best known in Europe and he became one of the Continent's most famous ophthalmologists. None of this would have been if he had not had a strong, even domineering, personality, a deep sense of duty, great skill, a willingness to work hard, and the ability to use his time frugally.

His clinical work was always marked by great care and accuracy.[1,4] Those who assisted him had to be equally careful and accurate. He reviewed all important cases in the clinic. If anything was omitted or overlooked, he was sure to find it. Visual fields were taken with great frequency and care; every case of diplopia was accurately measured; drawings were made routinely of external and fundus diseases. In his clinic every patient received a urinalysis. Accurate and thorough work was the hallmark of his institution. When he failed with a case, he experienced a deep emotional upset that would last for days. Industrious, careful, and observant, he gathered records, clinical histories, drawings, and pathologic preparations, and then used the material in his many publications. His students were trained to do the same.

Hirschberg conducted his policlinic and private practice from 1869 to 1907. During that time he examined and treated a total of 256,647 patients. Between 1886 and 1907, the major operations totaled 7,915. These are indeed impressive figures.

Although ophthalmology was Hirschberg's great love, he made the entire field of medicine his province.[1] In his writings he laid great stress on the relationship of diseases of the eye to diseases of other parts of the body and systemic diseases. Two such diseases that received his attention were congenital syphilis and diabetes. He reported his findings in 1,000 cases of congenital syphilis. He wrote that a specific type of retinitis could be the first sign of the disease, that the blood vessels that formed on the cornea never disappeared and could often be seen years later, and that small vitreous opacities and the resulting flicker might be the patient's first sign. As for treatment, he found that an iridectomy relieved the iritis. He could offer his patients nothing except mercury ointment and potassium iodide. Arsphenamine, Dr. Ehrlich's "magic bullet," was in the future. He knew all the ocular manifestations of diabetes, but, once again, he could offer his patients no relief. Insulin was in the future.

Perhaps his greatest contribution to clinical ophthalmology was his work on the electromagnet and metallic foreign bodies.[6] The modern technique of magnet extraction is based mainly on his work. He improved on the giant magnet and constructed the first practical hand magnet. In his writings he stressed the value of careful preparation before each extraction and advised when to use the

large magnet and when to use the small magnet. He wrote from experience, for in 28 years he had performed 347 extractions. More than 56% of his patients retained good visual acuity. He is credited with being the first to give a clear and complete description of siderosis and its development and the first to emphasize the absolute necessity of promptly removing every metallic foreign body from the interior of the eye.[1]

In the years when trachoma was still the curse of much of Europe, Hirschberg headed two commissions to investigate the problem in the eastern and western provinces of his native Prussia. His foreign travels had made him an authority on the distribution of the disease in other parts of the world. He concluded that trachoma was linked to poverty and malnutrition and that race played no role. His prophylactic recommendations made good sense and his operative recommendations were the best the times allowed.

THE TEACHER

Hirschberg was a first-class educator. During the last third of the 19th century, Berlin came to rival, if not to surpass, Vienna as the mecca of foreign medical students. He was one of the attractions that drew them to the northern city.[7] Once there, many of them came to prefer his instruction to that offered in the Berlin University Ophthalmic Clinic. His clinic was found to be most interesting and was always crowded with Americans, as well as doctors from other countries, who listened to his lectures and studied his painstaking clinical methods. Short of stature, rotund of form, blond of hair and beard, he dominated his classes by virtue of his qualities as a teacher and the clarity, purity, even the elegance of his language. In more than 35 years of teaching, he taught dozens of Americans in his regular and special courses. He found that they were, on the average, older than his German students. They were equal to the Germans in ability and diligence, but were less well prepared. Language difficulties played no decisive role. They learned German rather quickly and were able to examine patients in a short time. One American, Harry Friedenwald, served as clinical assistant to Hirschberg for three years.

Friedenwald tells us that Hirschberg was totally opposed to women in medicine.[8] When they started to come to Berlin for advanced training, he termed them "intruders" and "adventuresses." He vowed that none would be allowed to enroll in his courses. He did not anticipate, however, the persistence of one, the tall blonde Dr. Hubbard from Boston, and her effect on the soft heart of Harry Friedenwald. Hubbard took a private course in ophthalmology with a Privat-Docent, one of Hirschberg's assistants. With this to her credit, she asked Friedenwald to obtain permission for her to observe in Hirschberg's policlinic. Hirschberg was surprised to learn that the young woman had been trained by one of his assistants. This placed him in the awkward position of being unable to say no. He told Friedenwald, "She may come and sit quietly and listen to what I say, and she may take a case now and then and examine it with the ophthalmoscope, but only women and children, for our men are not satisfied if we allow women to examine them." This concession was all Friedenwald and Hubbard wanted. We do not know if Hirschberg ever admitted another woman to his courses.

Despite his talents, qualifications, and popularity in certain circles, Hirschberg never attained the rank of Professor in Berlin. His first academic appointment, that of Privat-Docent in Ophthalmology, came to him on March 24, 1870. He was too young to succeed von Graefe as Professor when that master died in the

summer of 1870. The professorship went to Schweigger who held it for 30 years, until 1900. During those years Hirschberg advanced to the position of Extraordinary Professor and then to Medical Counsellor (Medizinalrat). On the resignation of Schweigger, the professorship that perhaps should have gone to Hirschberg went to Julius Michel. Ordinary Honorary Professor became Hirschberg's academic rank in 1900.

His personality

Hirschberg often spoke of himself as "a child of von Graefe." His students and assistants he called "the grandchildren of von Graefe." Two of his assistants, Harry Friedenwald and Oscar Fehr, have left records of what it was like to be an underling of Hirschberg.[1,8,9] First, the pay was small, so small that Friedenwald was ashamed to tell his father the exact sum—60 marks a month. Small in money matters, Hirschberg could also be small in giving credit for original work done by an assistant.[8] He required them to be on duty at all times and, if necessary, to come down from their attic bedroom in the middle of the night to tend a restless patient. They met him the first thing in the morning, assisted at his operations, accompanied him on his rounds, made the preliminary examinations of his private patients, and, of course, cared for patients in the policlinic. During his absences, they managed the affairs of the clinic and performed operations. Their time away from hospital duties had to be spent in research, reading medical books and journals, and doing minor writing and editorial chores for Hirschberg's journal, "Centralblatt für praktische Augenheilkunde," and they had to participate in the teaching programs as assistants. They worked seven days a week, holidays were few, and vacations were granted grudgingly and could be terminated without notice.

Like assistants since time began, they gossiped about their Chief whenever they had the chance. Friedenwald tells of one session where one of the topics was the rudeness, almost brutality, of Hirschberg.[8] The young men present thought it necessary to give patients hope and cheer even when they knew that the patients would eventually become blind. One day a patient, a young man 18 or 20 years old who had vitreous opacities, came into Hirschberg's policlinic. Hirschberg examined him and then asked if he belonged to a company insurance group. The boy's answer was no. "Well, then," said Hirschberg, "you had better get into one so you can go to their clinic—you are going blind." These cruel words were said with unsympathetic coolness. The boy said nothing, but when the assistant tried to examine him, the examination could not be done because of tears in the boy's eyes. Friedenwald hoped and prayed that he would never cause such pain to any person—that was not his object in becoming a physician. The tale ends with these words—"Hirschberg is a brute."

As the story demonstrates, Hirschberg could be harsh in his attitude toward clinic patients. In this he was not alone. There are many accounts of patients receiving what bordered on inhuman treatment at the hands of the chief physicians of some of the German clinics in the 19th century.[7]

Others have written that Hirschberg was a man of charm and of warmth and that his face could be full of love when he saw students or private patients. Both Fehr and Friedenwald submitted to his demands, came to fear him, to respect him, and then to love him dearly throughout his life. When he died, both men wrote warm eulogies.[5,10]

When he wished, Hirschberg could be quite charming and almost sociable. He went to the theatre, concerts, or the

opera, especially if a work of Wagner's was being presented. His extraordinary mind allowed him to memorize pages of German literature, particularly Goethe. Without being asked he recited long passages to some unsuspecting soul and did not know when to stop. Although he enjoyed playing the host, his guests did not always enjoy being guests. One writer[1] stated:

> He much preferred talking to listening, and expressed his views in a dogmatic way, tolerating no contradiction. He did not converse, he made speeches. If he noticed that somebody was inattentive, he stopped and addressed the person: "You, listen to what I am saying." He was something of a bore.

John E. Weeks described one evening at the Hirschberg home.[11] The time was 1890 and the occasion was the International Medical Congress. Hirschberg gave a dinner at his home for ophthalmologists from 17 different countries. There were 21 guests. The only woman present was the beautiful and gracious Madame Hirschberg. After an excellent dinner, all the guests were called upon to give short addresses. The host had welcomed the guests and had invited them to speak in five different languages. One guest, an Englishman representing Turkey, surpassed Hirschberg by speaking six languages. It seems that Turkish was not one of the languages mastered by Hirschberg.

It was his habit to break his year of intense labor by spending long vacations in foreign travel. There were few countries in Europe that he did not visit at one time or another. Extensive study preceded each of his trips. While traveling he took copious notes. When he returned to Berlin, he wrote of his experiences in articles and books. Among these were "Parisian Eye Clinics" (1870), "English Eye Clinics" (1877), "Tunis" (1885), "Egypt, Historical Studies" (1890), "Journeys to America" (1888 and 1905), "Visits to Greece" (1910), and "Around the World" (1894). His publications dealt with ophthalmology and medicine in the various countries; unexpectedly, they also dealt with the geography, economy, social life, and politics of these countries. In his writings on America, for example, he gave his impressions of American railroads, the amazing growth of Chicago, and the influence of German emigrants. Chicago fascinated him, especially its meat packing plants. He commented on the working class and their independence, the lack of servants, and the hopes of blacks.[7] Hirschberg was a good observer and his books are interesting reading. Why did he travel? In order to attend a congress or medical society meeting, or perhaps to give an invited lecture; more often than not, he wanted to examine a rare historical document or book in its home library.

Friedenwald relates another interesting aspect of Hirschberg's personality.[8] Although he was Jewish, he seldom concerned himself with Jewish life other than to contribute annually to the Jewish Charities of Berlin—until the early weeks of 1914. The Twelfth International Ophthalmological Congress was scheduled to be held in St. Petersberg in August 1914. The Russian authorities sent out a circular stating that the passports of Jewish doctors planning to attend the Congress would be specially stamped and that their stay in Russia would be limited to one week. No such regulations would exist for non-Jewish doctors. When Hirschberg learned of this he acted quickly. He wrote strong letters to his many colleagues around the world urging them to resign as he was doing unless the restrictions were lifted. Friedenwald joined him in the fray. Before long the Organization Committee in St. Petersburg received protests and resignations from societies and ophthalmologists in Maryland, New York, Brussels, and Naples. The International Committee of the Congress backed

Hirschberg. The Russian government was forced to lift the restrictions. Another circular was sent from Russia, one stating that "the Central Bureau announces that the Ministry of the Interior has authorized free entrance and an unlimited stay in Russia to Members of the Congress without exception." One letter of congratulation that arrived after the incident read, "Gutta cavat lapidem" ("The drop hollows out the stone").

For one of the final issues of the 1914 volume of his "Centralblatt," Hirschberg wrote a history of the International Congresses of Ophthalmology. When he came to record the Twelfth Congress he wrote, "The Congress in St. Petersberg planned for August 1914 did not take place. The story is over." ("Die Geschichte ist aus").

His writings

In his day no author was cited more frequently than Hirschberg.[5] The vast amount of his writings makes this statement believable. During his first year in private practice, 1869, he published his first textbook, "Glioma of the Retina." (With two exceptions all of Hirschberg's books are available only in German. I have translated the titles freely.) Five years later he brought out his "Mathematical Basis of Medical Statistics." This was followed by the three volumes of "Clinical Contributions." His knowledge of Latin, Greek, and Arabic enabled him to compile a "Lexicon of Ophthalmology" in 1887. Later came his two-volume work, "Introduction to Ophthalmology." On his 70th birthday, in 1913, his shorter writings, more than 100 articles, were republished in an 864-page Festschrift. He also wrote four books on Arabic ophthalmology, other historical books, and the 11 volumes of his "History of Ophthalmology." This includes only his writings on medicine and medical history, and not his work in other areas. He must have been the most prolific writer about ophthalmology of his generation.

As an author and historian Julius Hirschberg's great gift to ophthalmology was his "History of Ophthalmology" ("Geschichte der Augenheilkunde"). This masterpiece, published between 1899 and 1918, forms 11 volumes of the second edition of the "Graefe-Saemisch Handbuch der gesamten Augenheilkunde." (For some reason, most sources list only nine volumes. Of all the publications consulted for this study, only Duke-Elder's textbook[6] credits Hirschberg with 11 volumes.) Ranging in price from $3.30 to $4.50, the early volumes soon sold out. Circumstances prevented the later volumes from being so successful. Critics and medical historians called the work "authoritative and exhaustive," "the most complete and scholarly history of the science that has ever been written," and "a wonderful monument to German thoroughness." One said, "His monumental work today remains the authoritative history of ophthalmology. It thoroughness and critical judgment mark it as one of the greatest histories of scientific subjects." I may add that it is an unfailing lode that has been mined by three generations of medical historians.

In 1907 he gave up his policlinic and practice and devoted all his time to writing his great history. When he was finished in 1918, at the age of 75, he had written 11 volumes, a total of 4,720 printed pages. Only an individual with Hirschberg's rare combination of talent and industry could have accomplished the task. He was a master of languages, ancient and modern, a student of Egyptian hieroglyphics, and a translator of Arabic and Greek texts. As a physician and as an ophthalmologist, he was of the first rank. With his knowledge of the present, he could look to the past, read its literature, and judge the worthy and the unworthy. He had lived and worked

during a period of great change and productivity in the specialty. He knew all the giants. Over the years he had come to possess one of the largest private libraries of ophthalmic literature in the world. A figure of international note and a world traveler, he could ask others for information and it would be given to him. And, as Duke-Elder put it, "his greatest joy was perhaps his profound knowledge of and sympathy with classical and Arabic scholarship."[6] He loved his work, loved using his mind. And, most important, he had the ability to write in a delightfully attractive style. One critic went so far as to term him "a genial writer."

The first volume of his history was concerned with ancient ophthalmology, that of the Hindus, Babylonians, Egyptians, Greeks, and Romans. The contributions of the Arabs, Europe in the Middle Ages, and the beginnings of the modern era were dealt with in the second volume. In the third volume he continued the story of the modern era up to 1800. These three volumes are genuine works of scholarship. Information, much of it new to Western readers, was presented in a lively yet orderly fashion. The next four volumes told of the development of ophthalmology in Germany, France, England, and Italy from 1800 to 1850. There was one volume for each country. These were followed by a volume devoted to the history of ophthalmology in the United States in the 19th century, and another volume on the history of the smaller countries of Europe and Russia from 1800 to 1875. The largest volume of the series came next, the one Hirschberg entitled "Reform in Ophthalmology." The great changes, the reformation, came to the specialty with the introduction of the ophthalmoscope by Helmholtz, with Donders' work on refraction, and with von Graefe's pioneering clinical work. Hirschberg included lengthy biographies of the three men along with analyses of their contributions. The rest of the volume was primarily a history of ophthalmology in Germany and the German-speaking nations up to the beginning of World War I. The last volume contained additions, corrections, and a general index.

The enormous scope of Hirschberg's enterprise almost guaranteed that the volumes would not be equal in quality and scholarship. Nowhere is this better demonstrated than in "American Ophthalmology in the 19th Century." As a work from the pen of a lesser historian it might be acceptable, but it was a poor performance from one such as Hirschberg. Hirschberg knew America. He had traveled extensively there on three occasions, visited schools and hospitals, talked with ordinary people, and mingled with professionals. For many years he had reviewed and abstracted ophthalmic studies from the United States for his "Centralblatt." Thus, when it came time to write the history of American ophthalmology he had personal knowledge to draw on. He was willingly supplied with information by a host of acquaintances he had made. The writings of Hubbell, Friedenwald, and others were his to use. Out of all this something better should have come.

Hirschberg confessed that he met with two difficulties in composing his American history. The first arose from the complicated structure and diversity of the colleges and medical schools. The second arose because ophthalmology in the United States had developed slowly until the 1870s. He wrote that a complete survey would have come very near to his own time, and would have dealt with still unsolved problems and living physicians about whom he felt he had to withhold judgment. It is only fair to agree with him in this. But this could not have been the cause of the errors he allowed to mar his work. These include the following: Wil-

liam Charles Wells appears as William Charles Bell, James A. Bolten as James A. Bolters, Ezra Dyer as Esra Dyer, Henry Drury Noyes as Henry Dewey Noyes, and Alvin Allace Hubbell, one of his principle sources of information, as Alvin Allace Hubbel.

Remembering the great value of his other volumes, perhaps it is wrong to be too critical of this volume, to be too concerned with small errors of fact and spelling. Perhaps it is wiser to agree with the writer who reviewed the book for American readers when it was issued.[12] He felt that all American oculists should be grateful to Hirschberg for having spent time on such an immense labor. He said the book would be read all over the world and would greatly help to correct the erroneous conceptions, or in some instances the complete lack of knowledge, about America, still largely prevalent abroad. Hirschberg clearly left the writing of a definitive history of American ophthalmology to others.

One general criticism concerns the index. The history is in 11 volumes with a total of 4,720 pages divided into 1,312 sections. Each volume was paginated separately and the sections were numbered consecutively. When Hirschberg provided a general index for the whole work, he chose to index the names and subjects by sections rather than by volume and page. This can lead to a great deal of frustration. The reader must go through the volumes, locate the right section, and then go through the section, some of which are several pages long, to locate the desired name or fact.

Hirschberg's history has often been praised. His former assistant, Harry Friedenwald, summed up all the praise in these words[5]:

> It is the most comprehensive medical historical work that has ever been published, not of ophthalmology alone, but in the whole field of medical history. It is not an exaggeration to claim that Hirschberg has produced a complete history of ophthalmology; in doing this he has shown to the medical world the wealth of the heritage of ophthalmology.

Hirschberg's writing on medical history was not confined to his "Geschichte der Augenheilkunde." This amazing man also wrote, translated, and edited studies on ophthalmology in ancient Egypt, on Ibn Sina, Aetius of Amida, Hippocrates, Aristophanes, Shakespeare, von Graefe, and two books on early Arabic ophthalmologists. In addition to these textbooks, he wrote several journal articles and notes on the history of ophthalmic medicine and surgery. His accomplishments can only be viewed with awe.

He had no children. Perhaps he felt that, "These books shall be my posterity, they shall perpetuate my name." They do.

Hirschberg was an editor of note as well as an author. His "Centralblatt für praktische Augenheilkunde" (Leipzig, 43 volumes between 1877 and 1919) was his second great gift to world ophthalmology. In publishing his own journal he followed the pattern of the elite of 19th century German medicine. His two idols, Virchow and von Graefe, had had their journals, and he felt it was his turn. It was a risky business. Considering the number of men practicing ophthalmology at the time, the German language probably had enough journals devoted to the specialty. Hirschberg overcame this problem by including the most attractive features of his rivals in his journal.

Each year he brought out 12 issues, totaling 400 or 500 pages, in a volume. A typical issue contained original articles, proceedings of society meetings, clinical case presentations, book reviews, a survey of reports in other journals, obituaries, correspondence, and bibliographies. Something for everyone. Many of the original articles were by Hirschberg or his assistants, although he did not hesi-

tate to publish worthwhile offerings from as far away as Java. Societies, national, regional, and international, could have an abstract of their proceedings in his journal. He made certain that the proceedings of the Berlin Ophthalmological Society, a society he had founded, appeared regularly. He also made certain that the proceedings of the Colorado Ophthalmological Society, a society founded by his friend Edward Jackson, appeared regularly. The clinical cases came from his own clinic, or from the clinic of someone he knew and respected. His book reviews were brief, factual, and fair. The books themselves, usually sent by authors or publishers, went into his large personal library. When he surveyed a journal, he took an entire issue and abstracted every original article. He surveyed two, three, sometimes four journals in one issue. His gift of languages allowed him to review the publications of all the major European countries. Hirschberg must have loved obituaries because he wrote so many. Correspondence was selected from letters that crossed his desk. The bibliographies, some of them several pages long, were of subjects he felt would be of interest to his readers.

The "Centralblatt" was an excellent production and very much Hirschberg's personal journal. He wrote much of it himself and edited all of it. It was the instrument that made him world-famous. One writer commented that the "Centralblatt" was the best guide to the personality of Hirschberg, for its 43 volumes demonstrated the universality and versatility of his mind and the clarity of his judgment.[1] The volumes also disclosed a less attractive side of the man's nature—his penchant for self-promotion.

In 1910 Hirschberg published a small monograph on the treatment of myopia. G. Lindsay Johnson, a British ophthalmologist, bought a copy and, during a long voyage from Cape Town to London, translated it into English. He wrote a letter to Hirschberg telling him of the translation and of the high regard he had for Hirschberg's work. Hirschberg published the letter in its entirety in the correspondence section of his "Centralblatt." A few months later, in the same section, his readers were informed that the Johnson translation of his book had been published. In several subsequent issues his readers were treated to brief laudatory notices of the book taken from reviews and letters. All of these letters and notices were in English, not in German, which was the language of the journal. It was among the readers whose native tongue was English that he hoped to promote and sell his book. He used this same ploy throughout the 1915 volume of his "Centralblatt" when his book on the history of ophthalmology in America was issued.

Hirschberg had one other tour of duty as an editor. From 1879 to 1881 he served as co-editor of Herman Knapp's Archives of Ophthalmology (New York) and Archiv für Augenheilkunde (Wiesbaden).

Shyness was never a part of Hirschberg's nature.[1] He was always certain of his importance in the scheme of things, always aware of his dignity, and he expected others to be equally aware. He was very much the archetypal "Herr Professor." His many titles and honors gave him inordinate pride. Everyone, regardless of their station, was required to address him as "Geheimer Medizinalrat" ("Privy Medical Counsellor"). He fitted himself to the mold of many of his peers in the ranks of German medicine of the time. Like them, he demanded work, enormous quantities of it, of himself; like them he demanded recognition and honor for what he had done.

On one occasion when he wrote of himself, Hirschberg managed to keep his ego under control. The last full volume of his history is concerned with what he

termed the reformation in ophthalmology, the period after the introduction of the ophthalmoscope. He often devoted pages to lesser figures of the period; for himself he published his portrait, a brief autobiography—less than half a page—and a list of his publications. The entire piece is little more than an outline.

THE WAR YEARS

The summer of 1914 found Hirschberg at his peak as a figure in international ophthalmology. His journal and his clinical and historical writings had made his name known throughout the Western world. His travels had served to introduce him to ophthalmologists as far away as Japan. His students had publicized him wherever they went. His actions against the Russian bureaucracy concerning passports for Jewish physicians had marked him as a courageous and liberal man. But after the outbreak of World War I, things were never the same for Julius Hirschberg.

In the August-September 1914 issue of his "Centralblatt" he published the following notice on the front page:

> For the Fatherland
> Since the July number of the "Centralblatt" was dispatched to subscribers at home and abroad, the fearful war has broken out, wickedly forced upon us by our enemies.

The last phrase of this notice indicated that Hirschberg accepted without question the government position that Germany was the innocent victim of a systematic, long-planned international plot and that the nation had gone to war to survive. Some of those who had accepted Hirschberg as an international leader expected him to have a different attitude, a more liberal one, not the narrow nationalism that he did adopt. There is some evidence that Hirschberg suffered because of his ultra-patriotism. For example, neither THE AMERICAN JOURNAL OF OPHTHALMOLOGY nor the British Journal of Ophthalmology reviewed his books published during the war or before his death in 1925.

During the war years he kept busy. Although 71 years old, he returned to his clinic from time to time; much of the staff had gone to war, and he saw as many private patients as he could. He worked on the last five volumes of his "Geschichte der Augenheilkunde" and saw them published. He continued the "Centralblatt." He began his study on Shakespeare; he lectured. He received the Leibnitz Silver Medal of the Berlin Academy of Science. Meanwhile he confidently awaited the final victory of his nation.

When World War I ended, Hirschberg's life and his country were both in ruins. He was too old to build anew. He could rejoice that his great history was finished and safely in print. He could take no comfort from his "Centralblatt," for he was forced to abandon the journal at the end of 1919 because of competition and the lack of a successor as editor. But it was the fate of his library that must have broken his heart.

THE LIBRARY

The library was one of his proudest possessions. Over the years it had grown until it was one of the largest, most complete and famous ophthalmic libraries in the world. A catalogue of it published in 1901 consisted of 434 pages. It had always been his plan to leave it after his death to the Berlin Medical Society. His patriotism made this impossible. During the war he had invested much of what he owned in German war bonds. With his nation's defeat, the bonds became worthless. To obtain funds to live modestly, he was forced to sell his library.

The sale of Hirschberg's library and its later history is a story in itself.[13] Dr. Jujiro Komoto, who had become the first Professor of Ophthalmology at Tokyo University in 1890, had spent part of his student

days in Berlin and had come to know Hirschberg. He and Hirschberg became better acquainted after Hirschberg's visit to Japan in 1892 and Komoto's later visit to Berlin. On Dec. 2, 1920, Hirschberg wrote to Komoto offering to sell his library for 40,000 yen. The collection consisted of 3,200 books and 12,500 reprints and included:

> historical documents covering old Greek, Egyptian and Roman times, the Middle Ages up to the 19th century works. They are not only of European origin, but also from Eastern and Middle Eastern Countries. There are original papers on discoveries in the 19th century, and books and reprints that cover all possible fields of ophthalmology.[13]

There were no public funds to make the purchase, so Dr. Komoto decided to buy the library himself, and also to meet the costs of shipping and insurance. The money was to be paid to Hirschberg in 10,000-yen yearly installments. Unfortunately, Hirschberg died before the last payment reached him. In 1925, just after the Great Earthquake, the collection reached Japan, and Komoto immediately donated it to the Library of Tokyo University. For a number of reasons the collection was not completely catalogued and classified until 1938. The collection was then named the Komoto Library and became a part of the Central Library of Tokyo University.

Komoto wrote of Hirschberg's collection:

> His Library is the only one of its kind in the world and is the object of envy in many countries. We have the Library in Japan and I hope that the Library will continue to be of benefit to ophthalmology in Japan after my death.

The hope of Dr. Komoto has been realized. The respect in which the collection is held is demonstrated by this quotation from the Japanese Journal of Ophthalmology.[13]

> It is indeed the world's treasure not only of ophthalmology but also of human art. We are very happy that this large, invaluable collection has survived two World Wars and the Great Earthquake, and will be the possession of mankind forever.

Scholars may consult the Komoto Library, formerly the Hirschberg Library, by permission of the Library Department of Tokyo University. The name of Julius Hirschberg is an honored one in Japan.

THE LAST YEARS

In 1907 Hirschberg's policlinic building was torn down and a new one erected. Wilhelm Mühsam was put in charge. In his last years, Hirschberg made daily visits, walking through the corridors, and observing the activities. He had always dreamed that someday his clinic would be associated with the Berlin University as the "Julius Hirschberg Museum." This, of course, never came to be. After Hitler took power, Mühsam was forbidden to enter the premises.[1] The ocular clinic ceased to exist and the building became a children's ward.

In spite of his disappointments and hardships, Hirschberg took old age well. Harry Friedenwald visited him in the summer of 1923 and found him less vigorous but just as interesting; his mind and unfailing memory were just as clear as in the days when it was Friedenwald's privilege to assist him in the clinic.[5] He did not seem 80 years old. A year earlier he had given a course of lectures on Hippocratic medicine. He was working on his last book, von Graefe's lectures.

On Feb. 17, 1925, Julius Hirschberg, the most brilliant pupil of Albrecht von Graefe, died.[7] His passing was noted in the German medical press. Two of his former assistants, Oscar Fehr and Wilhelm Mühsam, wrote eulogies for the German ophthalmic journals.[10,14] In the English-language journals there was only one obituary, that of Harry Friedenwald in the Archives of Ophthalmology.[5] THE AMERICAN JOURNAL OF OPHTHALMOLOGY

and the British Journal of Ophthalmology printed brief notices. The French-language journals followed suit.

A great man had died, a great contributor to ophthalmology was no more, and outside of his native land little attention was paid. Why? Hirschberg may have been the victim of the chauvinism of World War I and of the years that followed. Perhaps he had lived too long, with his great days too far in the past. Perhaps the area in which he did his greatest work, the history of ophthalmology, was interesting to only a few in the specialty. How many ophthalmologists were interested in Ali ibn Isa of Baghdad and the translator of his Memorandum Book? Whatever the reason, amends were made some years ago by Sir Stewart Duke-Elder when he wrote of Julius Hirschberg and his history in the "Textbook of Ophthalmology."[6] Sir Stewart, ever the master of the felicitous phrase, closed his tribute with these words: "No other branch of medicine—no, indeed, so far as I am aware, of knowledge—has had a disciple so earnest, or has been presented with a gift so great."

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NOTES, CASES AND INSTRUMENTS

A CORNEAL PIGMENTED LINE ASSOCIATED WITH SALZMANN'S NODULAR DEGENERATION

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A 62-year-old woman with Salzmann's nodular degeneration had a pigmented line at the base of the corneal nodules in each eye. The lines ran horizontally along the superior one third of the cornea and faded at the corneoscleral limbus on both sides. The lines curved around the bases of the raised corneal lesions.

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Pigmented lines of the superficial cornea are commonly seen at the junction of the upper two thirds and lower one third of the cornea,[1,2] at the base of the cone in keratoconus,[3] at the apex of a pterygium,[4] and anterior to a filtering bleb.[5] We studied a new corneal pigmented line located at the base of the nodules in a patient with Salzmann's nodular degeneration.

CASE REPORT

A 62-year-old woman was referred here for evaluation of deteriorating vision and raised nodular lesions. She complained that she had had a gritty feeling in both eyes for the past ten years.

Her best corrected visual acuity was R.E.: 6/7.5 (20/25) and L.E.: 6/22 (20/70) with pinhole improvement to 6/12 (20/40) in the left eye. The eyelids, adnexae, pupils, and extraocular muscle function were normal in both eyes. We noted three raised, midperipheral, semiopaque superficial lesions in the right cornea at the 10, 12, and 2 o'clock positions. Slit-lamp examination disclosed a light-brown pigmented line at the level of deep epithelium or Bowman's membrane. The line was horizontal along the superior one third of the cornea and faded near the corneoscleral limbus on both sides. The line curved around the bases of the raised lesions (Figure, left).

We also noted two similar raised, semiopaque,

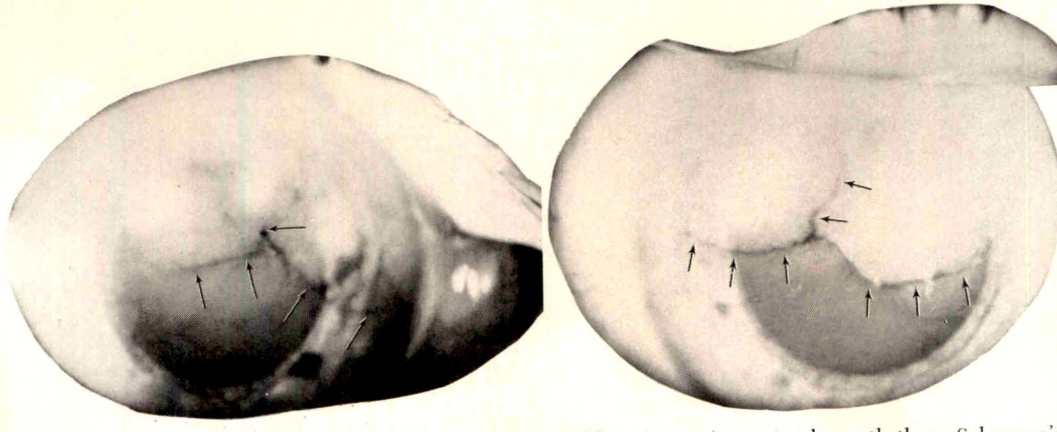


Figure (Reinach and Baum). Left, Right eye. Pigmented line (arrows) running beneath three Salzmann's nodules. Overexposure used to highlight pigmented line. Right, Left eye. Pigmented line (arrows) running beneath two Salzmann's nodules. Overexposure used to highlight pigmented line.

superficial lesions in the cornea of the left eye at the 10:30 and 2 o'clock positions in the midperiphery. The epithelium surrounding these lesions and in the area between the nodules demonstrated a fine surface irregularity that did not stain with fluorescein. Slit-lamp examination disclosed a light-brown pigmented line like that found in the right eye. A vertical extension of the line ran nasally to the nodule at the 2 o'clock position (Figure, right). There was a filamentous plaque at the 11 o'clock position peripherally in the left eye, but there was no corneal neovascularization. The remainder of the ocular examination gave normal findings.

Discussion

Salzmann's nodular degeneration is a relatively uncommon, noninflammatory corneal degeneration characterized by raised blue-white nodular corneal lesions that are often arranged in a circular distribution around the pupil. To the best of our knowledge, an associated pigmented corneal line in Salzmann's nodular degeneration has not been described previously.[6-12]

The pigmented line in our patient most closely resembled the Hudson-Stähli line in its coloration, horizontal course, and superficial location. Among the variations in the Hudson-Stähli line that we found in our case were branching, oblique orientation, and deviations around corneal macular scars.[13] However, its location in the superior one half of the cornea and its relationship to the raised nodular corneal lesion differentiated this line from the classic Hudson-Stähli line.

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A GONIOSCOPY LENS FOR PULSED-LASER TRABECULOTOMY

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We designed an indirect gonioscopy lens capable of handling the high powers generated by short-duration pulsed lasers. The lens is composed of an equilateral quartz prism within which the entire laser beam is internally

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reflected and connected to the cornea with saline solution. The reflected beam subsequently passes through the index-matched cornea to the trabecular region. Chromatic aberration is minimal because refraction is eliminated by arranging the laser beam so that it crosses the dielectric interfaces in a near-normal direction. The reflection losses amount to less than 5%, even without antireflection coatings. A prism of glass is sufficiently robust to handle the 100-kW pulses from a dye laser.

In recent years the continuous-wave argon laser has been successfully applied to the perforation of the trabeculum for the relief of open-angle glaucoma.[1-6] The laser beam is conducted to the target site with a conventional indirect gonioscopy lens designed for observation of the anterior chamber. Theoretical analysis[7,8] indicates that the short-timescale pulsed laser should be more effective for perforation since the target damage it produces is far more localized than that of the continuous-wave laser. Krasnov[9,10] verified this experimentally with a Q-switched ruby laser. However, the high power of the pulsed laser beam burns the reflective coatings of the conventional lens, and the optical pathways, formed from transparent plastic, tend to cavitate. Moreover, in our opinion, there is no satisfactory antireflection coating that has a long life when exposed to high-power pulsed radiation and to the clinical sterilization process required for such a lens. The lens described here* is primarily intended for use with the pulsed-dye laser system developed in this laboratory. This system, which incorporates a modified slit-lamp, generates a 100-kW pulse and is currently used to perform iridotomies in a single application.[11]

*A patent has been applied for.

Material and Methods

The necessary absence of plastic material and optical coatings implied that the reflection required within the indirect gonioscopy lens had to be at an optically dense-to-rare interface, such as that between quartz and air. Because the lens was also likely to be used for diagnosis and color photography, it had to have minimal chromatic aberration. This consideration also influenced the precision of target location because our apparatus uses a colinear aiming beam, derived from a continuous-wave helium-neon laser, that is some 40 nm longer in wavelength than the operating beam of the dye laser. Refraction is minimal if all working light paths within the lens traverse the relevant dielectric interfaces in a near-normal direction. Finally, the design had to be such that the approach to the patient's eye was in the normal direction because our apparatus, used for routine clinical laser iridotomy, was adapted to this situation.

These three requirements were satisfied by a simple arrangement of a single equilateral quartz prism connected to the eye by saline solution with the same refractive index as the cornea (Figure). The prism, which has 1.91-cm triangular sides and a height of 1.27 cm, is fitted into an acrylic plastic (Perspex) holder worked from a solid circular cylinder 2.90 cm in diameter. A recess for the prism is milled into the side of the cylinder with a 1.27-cm slot drill to a radial depth of 2.06 cm and to an axial length of 1.65 cm from the end face. The prism fits snugly into the recess with its

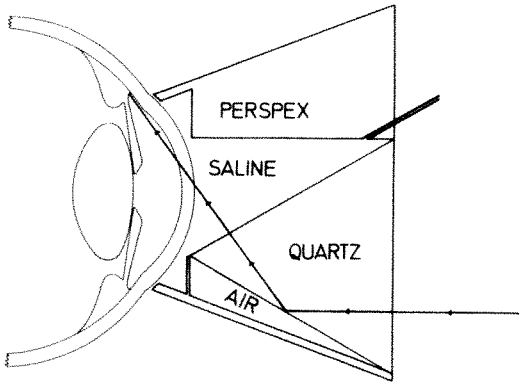


Figure (Wheeler and Bass). Schematic diagram of the gonioscopy lens.

rectangular face flush with the end face of the cylinder and with its apex on the bottom of the slot 0.36 cm from the axis of the cylinder. This is the optimum position of the reflecting plane that Becker[12] recommended. Any excess acrylic plastic is removed by uniformly tapering the cylinder over an axial length of 2.06 cm into the frustum of a cone from a diameter of 2.90 cm at the end face to a diameter of 1.73 cm at the corneal face. The corneal cavity is formed by turning out the acrylic plastic over a diameter of 1.33 cm to a depth of 0.31 cm, leaving a ledge of acrylic plastic 0.10 cm thick on which the prism apex stands and a corneal rim 0.20 cm thick. This rim is shaped to match the average corneoscleral limbus and highly polished so as not to scratch the cornea. The saline reservoir, which includes the corneal cavity, is completed by breaking through and removing part of the ledge with a Swiss file while retaining a ledge sufficient for the prism to act as a water-tight side to the reservoir. A hole to accommodate a hypodermic needle is drilled through the acrylic plastic from the front face to the reservoir in order to admit saline solution from a syringe via a plastic pipe. Finally the prism and needle are fixed in position with Araldite.

The energy losses by reflection are acceptable even in the absence of antireflection coatings, amounting to 4% at the front face and 0.4% at the quartz-to-saline interface. This front-face reflection does not present a hazard to the surgeon because the associated laser equipment incorporates a safety shutter that occludes his view when the laser is fired. The weaker reflection generates, by further reflection at the front face, a beam directed toward the eye with an intensity 2×10^{-4} times that of the primary beam. However, this reflected beam is colinear with the primary beam, even when the beam does not traverse the prism normal to its faces. In practice the lens is initially held horizontally and filled with saline solution. The patient's head is inclined horizontally and the lens applied to the corneoscleral limbus; saline solution is admitted from the syringe until all air bubbles are vented around the rim. The head is then raised to the upright position with the lens held lightly against the eye to retain the saline solution. Clinical trials of this lens in conjunction with the als of this lens in conjunction with the dye laser generating a 100-kW pulse have shown that a prism of BK7 glass may be used instead of quartz without incurring radiation damage.

ACKNOWLEDGMENT

Mr. L. Yerlett assisted in the design of this lens.

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OPHTHALMIC MINIATURE

Oh, this terrible gift of second-sight that comes to some of us when we begin to look through the silvered rings of the arcus senilis.

Oliver Wendell Holmes, *The Autocrat of the Breakfast-Table*
Boston, Nov. 1, 1858

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A GLIMMER OF HOPE

The notoriously dismal visual results in spite of successful surgery in patients with unilateral congenital cataract have always been frustrating for the surgeon and the patient's parents. Until Scheie reintroduced the aspiration method into modern ophthalmology, the complications associated with the older surgical methods, such as a discission followed by lavage and linear or intracapsular extractions, may have contributed to the unfavorable results. However, even after these techniques were replaced with single-stage and minimally traumatic lens aspiration, the visual results remained disappointing despite contact lens correction of the aphakic refractive error and long-term occlusion of the normal eye. Indeed, whether surgery for a unilateral congenital cataract in a patient 3 months old or older is justified at all has become a philosophic rather than a medical question. With the advent of ultrasonography, we no longer need to remove a cataract to learn whether an eye harbors a tumor in the posterior pole and it is debatable to what degree a patient with good vision in one eye benefits functionally by having visual acuity in his other eye improved, for example, from light perception to 6/120 (20/400) by cataract surgery. What causes severe reduction of visual acuity despite clear ocular media?

Various investigators have suspected that associated ocular anomalies, such as incomplete or abnormal retinal development, aplasia of the macula, and congenital or organic amblyopia, contribute to the dismal visual results. However, histologic or other data to confirm these suspicions have never been presented. Experiments in newborn kittens and monkeys, however, have shown that unilateral stimulus deprivation causes a predictable set of behavioral, histologic, and electrophysiologic disturbances in the afferent visual apparatus (visual deprivation syndrome). On the basis of this work, it was reasonable to assume that amblyopia

from stimulus deprivation, caused preoperatively by the opaque lens and postoperatively by the uncorrected aphakic refractive error, is responsible for the poor vision in these children rather than some obscure associated ocular anomalies. If this assumption is correct, amblyopia may be preventable by aspirating a unilateral congenital cataract that is not accompanied by other ocular anomalies within the first days of life, followed by almost immediate contact-lens correction and occlusion treatment of the sound eye.

Alas, despite our constant pleas to pediatricians and neonatologists to refer infants with congenital cataracts immediately after birth, we continued to examine these children for the first time when they are 3 to 6 months old. It is not at all uncommon, although it is quite disheartening, for parents of infants in whom a unilateral cataract was diagnosed at birth to be advised by some ophthalmologists to wait for surgery until the child is 3 to 6 months old. Evidently, theoretical considerations, results from experiments in animal models, and even isolated published reports of visual recovery after unilateral congenital cataracts have not been sufficiently convincing to turn the tide in favor of neonatal treatment of this condition. What was needed was a clinical study to show that neonatal surgery, followed by immediate treatment of the amblyopia, makes visual rehabilitation possible. The article by Beller and associates, in this issue of THE JOURNAL is such a study, and I expect this paper to have considerable impact on the clinical management of unilateral congenital cataracts in years to come. The data of Beller and his co-workers should also encourage us to renew our efforts to have pediatricians and neonatologists in our community screen all newborns for cataracts and refer such cases as emergencies to qualified ophthalmic surgeons.

Although Beller and associates offer a glimmer of hope with respect to the visual prognosis in a disorder formerly thought by most to be incurable, we must not for a moment minimize the extraordinary difficulties associated with the postoperative management of these patients. Treatment must begin at birth and last through the entire childhood. The necessity of frequent adjustment and replacement of the contact lens, the careful monitoring of visual acuity of both the operated upon and the occluded eye, and the extraordinary conscientiousness and patience demanded of the parents of such children and of the physician during many years of follow-up care clearly point out that this therapy is not for every patient, nor should every ophthalmologist assume such a responsibility.

There remain many unsolved questions regarding the postoperative management of these infants. For instance, how long can one safely occlude the sound eye in newborns before visual deprivation amblyopia occurs in the patched eye? Is it advisable to patch both eyes during certain phases of the treatment (that is, while waiting for the contact lens) to avoid unequal visual input that contributes to the development of amblyopia? Clearly, these and many other problems must be addressed by future studies. Physicians need to be informed about special pediatric ophthalmology referral centers where neonatal surgery can be performed without risk to the general health of the infant and where the expertise in fitting infants with contact lenses and the laboratories equipped to measure the visual acuity of infants by behavioral or electrophysiologic methods are available. The work by Beller and associates is decidedly a step in the right direction.

GUNTER K. VON NOORDEN

BOOK REVIEWS

Ophthalmology in Internal Medicine. By Lee C. Chumbley. Philadelphia, W. B. Saunders Co., 1981. Hardcover, 280 pages, index, 199 black and white illustrations. $30

The author of this book tells us in his introduction that he was trained first in internal medicine and then in ophthalmology. His formal training has been augmented by service in a U.S. naval hospital, in Rhodesia, and at the Mayo Clinic where he is a member of the ophthalmology department. He has also spent a large amount of time in the medical library.

The author groups his subjects by the classic areas of internal medicine: Endocrine, Metabolic, Cardio-Vascular, Rheumatology, Infections, Hematology-Oncology, Dermatology, Gastrointestinal-Nutritional, Renal, Pulmonary, and Neurology. There are excellent descriptions of the empiric data on and the current theories of many diseases.

The section on the biochemical aspects of diabetes mellitus is excellent and offers an explanation of why poorly controlled diabetes results in vascular disease.

Although the book is excellent on internal medicine, ophthalmologists will be disappointed with the fine nuances of ophthalmic detail, the minutiae of external and slit-lamp examinations, and the ophthalmoscopic findings. Many of the black and white photographs, which have been converted from color transparencies, do not show the desired detail.

This book is the most up-to-date book of its type. It is more concise than Walsh and Hoyt, and broader in scope than Ryan and Smith or Masoulf. It has the advantage of being written by only one person. Furthermore, the references are superb. The ophthalmologist will find the book a useful guide to his patients' systemic diseases and the references will give him secondary sources of information. The internist will read it to understand what his ophthalmologist colleagues are talking about.

DAVID L. KNOX

Goniodysgenesis. By Tord Jerndal, Hans Arne Hansson, and Bill Anders. Copenhagen, Scriptor, 1978. Hardcover, 212 pages, references, index, 131 figures.

This book is of interest mainly to the scholar who wishes to consider novel perspectives and hypotheses while trying to understand the basic nature of the trabecular meshwork abnormality in glaucoma.

The term "goniodysgenesis" refers to an abnormal embryonic development of the anterior chamber angle, and particularly of an endothelial membrane that the authors believe normally lines the angle during one stage of development and subsequently becomes fenestrated to produce the trabecular meshwork. The authors believe that goniodysgenesis accounts for almost all the glaucomas—not only congenital glaucomas (including Reiger's anomaly, aniridia, ectopia of the lens, primarily infantile glaucoma, and others), but also diverse adult glaucomas such as pseudoexfoliation, glaucomatocyclitic crisis, corticosteroid-induced glaucoma, pigmentary glaucoma, some cases of chronic angle-closure glaucoma, and, most importantly, almost all cases of chronic primary open-angle glaucoma.

The evidence offered here includes their own careful gonioscopic observations, family pedigrees suggesting an autosomal dominant inheritance, and a number of histopathologic specimens, examined mainly by scanning electron microscopy. They recognize definite abnormalities in the configuration of the

anterior chamber angle structures, although they are sometimes subtle, in all these types of glaucoma and hypothesize that the age at onset and severity of the galucoma depend on the degree of the abnormality and the degree to which the outflow channels are challenged with corticosteroids, pseudoexfoliation material, pigment, and so on. They fit many of the clinical manifestations and responses to medical and surgical treatment into their theory that goniodysgenesis is at fault in these types of glaucoma.

The question is whether this unifying thesis will hold up when others try to confirm the findings and conclusions of the authors by their own observations. Some will reject this "new perspective" outright, because it is certainly outside the mainstream of current thinking. In any event, only a few will accept the notion without studying the matter further themselves. If you are interested, read the book and make up your own mind.

DOUGLAS R. ANDERSON

Hemorrhages in Ophthalamology. By Maurizio Pandolfi. Stuttgart, Georg Thieme Publishers, 1979. Softcover, 84 pages, references, index, 61 black and white illustrations. $20

This unpretentious book packs a great deal of information into relatively few pages. The author begins with a review of the mechanisms regulating hemostasis. He discusses the three basic mechanisms active in the arrest of bleeding. These are the primary vascular response, the humoral response in which there is activation of the coagulation system, and the anatomic repair of the damaged vessel wall. This is logically followed by a discussion of disorders resulting from alterations in the basic hemostatic mechanism. This section also includes a discussion of drugs that can alter the basic hemostatic mechanisms.

A section on laboratory tests reviews some of the most common coagulation and fibrinolytic tests, the principles of the tests, their indications, and the information these tests can be expected to give. Those physicians who have been out of medical school for some time will find these two sections alone worth the price of the book.

The section on ocular hemorrhages deals with hemorrhage into the anterior chamber, the vitreous, retina, and choroid and discusses the hemostatic mechanism, or the lack thereof, associated with various disorders.

The section on hemorrhages during ocular surgery begins, "The best screening method of a bleeding tendency is a detailed bleeding history." This is perhaps the most important sentence in the book and one that should be remembered. A short section on the main hemorrhagic disorders and their management in ocular surgery follows.

The last section deals with retinal thromboembolism and diabetic retinopathy and should be of interest to all ophthalmologists.

The references are good and provide a starting place for those interested in pursuing the various subjects in more detail.

In general, this is a good little book that can be recommended to those interested in learning about the subject of hemorrhagic disorders in ophthalmology. The book initiates us into the subject in a way that is comprehensible and sensible. The book can be highly recommended.

J. GRAHAM DOBBIE

Manual for Eye Examination and Diagnosis. By Mark W. Leitman, Samuel Gartner, and Paul Henkind. Oradell, New Jersey, Medical Economics Co.,

1981. Softcover, 75 pages, 75 black and white illustrations, 8 color figures. $8.95

Drs. Leitman, Gartner, and Henkind have written a short, inexpensive manual on eye examination and diagnosis that is particularly valuable to the medical student during an ophthalmology rotation. The book is divided into four sections: History-Taking, Visual Acuity Determination, Examination, and Differential Diagnosis.

The authors give a cogent sketch of how the ocular history should be elicited from the patient. The next section presents the common parlance of visual acuity and a succinct explanation of common refractive errors and techniques of refraction. The examination section makes up the largest part of the book, but can be mastered quickly. It covers basic ophthalmic terminology and external examination. Additionally, it briefly discusses strabismus, glaucoma, visual fields, and ophthalmoscopy. The text is enhanced by many clear and simple diagrams. At the end of the text there are tables which cover the differential diagnosis of headache, red eye, and common ocular complaints and their causes. Additional tables on ophthalmic signs with systemic implications, such as the diagnosis and pharmacology of Horner's syndrome, would be useful. Finally, there is an excellent basic ophthalmology book list that serves as an ideal starting point for a medical student interested in the field.

JOSEPH P. KIERNAN

Handbook of Ophthalmology for Developing Countries, 2nd ed. By Geoffrey G. Bisley. Nairobi, Oxford University Press, 1981. Softcover, 157 pages, index, 50 black and white illustrations. $13.95

This small handbook is geared entirely to developing nations of the Third World, where conditions are different from those in our country. Specific diseases affecting the eyes that receive no attention in most of our handbooks, but which are not uncommon in developing countries, are covered concisely. Onchocerciasis and leprosy are seldom found in developed areas.

The book was written for undergraduate medical students in developing countries to increase their ability to diagnose, prevent, and treat the many ocular conditions in these areas, which have the highest rates of blindness in the world. Ophthalmologists and other personnel can make good use of this book in providing primary eye care to supplement primary health-care programs.

This book will be useful not only in Kenya and other East African countries, where the author performed outstanding work for 33 years, but also in other countries struggling to reduce blindness. The author developed a system of training ophthalmic clinical assistants that is now the first line of defense against blindness in other areas of the world and not just in Africa.

The book is a must for ophthalmologists who train paramedical personnel. The information on the practice of ophthalmology in rural areas emphasizes the need and the importance of mobile eye units in taking eye care directly to the poorest of the poor in small villages. The book is written in simple language and offers many practical details about simple methods of treatment and prevention.

The ophthalmologist content with the security of a big-city practice might develop some concern for his colleagues helping in the Third World if he read this excellent handbook.

J. H. KING, JR.

SYMPOSIA

Advances in Ophthalmology. Volume 42. Edited by M. J. Roper-Hall, H. Sautter, and E. B. Streiff. Basel, S. Karger AG Publishers, 1980. Hardcover, 164 pages, index, 38 black and white figures, 16 tables. $58.75

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Principles and Practice of Ophthalmology. Volumes I, II, and III. Edited by Gholam A. Peyman, Donald R. Sanders, and Morton F. Goldberg. Philadelphia, W. B. Saunders Co., 1980. Hardcover, 881 pages, index, 694 illustrations, 6 color plates. $250

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Current Eye Research, Vol. 1, No. 1. London, IRL Press Ltd., 1981. Published monthly. Annual subscription, $200.

Volume 1, No. 1 of this new periodical was received on March 28. The lead article was submitted on Jan. 5 and accepted for publication on Feb. 20, 1981. Another article was submitted on Feb. 3 and accepted on Feb. 19. The material is directly reproduced by photo-offset printing from typed pages. Thus, this new journal publishes basic and clinical research papers without delay.

Two articles were prepared on word processors so that the right and left sides of each column are parallel (justified). The remaining articles were neatly typed on different brands of typewriters with various type faces. In some articles the references are given completely; in others only the authors and the journal are given.

The first issue provides seven original articles and one short communication. All begin on the right side of the page, so that four of the 56 pages are blank.

The subscription price to institutions is $200; an individual subscription is $100 if the institution already subscribes. This brings the institutional cost to $0.32 a page, in contrast to "Investigative Ophthalmology and Visual Science" which costs about $0.05 a page; the "Archives of Ophthalmology" costs about $0.025 a page and THE JOURNAL currently costs a little over $0.01 per editorial page.

FRANK W. NEWELL

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Ophthalmologica

STATIC PERIMETRIC AND AMSLER CHART CHANGES IN PATIENTS WITH IDIOPATHIC CENTRAL SEROUS RETINOPATHY. Natsikos, V. E., and Hart, J. C. D. (Dept. Ophthalmol., Univ. Bristol, Bristol Eye Hosp., Lower Maudlin St., Bristol BS 1 2LX, England). Acta Ophthalmol. 58:908, 1980.

A group of patients with idiopathic central serous retinopathy were examined by static perimetry and with the Amsler chart. All the patients showed abnormal responses to both forms of examination during the acute phase but, after the edema fluid had absorbed, 63% of the patients retained abnormal static profiles while 81% recorded defects on Amsler charting. This indicates that Amsler testing carried out quickly without recourse to any special technical expertise is as good as static perimetry for evaluating central field disturbances and superior in assessing residual field defects. (6 figures, 9 references)—David Shoch

COMPUTERIZED VISUAL FIELD SCREENING IN THE MANAGEMENT OF PATIENTS WITH OCULAR HYPERTENSION. Dyster-Aas, K., Heijl, A., and Lundqvist, L. (Dept. Ophthalmol., Univ. Lund, S-214 01, Malmö, Sweden). Acta Ophthalmol. 58:918, 1980.

Visual field testing with a fully automatic computerized perimeter employing a supraliminal screening test procedure was used to examine 1,013 eyes with ocular hypertension in which earlier routine perimetry (kinetic and static) on the Goldmann perimeter had yielded a normal result. The automatic screening was repeated if positive, and manual control perimetry was used in order to confirm or reject identified field defects. This procedure disclosed field defects that could be confirmed with both automatic and manual perimetry in 3.6% of the eyes. In a control group, the incidence of field defects found at manual perimetry during the same time interval was calculated at 0.4%. Thus, automatic screening disclosed several times more field defects than manual routine perimetry. Eyes in which repeated automatic screening had indicated defects that manual control perimetry failed to confirm showed a high percentage of field loss at later follow-up. The results are discussed, and the conclusion is drawn that automatic screening is clearly superior to manual routine perimetry used at present. The most practical solution in many eye departments would be to use a computerized perimeter for the visual field screening of glaucoma suspects. (4 figures, 14 references)—David Shoch

FILTRATION BLEBS IN CORNEOSCLERAL WOUNDS SUTURED WITH DEXON 7-0 AND DEXON 8-0. Kleener, J. (Søndersø Park 54, 3500 Vaerløse, Denmark). Acta Ophthalmol. 58:957, 1980.

In a retrospective investigation the incidence of postoperative subconjunctival filtration with bleb formation was studied in 112 eyes which had undergone cataract surgery with a corneoscleral incision sutured with braided polyglycolic acid sutures (Dexon). In 61 consecutive cataract operations sutured with Dexon 8-0, there were filtration blebs in 24 (39.3%) eyes, and in 51 consecutive cataract operations sutured with Dexon 7-0 there were filtration blebs in 11 (21.6%) eyes. Although there is a difference between the two materials, this is not statistically significant. (4 tables, 13 references)—Author's abstract

Albrecht von Graefes Archiv für Klinische und Experimentelle Ophthalmologie

IDENTIFICATION OF A BASEMENT MEMBRANE PROTEOGLYCAN IN EXFOLIATION MATERIAL. Harnisch, J. P., Barrach,

H. J., Hassell, J. R., and Sinha, P. K. (Dept. Ophthalmol., Klinikum Steglitz, Freie Universität Berlin, Hindenburgdamm 30, D-1000 Berlin 45). Albrecht von Graefes Arch. Klin. Ophthalmol. 215:273, 1981.

The etiology of the exfoliation syndrome described more than 60 years ago still remains an enigma. The material deposited appears to be a protein and the staining pattern suggests that it is probably a glycoprotein or mucoprotein. The question remains whether this is a product of the lens capsule or a more generalized secretion of the eye. The authors have used an indirect immunoperoxidase technique to examine the lenses from three patients with the exfoliation syndrome. They demonstrate that the fibrils so typically found in this disease certainly contain basement membrane proteoglycans. This supports the hypothesis that this disease is caused by a disturbance in the biosynthesis of the basement membrane. (6 figures, 11 references)—David Shoch

Archives of Dermatology

PREMATURE CATARACTS IN A FAMILY WITH HIDROTIC ECTODERMAL DYSPLASIA. Hazen, P. G., Zamora, I., Bruner, W. E., and Muir, A. (Dept. Dermatol., Univ. Hosp. of Cleveland, 2065 Adelbert Rd., Cleveland, OH 44106). Arch. Dermatol. 116:1385, 1980.

Ectodermal dysplasias can generally be divided into two major categories, the hidrotic and the anhidrotic forms. In both types there is alopecia and dental anomalies. There are some differences in nail dystrophy but the major difference is inheritance, the hidrotic form being inherited as an autosomal dominant and the anhidrotic as an x-chromosome-linked recessive. In general, ocular findings are uncommon in either form. The authors describe a family of nine individuals in three generations, five of whom were affected with hidrotic ectodermal dysplasia. Of these five, two had already undergone cataract surgery, a third had obvious clinical cataracts, and a fourth had opacities noted only by slit-lamp examination. This is the first report of a family in which bilateral early onset cataracts appear to be inherited with hidrotic ectodermal dysplasia. (3 figures, 3 tables, 16 references)—David Shoch

Archives of Neurology

VISUAL FUNCTION OF THE EXTRAGENICULO-CALCARINE SYSTEM IN MAN: RELATIONSHIP TO CORTICAL BLINDNESS. Celesia, G. G., Archer, C. R., Kuroiwa, Y., and Goldfader, P. R. (Dept. Neurol., William S. Middleton Mem. Vet. Hosp., 2500 Overlook Terr., Madison, WI 53705). Arch. Neurol. 37:704, 1980.

A 72-year-old woman had a complete loss of area 17 function bilaterally and, although she maintained that she could see shadows, formal testing showed that she could not distinguish light from darkness and could not identify objects or detect shadows. Re-evaluation after the two years showed that the patient depended entirely on extravisual clues to cope with her environment. She could not detect a strong light flashed directly into her face and displayed no optokinetic nystagmus to a rotating drum. However, the electroretinography and visual-evoked potentials were normal. Computerized tomography showed extensive destruction of the occipital lobes involving bilateral areas 17 and part of area 18 on the left. There was sparing of the lateral part of the occipital lobes, including part of area 18 bilaterally and all of area 19. The authors conclude that in the presence of bilateral destruction of area 17, visual-evoked potentials are mediated by extrageniculocalcarine pathways to secondary visual cortices, but this system is not capable of providing conscious visual perception in humans. (3 figures, 37 references)—David Shoch

A "POSSIBLE" OPTIC NERVE MENINGIOMA. Ebers, G. C., Girvin, J. P., and Canny, C. B. (Dept. Clin. Neurol. Sci., Univ. Hosp., P. O. Box 5339, Postal Station A, London, Ontario N6A 5A5). Arch. Neurol. 37:781, 1980.

A 58-year-old woman had progressive visual failure in her left eye over a two-year period. All laboratory tests were negative, including cerebrospinal fluid examination.

X-rays, including tomography of the sella and optic canals, were similarly normal. The visual-evoked response was absent on the left. Carotid angiography was normal and computed tomographic scanning showed a minor asymmetry of the optic nerves that was not believed to be diagnostic. This was repeated with contrast material (metrizamide). The study indicated that the dye did travel down the optic nerve, arguing against the presence of the tumor. The patient was given prednisone for two weeks with some improvement, but as the corticosteroid was discontinued the visual acuity dropped to finger counting and an absolute defect was noted in the temporal field. Because of this, the orbit was explored via a Krönlein orbitotomy and a tumor was found along the course of the optic nerve. This was removed without vascular damage to the nerve. By the fourth postoperative day, visual acuity improved to 6/9 (20/30) and the visual field to normal. An optic nerve sheath meningioma has always been a great problem and has been described as "the impossible meningioma." In most of the cases reported, there has been no return of vision following surgical excision but the authors feel that in a rare case a full return can be obtained, as was done here. In certain cases, in the presence of continuing visual loss, surgical exploration would seem to be indicated. (3 figures, 22 references)—David Shoch

British Journal of Ophthalmology

PENETRATING EYE INJURIES: A HISTOPATHOLOGICAL REVIEW. Winthrop, S. R., Cleary, P. E., Minckler, D. S., and Ryan, S. J. (Dept. Ophthalmol., Univ. Southern California, Estelle Doheny Eye Found., 1355 San Pablo St., Los Angeles, CA 90033). Br. J. Ophthalmol. 64:809, 1980.

The authors report a high incidence of retinal detachment in eyes with severe penetrating injuries. The retina was detached in 32 out of 34 eyes examined, with 27 having evidence of traction on the retina. These eyes were characterized histologically by intraocular cellular proliferation producing cyclitic, epiretinal, and retroretinal membranes. Intraocular cellular proliferation was discernible or established within one week of injury and typically resulted in a cyclitic membrane at about six weeks. Epiretinal and retroretinal membranes were found between one and two weeks after injury in eyes with a detached retina. The results indicate that a damaged lens, the admixture of lens material and vitreous, and the presence of vitreous hemorrhage were all factors promoting intravitreal fibroblastic proliferation. Vitreous surgery may be a rational method of treatment for these severely injured eyes by removing the stimulus and vitreous scaffold for intravitreal fibroblastic proliferation. From this series it would appear that vitrectomy should not be delayed beyond the second week after injury, by which time massive cellular ingrowth may already be underway. (9 figures, 1 table, 22 references)—E. Lee Stock

INTERACTION OF TIMOLOL AND ADRENALINE. Öhrström, A. and Kättström, O. (Dept. Ophthalmol., Köping Hosp., S-731 01 Köping, Sweden). Br. J. Ophthalmol. 65:53, 1981.

In a prospective double-blind randomized trial, the interaction of timolol and adrenaline was studied in 20 patients with open-angle glaucoma and exfoliative glaucoma. After pretreatment for at least two weeks with timolol eyedrops, adrenaline was added in a double-blind manner, and the intraocular pressure was recorded during four days. The study showed a significantly additive hypotensive effect of adrenaline, which was more pronounced in patients with exfoliative glaucoma. (1 figure, 2 tables, 20 references)—Authors' abstract

British Medical Journal

CATARACT FORMATION IN ANOREXIA NERVOSA. Archer, A. G. (Ipswich Hosp., Ipswich IP4 5 PD England). Br. Med. J. 282:274, 1981.

Three patients in their late 20's or early 30's suffering from anorexia nervosa had concomitant cataract formation requiring cataract extraction. A transition from normal to low concentrations of serum potassium or magnesium is suggested as necessary to induce these changes in the lens. (1 table, 5 references)—E. Lee Stock

Cancer

TRANSIENT CORTICAL BLINDNESS SECONDARY TO VINCRISTINE THERAPY IN CHILDHOOD MALIGNANCIES. Byrd, R. L., Rohrbaugh, T. M., Raney, R. B., and Norris, D. G. (Div. Oncol., Children's Hosp. of Philadelphia, 34th St. and Civic Ctr. Blvd., Philadelphia, PA 19104). Cancer 47:37, 1981.

Three children with malignancies developed severe neurotoxicity, including transient cortical blindness, after chemotherapy treatment. Two of the children had non-Hodgkin's lymphoma and the third a rhabdomyosarcoma. The only drug in common was vincristine sulfate. All have completely recovered, although one child who was rechallenged with vincristine had a cardiorespiratory arrest, increasing neurotoxicity, and recurrence of blindness. Transient cortical blindness has not previously been attributed to vincristine sulfate therapy, but must be considered as a possible complication of treatment with this agent. (28 references)—David Shoch

Clinical Endocrinology

ORBITAL PSEUDOTUMOUR AND LACRIMAL INVOLVEMENT DEVELOPING IN A PATIENT WITH RIEDEL'S STRUMA RECEIVING STEROIDS. Wilmshurst, P., Melsom, R., and Gostelow, B. (Dept. Cardiol., St. Thomas's Hosp., London SE1 7EH). Clin. Endocrinol. 14:63, 1981.

Riedel's struma is a systemic fibrotic disease, sometimes called multifocal fibrosclerosis, that commonly involves the thyroid gland. Additionally, it also involves the tissues surrounding the thyroid gland. There is also sometimes an associated retroperitoneal fibrosis and mediastinal fibrosis. The patient described here was euthyroid and developed a neck swelling with tenderness. No ocular signs were present. During the next few weeks she became clinically hypothyroid and open biopsy showed that the entire gland had been replaced by white fibrous tissue. About two years later, she developed a right proptosis with a right superior rectus muscle palsy and a hard lacrimal gland. There were intermittent episodes of painful swelling of the eye with periorbital edema, conjunctival erythema, and chemosis. A lacrimal gland biopsy was done and the specimen was found to consist of dense hyalin fibrous tissue infiltrated by lymphocytes and plasma cells identical to the appearance of the thyroid biopsy specimen done two years previously. This case represents the fifth report of Riedel's struma of the orbit. The other four patients also had histories of involvement of the thyroid gland. (2 figures, 9 references)—David Shoch

Investigative Ophthalmology and Visual Science

GRAVES' OPHTHALMOPATHY—IMMUNOLOGIC PARAMETERS RELATED TO CORTICOSTEROID THERAPY. Sergott, R. C., Felberg, N. T., Savino, P. J., Blizzard, J. J., and Schatz, N. J. (Wills Eye Hosp., Neuro-ophthalmol. Unit, 9th & Walnut St., Philadelphia, PA 19107). Invest. Ophthalmol. Vis. Sci. 20:173, 1981.

Patients with Graves' ophthalmopathy appear to fall into two groups: some respond to treatments with systemic corticosteroids and some do not. In an attempt to find immunologic distinctions between these two groups, the authors have evaluated several tests in both groups of patients. In 17 patients undergoing successful prednisone therapy, there was both a percentage and an absolute increase in the numbers of thymus-derived active and total rosette-forming cells (A-RFC and T-RFC). In the second group of five patients, in whom was resistance to corticosteroids, the A-RFC decreased when treated with oral prednisone and the T-RFC were unchanged. Further, corticosteroids increased the lymphoblast transformation response to phytohemagglutinin in the corticosteroid-responsive patients, whereas the corticosteroids decreased the lymphoblast transformation response in corticosteroid-resistent patients. The findings may allow the clinician to predict which patients with Graves' ophthalmopathy are likely to benefit from corticosteroid therapy and which patients may be managed better by other methods. (6 figures, 5 tables, 24 references)—David Shoch

SPECIFIC DEFICITS OF FLICKER SENSITIVITY IN GLAUCOMA AND OCULAR HYPERTENSION. Tyler, C. W. (Smith-Kettlewell Inst. of Vis. Sci., 2232 Webster St., San Francisco, CA 94115). Invest. Ophthalmol. Vis. Sci. 20:204, 1981.

Temporal modulation sensitivity for a 5 degree flickering field was measured in central and peripheral retina for 46 eyes with glaucoma and ocular hypertension. Prior to showing any visual field loss by conventional perimetry, 90% of patients exhibited significant losses in sensitivity around a specific frequency of about 30 to 40 Hz. Low-frequency sensitivity and critical flicker frequency were often unaffected by such losses. The central field showed almost as pronounced a loss as 20 degrees in the periphery. (5 figures, 3 tables, 9 references)—Author's abstract

Laryngoscope

ANTRAL-ETHMOIDAL DECOMPRESSION OF THE ORBIT IN GRAVES' DISEASE: TEN-YEAR EXPERIENCE. Calcaterra, T. C., and Thompson, J. W. (10833 LeConte, Los Angeles, CA 90024). Laryngoscope 90:1941, 1980.

The basis for this paper is a group of 104 patients in whom antral-ethmoidal decompressions were performed for Graves' disease. The authors review the various modalities available for treatment of patients whose vision is threatened by the increased volume of orbital contents. The corticosteroids tend to have only a transient ameliorative effect and there is a threat of unpleasant side effects. Decompression from above has some small risk of intracranial complications and, additionally, pulsation transmitted to the globe from the intracranial vasculature may be annoying afterwards. A lateral decompression requires a facial incision extending posteriorly from the lateral canthus. For all these reasons the authors feel that the most effective and least damaging procedure for expansion of the orbital volume is a antral-ethmoidal decompression. They note that diplopia is not improved by this procedure and may in fact be aggravated. (3 figures, 14 references)—David Shoch

Neurology

TRANSIENT OCULOMOTOR PARALYSIS IN PSEUDOTUMOR CEREBRI. McCammon, A., Kaufman, H. H., and Sears, E. S. (Dept. Neurol., MSMB 7.044, 6431 Fannin St., Houston, TX 77030). Neurology 31:182, 1981.

A 23-year-old obese woman developed bifrontal headaches and transient visual obscuration. Examination disclosed bilateral papilledema and a diagnosis of pseudotumor cerebri was made. She was treated with prednisone and acetazolamide and her findings resolved. A year later she again had some momentary loss of consciousness and papilledema was again present. She was well for two years but some three years after her initial episode she was readmitted to the hospital with headaches and mental torpor and with markedly increased cerebrospinal fluid pressure. During the course of the investigation, she developed a complete painless left oculomotor palsy. Three-vessel angiography disclosed no evidence of herniation, aneurysms, or a mass lesion. The oculomotor paralysis gradually resolved in 14 days and a year later there was no evidence of papilledema, the visual acuity was normal, and there was no residual anisocoria. Sixth nerve palsy has been reported in from 10% to 40% of patients with pseudotumor cerebri, but third nerve palsy has not been reported previously. This may be a nonspecific sign of increased intracranial pressure. (28 references)—David Shoch

CERVICAL INTERNAL CAROTID ARTERY ANEURYSM WITH CALCIFIC EMBOLISM TO THE RETINA. Mokri, B., and Piepgras, D. G. (Mayo Clin., Rochester, MN 55901). Neurology 31:211, 1981.

It is a generally accepted aphorism that cholesterol emboli originate in atherosclerotic plaques in the internal carotid artery, whereas calcific emboli tend to come from cardiac valves. The patient described here had a sudden loss of vision in the left eye. Examination showed a large white calcific plaque in the inferior temporal artery of the left retina. A vague swelling on the left side of the neck at about the angle of the mandible was noted. Retinal artery pressures were the same in the two eyes. Transfemoral bilateral carotid angi-

ography showed an aneurysm arising from the left internal carotid artery several centimeters above the origin of this vessel. The aneurysm had an obvious calcific shell on a plain roentgenogram. The aneurysm was excised at surgery and the stumps of the internal carotid anastomosed. A second aneurysmal dilation was reinforced by wrapping with Teflon. Histologic examination of the aneurysm that was removed disclosed atherosclerotic changes associated with significant deposits of lipid and calcium. (2 figures, 43 references)—David Shoch

New England Journal of Medicine

PRIMARY EMPTY SELLA AND RIEGER'S ANOMALY OF THE ANTERIOR CHAMBER OF THE EYE: A FAMILIAL SYNDROME. Kleinmann, R. E., Kazarian, E. L., Raptopoulos, V. and Braverman, L. E. (Univ. Massachusetts Med. School, 55 Lake Ave., North, Worcester, MA 01605). N. Engl. J. Med. 304:90, 1981.

A kindred with seven members had Rieger's anomaly as well as empty sella syndrome. The authors speculate that in this anomaly the mesoderm that forms the anterior chamber of the eye and the dura is defective during fetal development, causing hypoplasia of the iris and the sellar diaphragm. (1 figure, 1 table, 25 references)—E. Lee Stock

Ophthalmic Surgery

TREATMENT OF DISTICHIASIS WITH CRYOTHERAPY. Frueh, B. R. (Dept. Ophthalmol., Univ. Michigan, 1000 Wall St., Suite 2549, Turner Bldg., Ann Arbor, MI 48109). Ophthalmic Surg. 12:100, 1981.

The surgical treatment of distichiasis has frequently resulted in disfigurement of the treated eyelid. Since the follicles of the aberrant eyelashes in eyelids with distichiasis are placed more posteriorly than the follicles of normal eyelashes, it is theoretically possible to destroy the follicles of the aberrant eyelashes without destroying the follicles of the normally placed eyelashes if transconjunctival cooling creates a sufficient temperature gradient from the conjunctiva to the anterior tarsal surface. The author uses nitrous oxide with a boiling point of −88.5 C and employs a freeze-thaw-freeze cycle. The first freezing lasts 60 seconds and the second 45 seconds. The treatment seems promising, but specific details of treatment regimen need to be investigated. (1 figure, 2 tables, 14 references)—David Shoch

Pediatrics

UNUSUAL EYE ABNORMALITIES ASSOCIATED WITH CONGENITAL CYTOMEGALOVIRUS INFECTION. Frenkel, L. D., Keys, M. P., Hefferen, S. J., Rola-Pleszczynski, M., and Bellanti, J. A. (Medical College of Ohio at Toledo, CS 10008, Toledo, OH 43699). Pediatrics 66:763, 1980.

The seven children described here had a variety of ocular abnormalities, including optic atrophy in four, anophthalmia in two, Peters' anomaly in one, and retinal pigment abnormalities in three. This very high incidence of ocular abnormality in children with congenital cytomegalovirus infection suggests that this viral infection be searched for in all children who have ocular abnormalities at birth. (1 table, 26 references)—David Shoch

NEWS ITEMS

EDITED BY THOMAS CHALKLEY, M.D.

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For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least six months before the date of occurrence.

INTERNATIONAL SYMPOSIUM ON GENETICS IN OPHTHALMOLOGY

An international symposium on genetics in ophthalmology will be held in Jerusalem, Israel, Sept. 20–23, 1981. For further information, write Prof. Saul Merin, Chairman, Organizing Committee, P.O. Box 983, Jerusalem, Israel 91009.

INTERNATIONAL SOCIETY OF GEOGRAPHICAL OPHTHALMOLOGY: SEVENTH INTERNATIONAL CONGRESS

The International Society of Geographical Ophthalmology will meet Nov. 7–10, 1982, in Asilomar, California. For further information, write William Miller, M.D., Rm. 418, 4141 Geary, San Francisco, CA 94118.

XV MEXICAN CONGRESS OF OPHTHALMOLOGY

The XV Mexican Congress of Ophthalmology will be held in Queretaro, Mexico, May 2–7, 1982. For further information, write Gil Villanueva-Diaz, M.D., Atlanta 157, Ciudad de los Deportes, Mexico 18, D.F.

UNIVERSITY OF WESTERN ONTARIO: ANNUAL CLINICAL DAY

The University of Western Ontario will hold its annual clinical day on Sept. 11, 1981. For further information, write Dr. A. J. McKinna, Chairman, Dept. of Ophthalmology, University Hospital, London, Ontario, Canada N6A 5A5.

SALLY LETSON SYMPOSIUM

The Sally Letson symposium on strabismus will be held Sept. 24 and 25, 1981, in Ottawa, Canada. For further information, write Dr. A. G. Watson, 267 O'Connor St., Suite 406, Ottawa, Canada K2P 1V3.

BASCOM PALMER EYE INSTITUTE: NEURO-OPHTHALMOLOGY COURSE

The Bascom Palmer Eye Institute will give a neuro-ophthalmology course, Dec. 3–5, 1981, in Miami, Florida. The fee is $250 for practitioners and $100 for residents and fellows. For further information, write Bascom Palmer Eye Institute, P.O. Box 610326, Miami, FL 33161.

NEW YORK UNIVERSITY: BASIC SCIENCE COURSE IN OPHTHALMOLOGY

The New York University Post-Graduate Medical School will hold a Basic Science Course in Ophthalmology, Sept. 8–Nov. 24, 1981. The fee is $1,300. For further information, write NYU Post-Graduate Medical School, 550 First Ave., New York, NY 10016.

AMERICAN DIOPTER AND DECIBEL SOCIETY MEETING

The American Diopter and Decibel Society will sponsor a meeting Nov. 30–Dec. 4, 1981, in Puerto Rico. The fee is $200 for members and $250 for non-members. For further information, write Charles S. McConnell, M.D., Cedar Crest Professional Park, Suite 110, 1251 S. Cedar Crest Blvd., Allentown, PA 18103.

UNIVERSITY OF WASHINGTON: COURSE ON RECENT ADVANCES IN STRABISMUS AND PEDIATRIC OPHTHALMOLOGY

The University of Washington will sponsor a course during a cruise to Alaska, Aug. 20–28, 1981. The course will be on recent advances in strabismus and pediatric ophthalmology. For further information, write Melvin Carlson, M.D., 901 Boren Ave., Suite 1030, Seattle, WA 98104.

RETINAL VASCULAR CENTER OF THE WILMER INSTITUTE: 11TH ANNIVERSARY CONFERENCE

The retinal vascular center of the Wilmer Institute will celebrate its 11th anniversary, July 12–17, 1981, at Big Sky, Montana. The conference is entitled "Diagnosis and Management of Retinal Vascular and Macular Disorders." For further information, write Program Coordinator, Office of Continuing Education, Johns Hopkins University, 720 Rutland Ave., Turner 22, Baltimore, MD 21205.

WILMER INSTITUTE: SYMPOSIUM ON VISION PSYCHOPHYSICS AND DISORDERS OF THE VISUAL SYSTEM

The Wilmer Institute will sponsor a symposium entitled "Vision Psychophysics and Disorders of the Visual System," Nov. 12–14, 1981, in Baltimore, Maryland. The abstract deadline is July 1, 1981. For further information, write Robert Massof, Ph.D., or Daniel Finkelstein, M.D., Room B-34, Wilmer Institute, Johns Hopkins Hospital, 600 N. Wolfe St., Baltimore, MD 21205.

UNIVERSITY OF ROCHESTER: ULTRASOUND IN CLINICAL OPHTHALMOLOGY

The University of Rochester will sponsor a course on ultrasound in clinical ophthalmology, Sept. 25 and 26, 1981, at the University of Rochester, Rochester, New York. For further information, write the Office of Continuing Professional Education, University of Rochester Medical Center, 601 Elmwood Ave., Box 677, Rochester, NY 14642.

NEW MEXICO OPHTHALMOLOGICAL SOCIETY: ANNUAL MEETING

The annual meeting of the New Mexico Ophthalmological Society will be held June 25–27, 1981, in Santa Fe. For further information, write Walter J. Levy, M.D., New Mexico Ophthalmological Society, 465 St. Michael's Dr., #114, Santa Fe, NM 87501.

OCULAR HISTOPLASMOSIS: SECOND SYMPOSIUM

The second symposium on ocular histoplasmosis will be held in Indianapolis, Indiana, Jan. 22 and 23, 1982. For further information, write T.F. Schlaegel, Jr., M.D., Indiana University Medical Center, 1100 W. Michigan St., Indianapolis, IN 46223.

GEORGETOWN UNIVERSITY CENTER FOR SIGHT: SECOND ANNUAL REVIEW COURSE FOR OPHTHALMIC MEDICAL ASSISTANTS

Georgetown University Center for Sight will hold its second annual review course for ophthalmic medical assistants, June 4-6, 1981, in Washington, D.C. For further information, write Phyllis Fineberg, C.O.T., Director, Ophthalmic Medical Assistants Programs, Georgetown University, Center for Sight, 3800 Reservoir Rd., Washington, DC 20007.

ORTHOPTIC COUNCIL: WRITTEN AND ORAL EXAMINATIONS

The Orthoptic Council is sponsoring written and oral examinations for students who have completed two years of approved orthoptic training. The written examination will be given in a number of training centers on June 25, 1981. The oral practice examination will be held in Indianapolis, Indiana, on Sept. 26, 1981. For further information, write Stewart M. Wolff, M.D., 803 Cathedral St., Baltimore MD 21201.

JULES STEIN, M.D.
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Jules Stein, M.D., founder of Research to Prevent Blindness, Inc., died Wednesday, April 29, 1981, in Los Angeles. Dr. Stein is survived by his wife, Doris; his daughters, Jean vanden Heuvel and Susan Shiva; and by four grandchildren.

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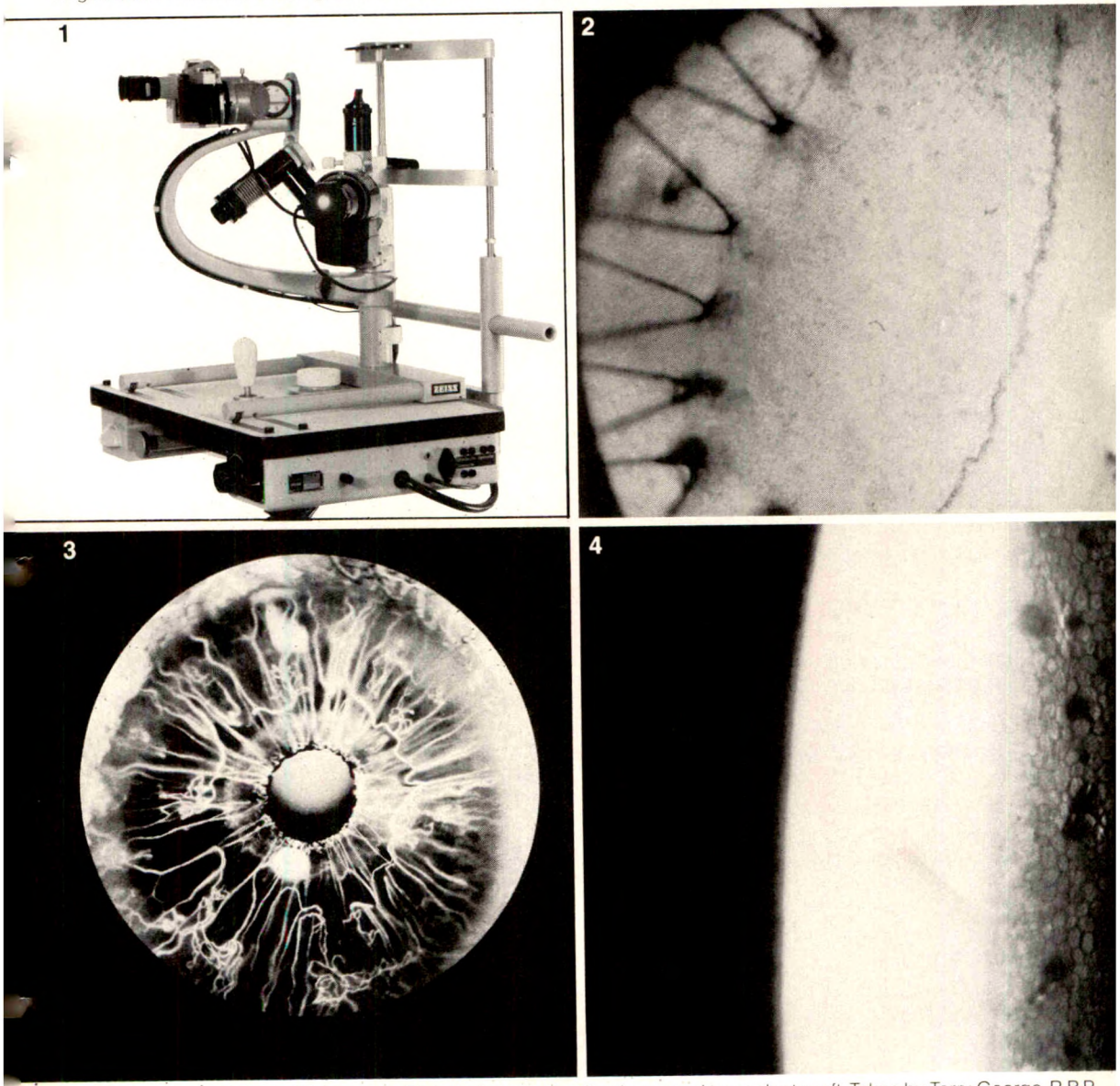
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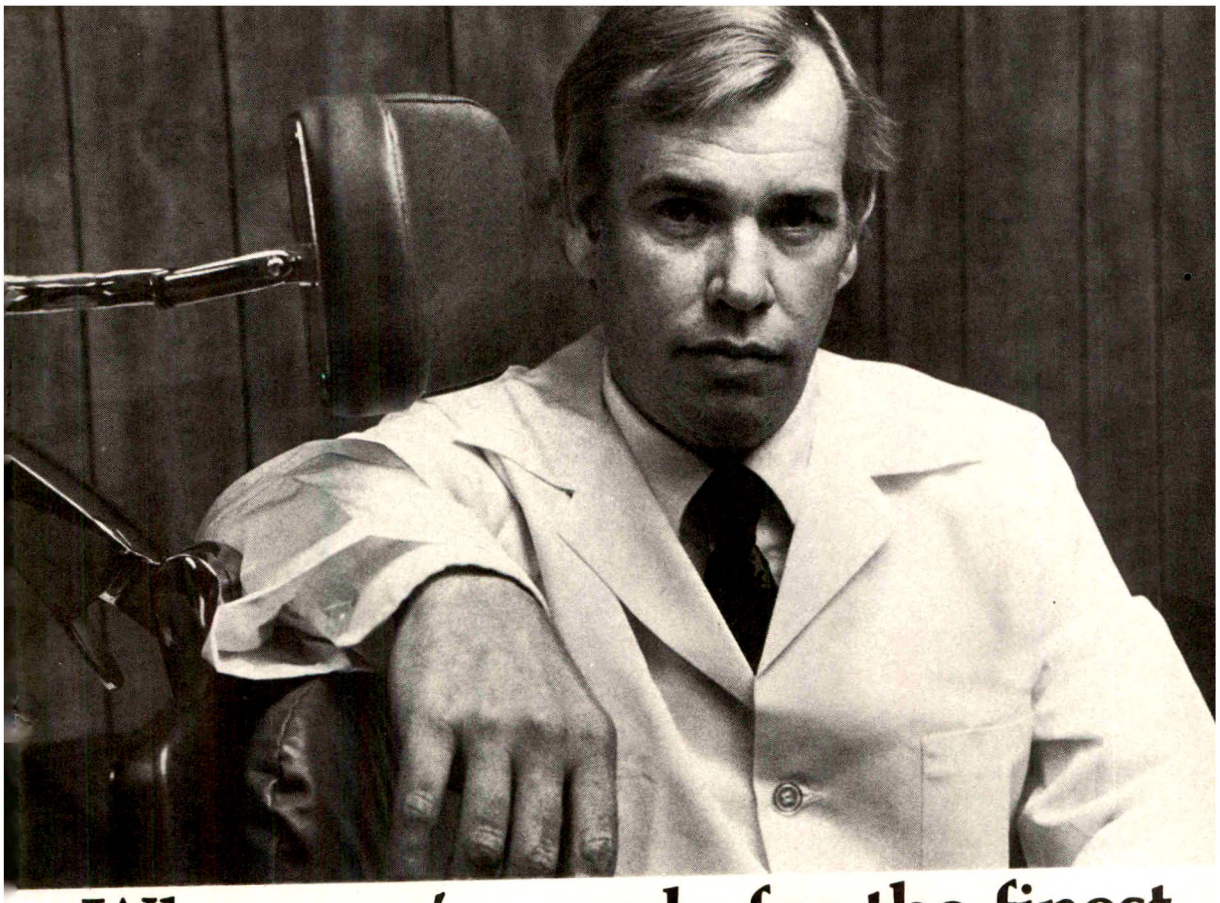
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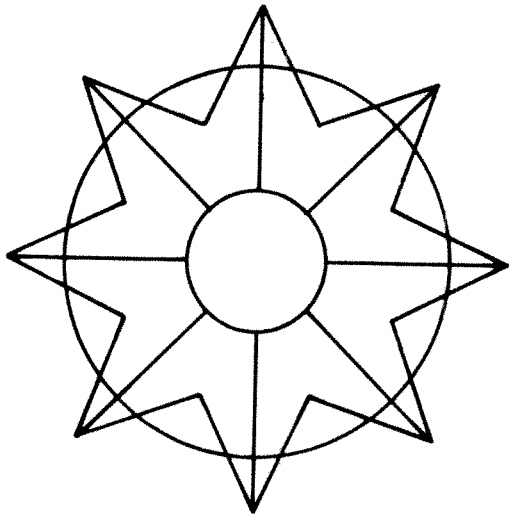
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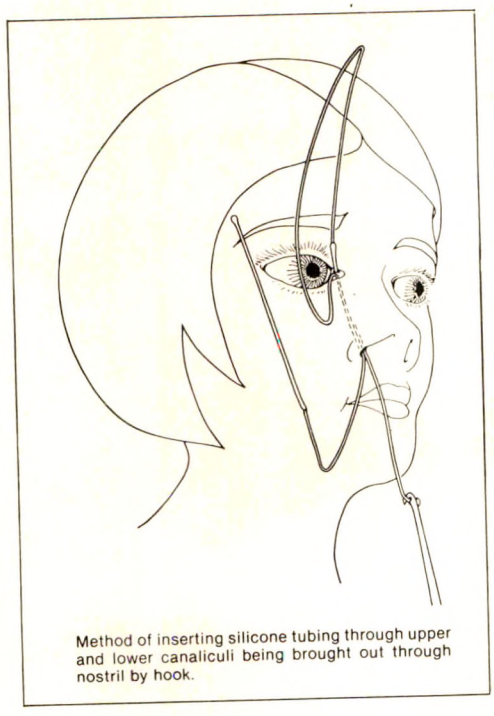
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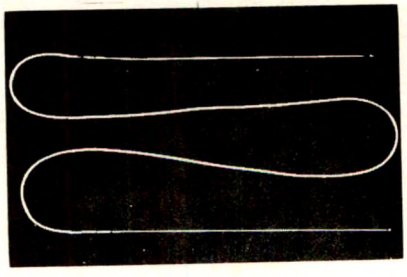
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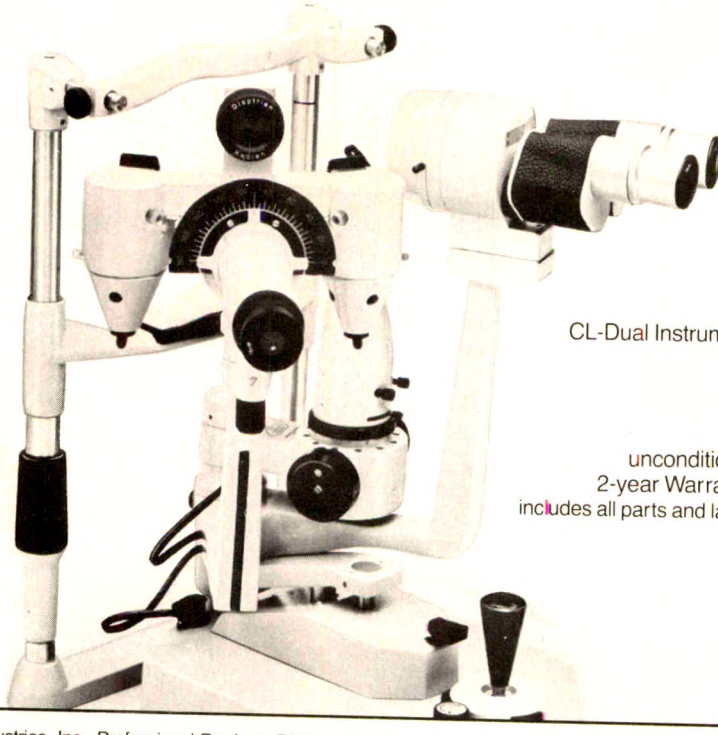
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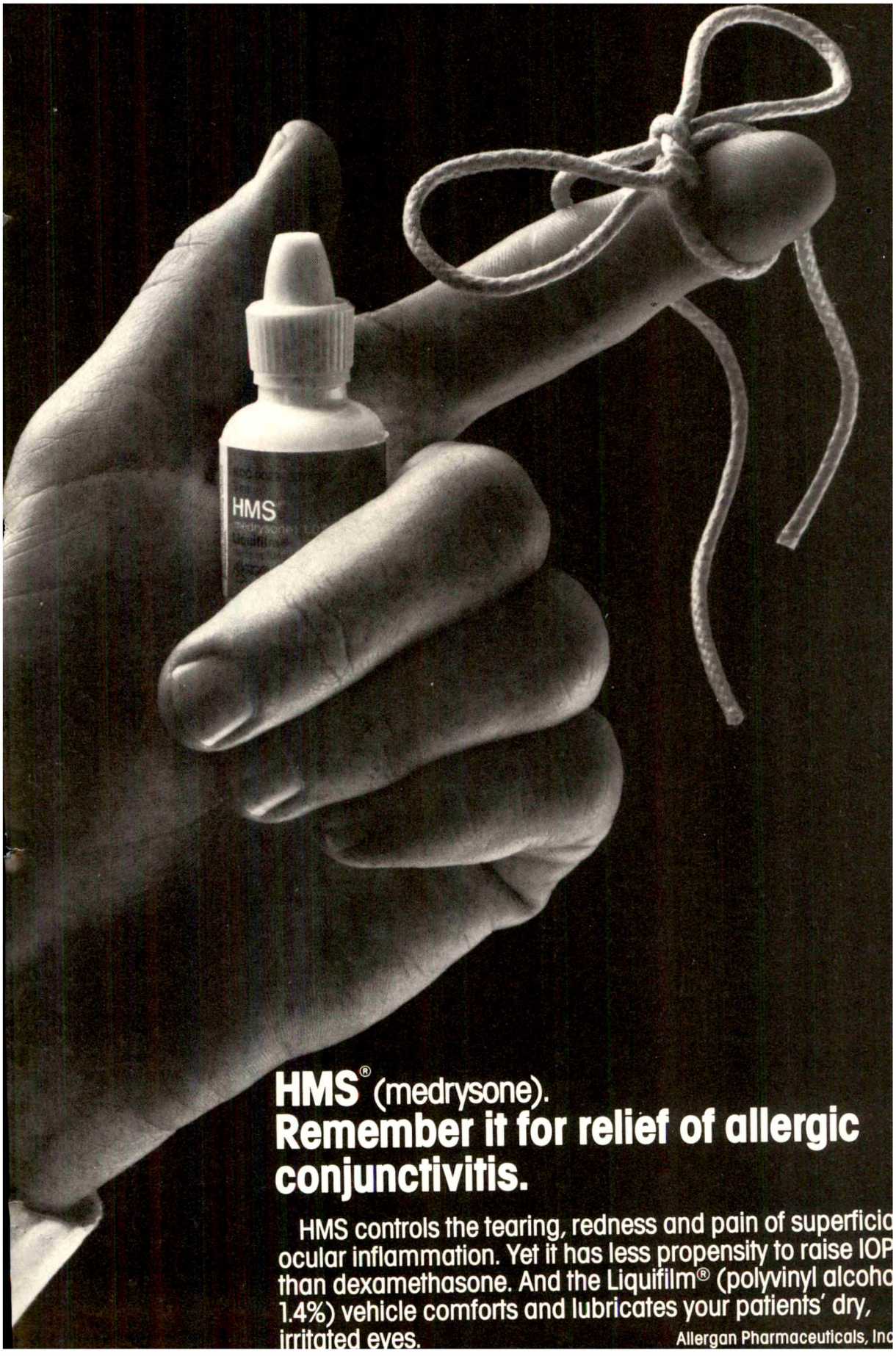
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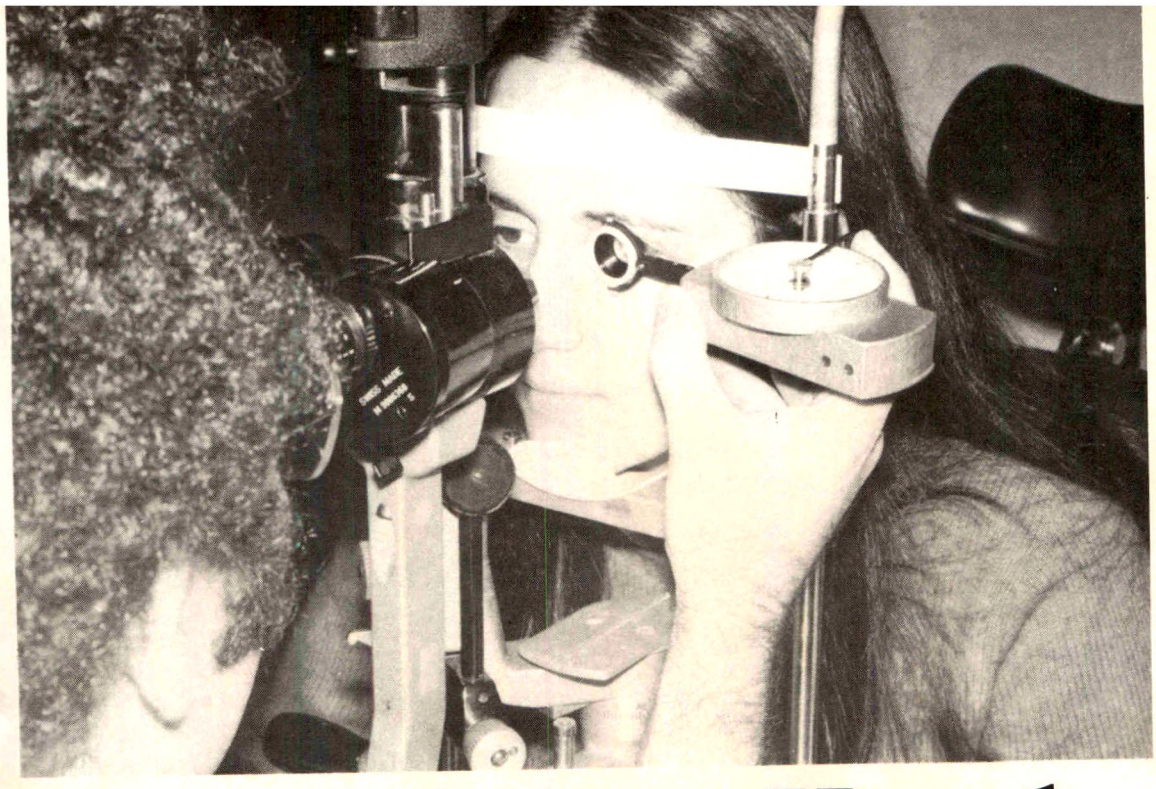


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REFERENCES: 1. Becker, B. and Kolker, A.E.: "Intraocular Pressure Response to Topical Corticosteroids." in Ocular Therapy, Complications and Management. Irving Leopold, Ed. St. Louis, C.V. Mosby, 1967, pp. 79-83. 2. Spaeth, G. Hydroxymethylprogesterone, Arch Oph 75:783-787, 1966.

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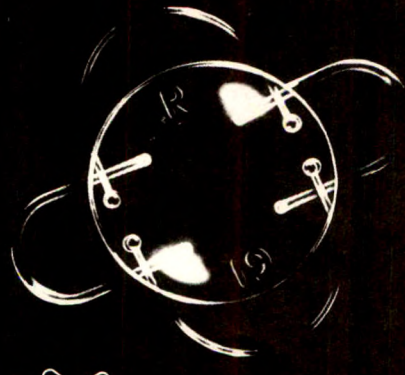
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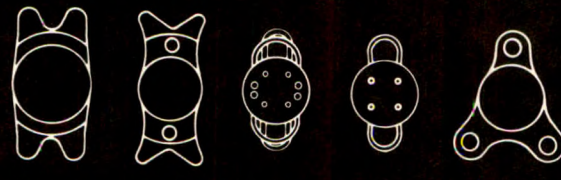
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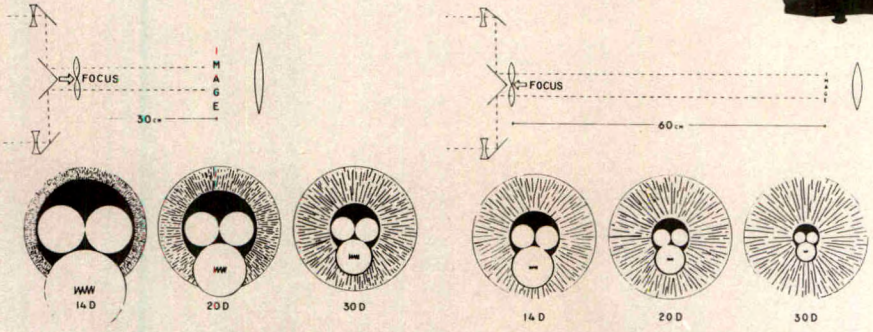
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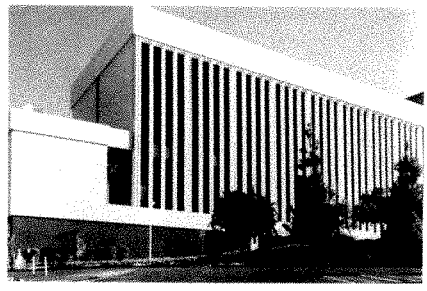
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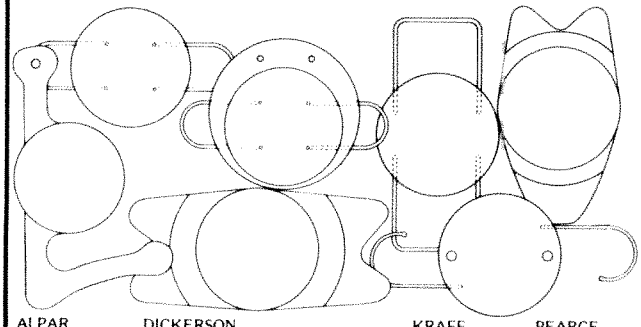
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DAVID SHOCH, M.D.
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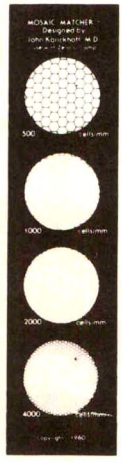
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16. Current unpaid bills: Location
17. Outstanding obligations: Notes, mortgages, pledges, charge accounts, credit checking accounts, etc. Details
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19. Savings Account(s): Location(s) of passbook(s)
20. Checking Account(s): Location(s), location(s) of checkbook(s)
21. Financial Records: Location
22. Cash: Location of receipts, unbanked cash, source of cash for immediate needs
23. Tax Information: Location
24. Accounts Receivable: Location, the Doctor's views on collecting current and delinquent bills
25. Notes, loans, mortgages, etc., due from others: Location of items
26. Notifying patients: Method preferred by the doctor, location of appointment book
27. Notification: Hospital(s)—(Administrator), phone numbers: colleagues, phone numbers, Societies, etc., phone numbers
28. Veterans Administration: Branch of Service, date of discharge, service number, VA number(s), location of discharge papers
29. Blue Cross, etc.: Policy numbers of Blue Cross, Blue Shield and other Health Insurance (see later discussion)
30. Narcotics: Location of drugs, tax stamp, narcotics ledger, narcotics order book
31. Equipment (major): Purchase price, sales outlet
32. The Doctor's views on disposition of property, securities, anticipated income, etc.
33. Birth and Marriage Records: Location
34. Citizenship Papers (if applicable): Location

In addition to the foregoing but subject to the rights and duties of the Executor or Administrator, you may become involved with the following:

I. Probate of the Will: Your attorney will do this.

II. Disposition of narcotics, drug samples and medications. You should contact your Regional Office of the Bureau of Narcotics and Dangerous Drugs, U.S. Department of Justice, requesting disposition forms which must be completed and returned.

III. Patient Records:

A. These are important and confidential records which should be carefully preserved. Apparently there is no set limit of time specified by law.

B. The contents of such records should be disclosed to another physician only if the patient so requests. Such a request should be in writing and should be retained with the records. Normally, the records are confidential and diagnostic information in them should not be disclosed directly to the patient because of the risk that he might misinterpret them.

C. All patients' medical records should be kept for a minimum of ten years. Records of patients who were minors when treated should be preserved until two years after the patient reaches twenty-one. When possible, records should be kept for twenty-five years. It should be kept in mind that requirements for the retention of medical records may vary from state to state. Accordingly, before any medical records are destroyed or disposed of, local requirements should be verified with either the attorney for the estate or the local medical society. Financial records should be kept for five years, which is the statute of limitations on such information.

IV. Telephone: If there is an answering service, this can be a very helpful way of notifying patients of the Doctor's death with possible suggestions regarding their continued care. It is suggested that this service be retained for at least a month.

V. Notifying patients: Perhaps a simple sign on the office door "Call ———————— (office phone number)" will relieve you of the burden of contact with most of the patients and the answering service can take care of the matter.

If the physician's secretary is retained temporarily she might perform this service by being present during usual office hours. She also could notify patients with future appointments during this time.

VI. Taxes: The accountant and/or attorney should take care of all of this in connection with the financial records.

VII. Stopping advertising mail: It may take a year for knowledge of the Doctor's death to reach all the mailing lists; there is no central clearing office. You may perhaps choose to let it run out in this fashion, or you may write "Deceased return to sender" on each piece and mail it back. While the latter course might entail some effort initially, it will rapidly and effectively cut off the flow.

VIII. Insurance Agent and/or Trustee: A preliminary brief conference might be in order to settle the question of immediate funds, documents to be located, and arrangements for a later, detailed conference. Prompt notification of such people is important.

IX. Consideration of Sale of Practice: If the practice is to be sold, with or without real estate, relatively rapid action is advised, for it will quickly lose value. It can be advertised in the State Medical "Journal" and the "Journal" of the American Medical Association.

X. Real Estate: Except for practice-connected real estate, a hasty decision on sale or retention is not advised. It is hoped that the doctor has made known his views on this subject.

XI. Temporary coverage of patients:

A. Some arrangement with a colleague should be made immediately as regards patients in the hospital.

B. While not required, it would be a courtesy to be able to suggest a colleague to other patients.

XII. Insurance:

A. Malpractice Insurance: A refund on unused premiums may be possible. Find the policy and consult the local agent.

B. Liability and other Insurances: Certain policies issued with reference to the practice might have provisions for refund of unused premiums. Consult the issuing company or your insurance agent.

C. Health and Hospital Insurance: The survivor of a physician who had hospital or other health insurance under a group or family plan may keep such insurance, but it should be transferred to individual instead of family policies, unless you have dependent children.

XIII. Veterans Administration: If the Doctor was a Veteran, notify the Veterans Administration with the indicated information and apply for deserved benefits. Help with this can be had through the Veteran's Service Officer of the Doctor's county or district, or the Veterans Administration Regional Office. In this connection, save the Doctor's discharge papers permanently. It is possible that Pensions or other forms of remuneration may be provided in the future.

XIV. Social Security: Nearly all physicians are now covered by the Social Security Act. Visit the Social Security Office serving your area to find out what benefits are available to you. Be certain to have the Doctor's Social Security or Taxpayer Identification Number.

XV. Automobile(s): Title, if in Doctor's name, can be transferred with assistance of an automobile club, if a member, or through the Department of Motor Vehicles. Be sure insurance coverage is retained.

XVI. Equipment: It is best disposed of as part of the practice, if possible. Otherwise, it might be possible to obtain an estimate of value from one of the reputable equipment vendors and then sold either privately, through an advertisement in the State Medical "Journal" or to a dealer of used equipment. One of the Doctor's colleagues can give you the names of the major supply dealers serving your area.

XVII. Current bills: These must be paid, but not until the court has approved the Executor or appointed the Administrator of the Doctor's estate.

XVIII. Magazine subscriptions: Those purchased for office use might be checked for possible stoppage and subscription refund if you do not wish to continue them.

XIX. Dues: There is ordinarily no refund on Medical Society dues unless hardship exists.

XX. Securities: It is worth noting that Securities can be used as collateral for loans to meet financial needs. This may be preferable to selling the securities.

The American Society of Internal Medicine has learned with regret of the Doctor's death. We wish to extend our condolences. We realize that you will have many tasks in connection with settlement of the estate. To assist you the Society has prepared this pamphlet.

It should be pointed out that many of the items appearing herein are matters which, technically, will be the professional responsibility of the Executor (if the Doctor died with a Will) or the Administrator (if the Doctor died without a Will). However, the information contained herein may prove to be helpful not only to the person having this responsibility but also to the Doctor's survivor. We recommend that the survivor discuss the contents of this pamphlet with the Executor or Administrator of the Doctor's estate as soon as possible.

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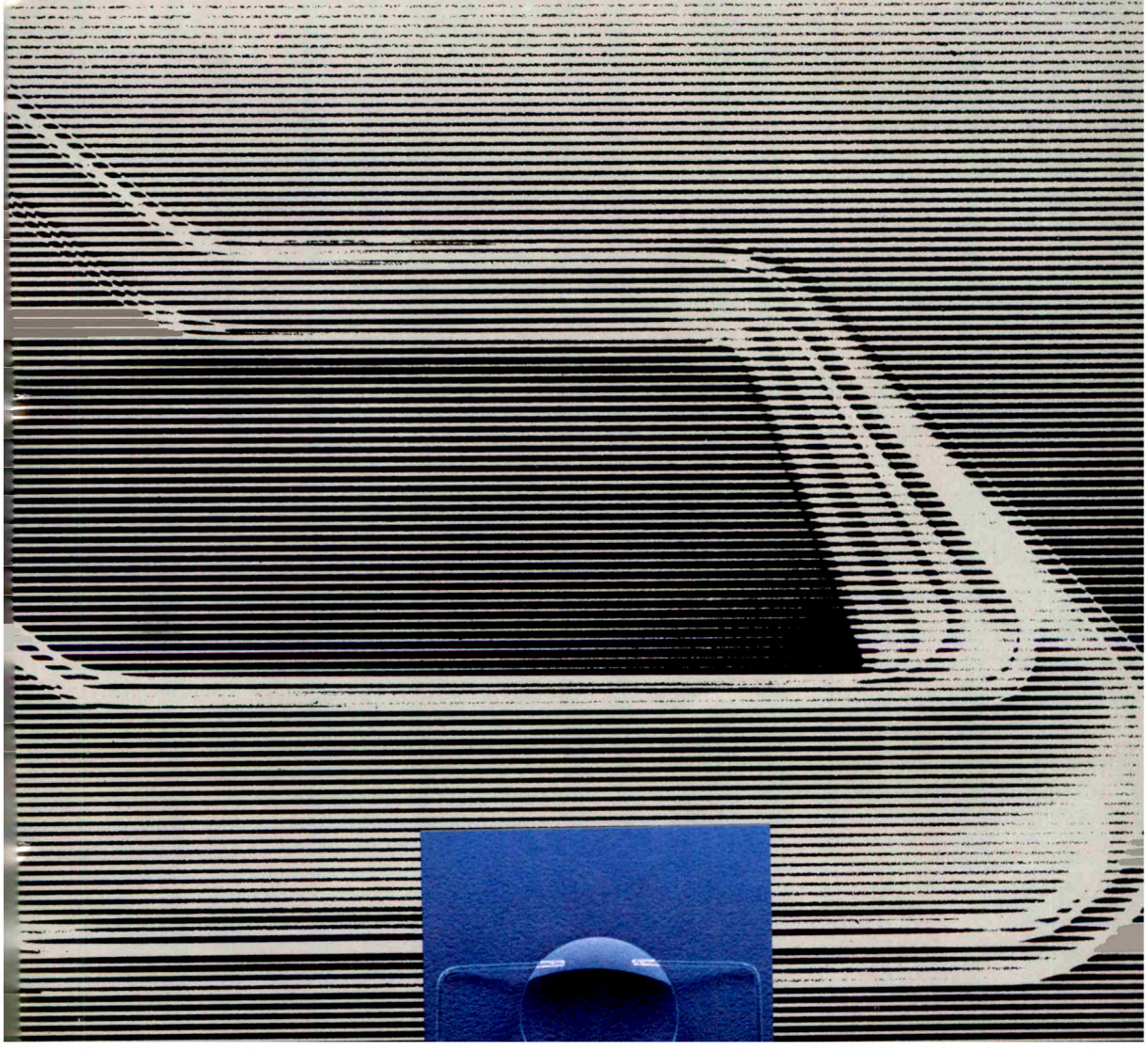
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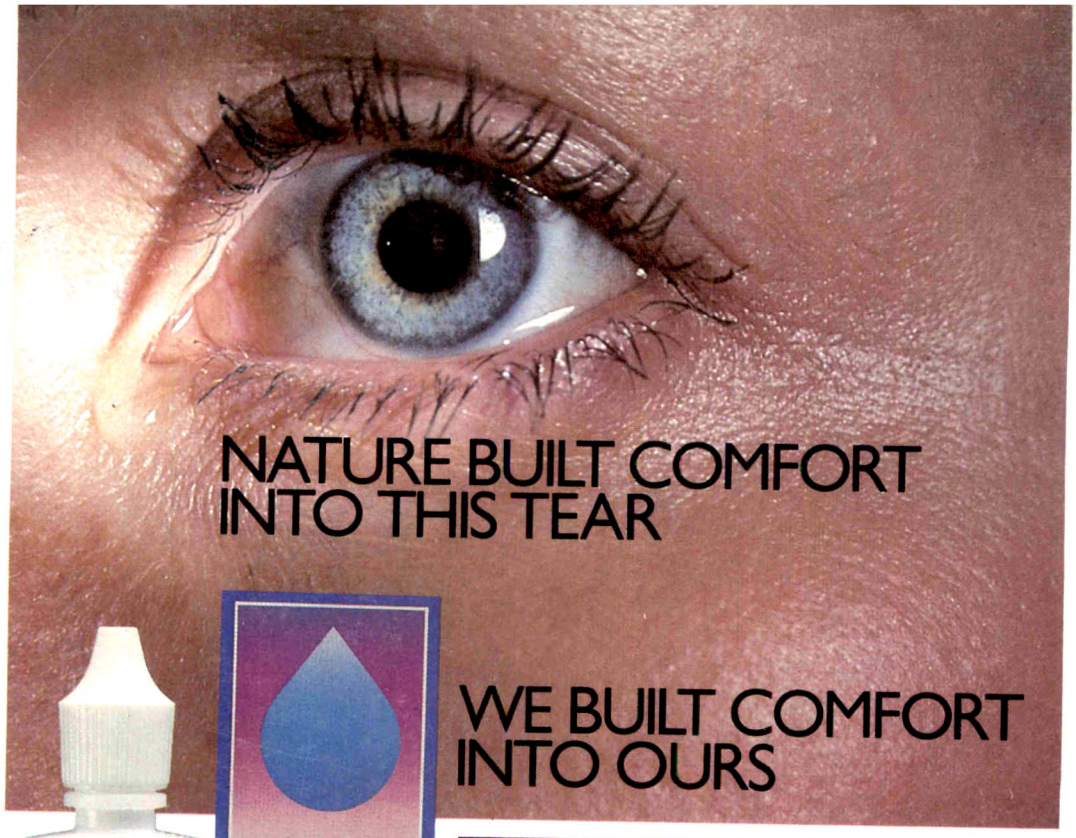
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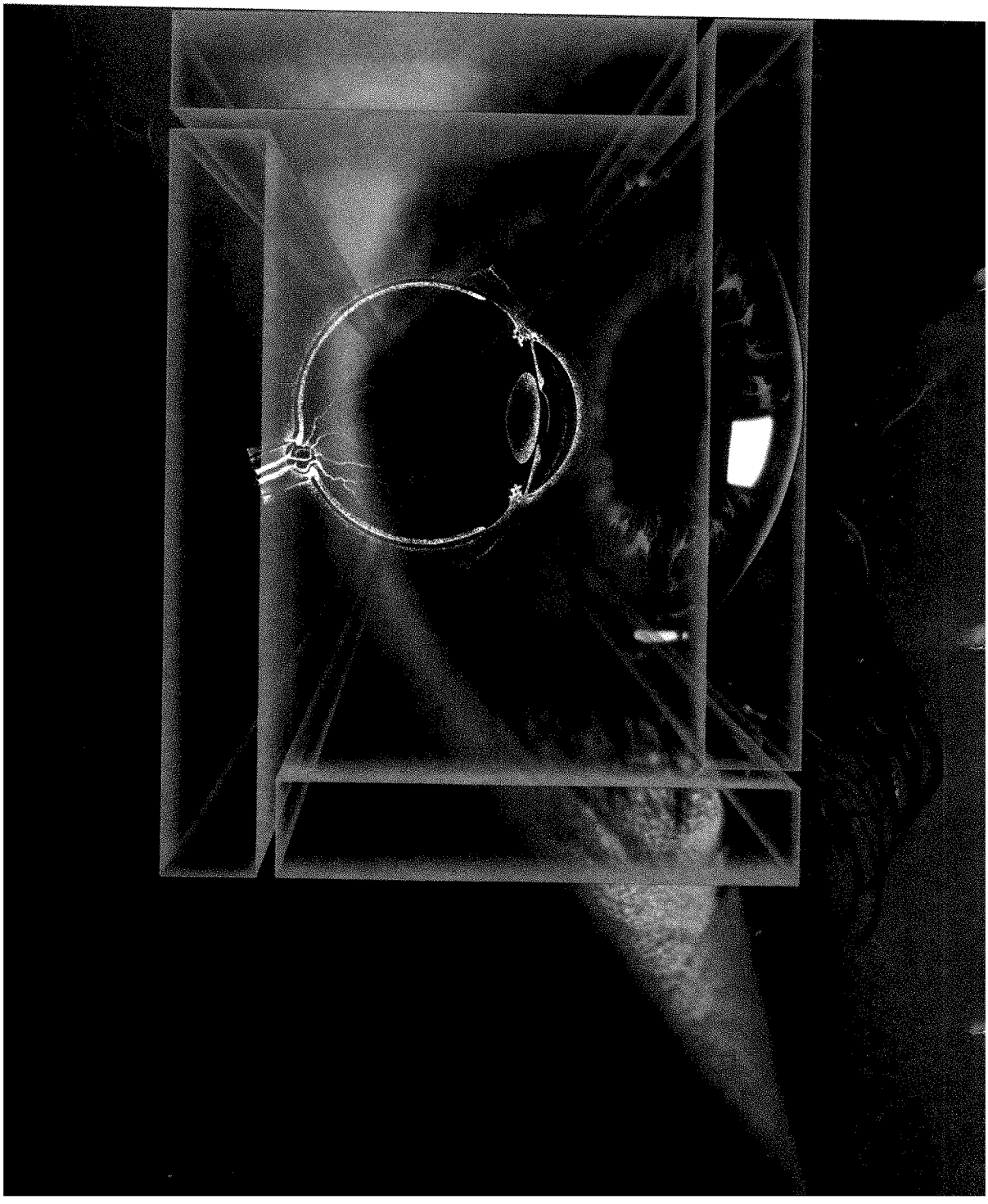
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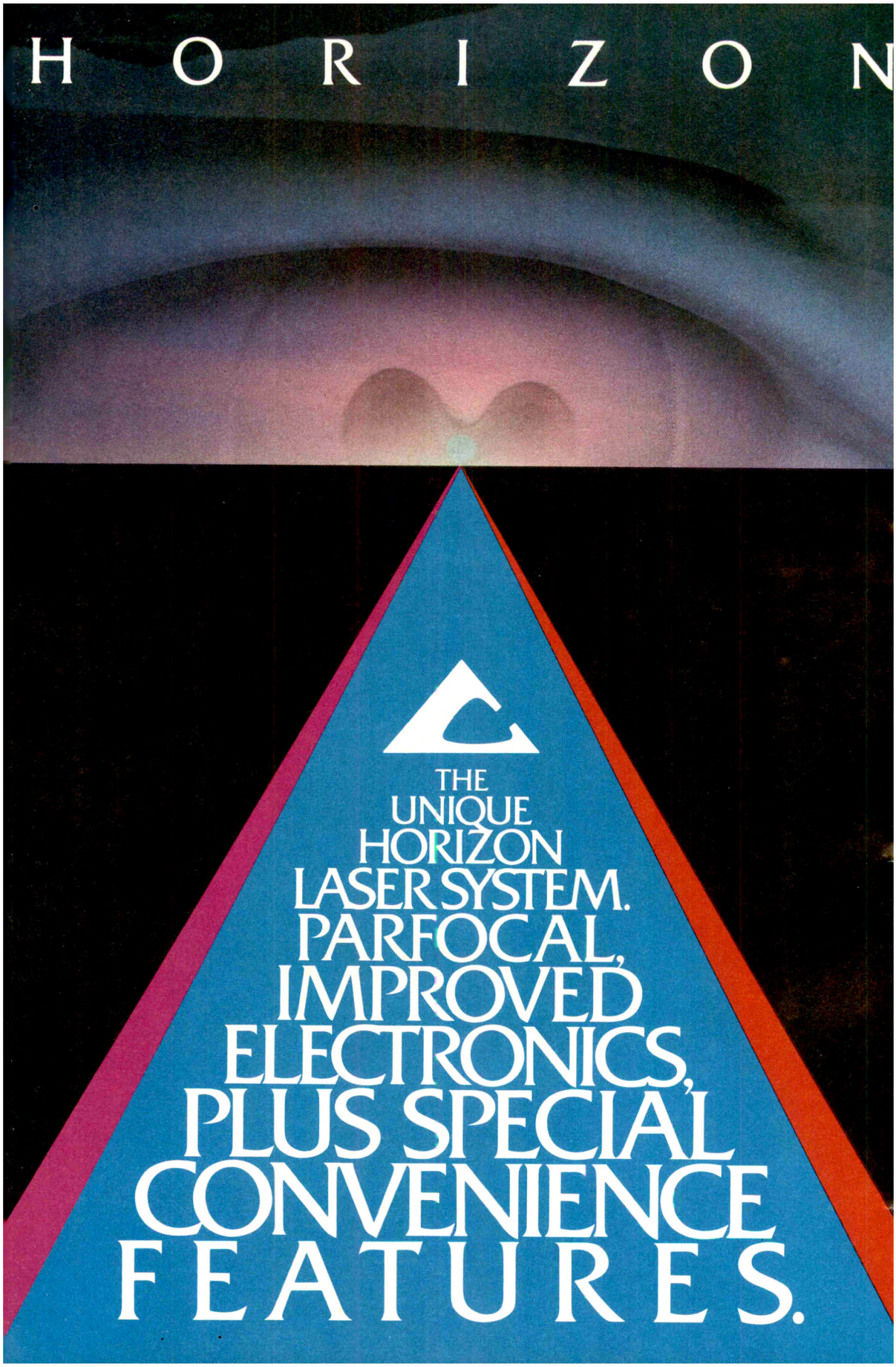
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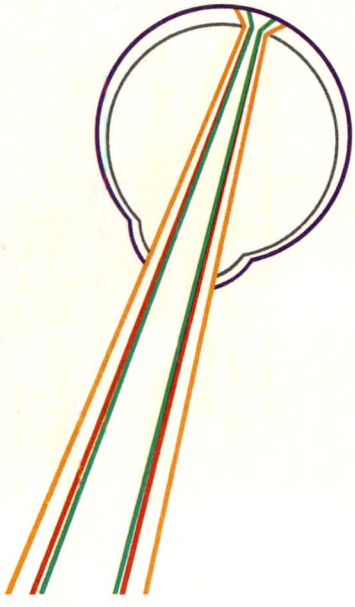
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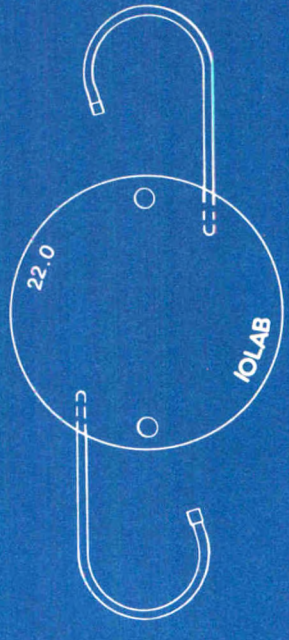
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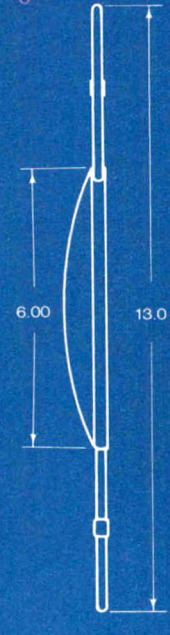
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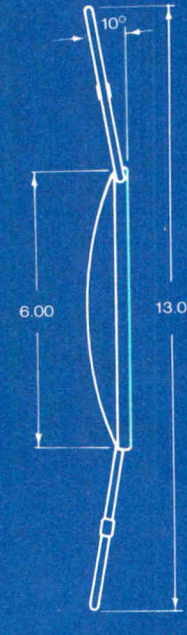
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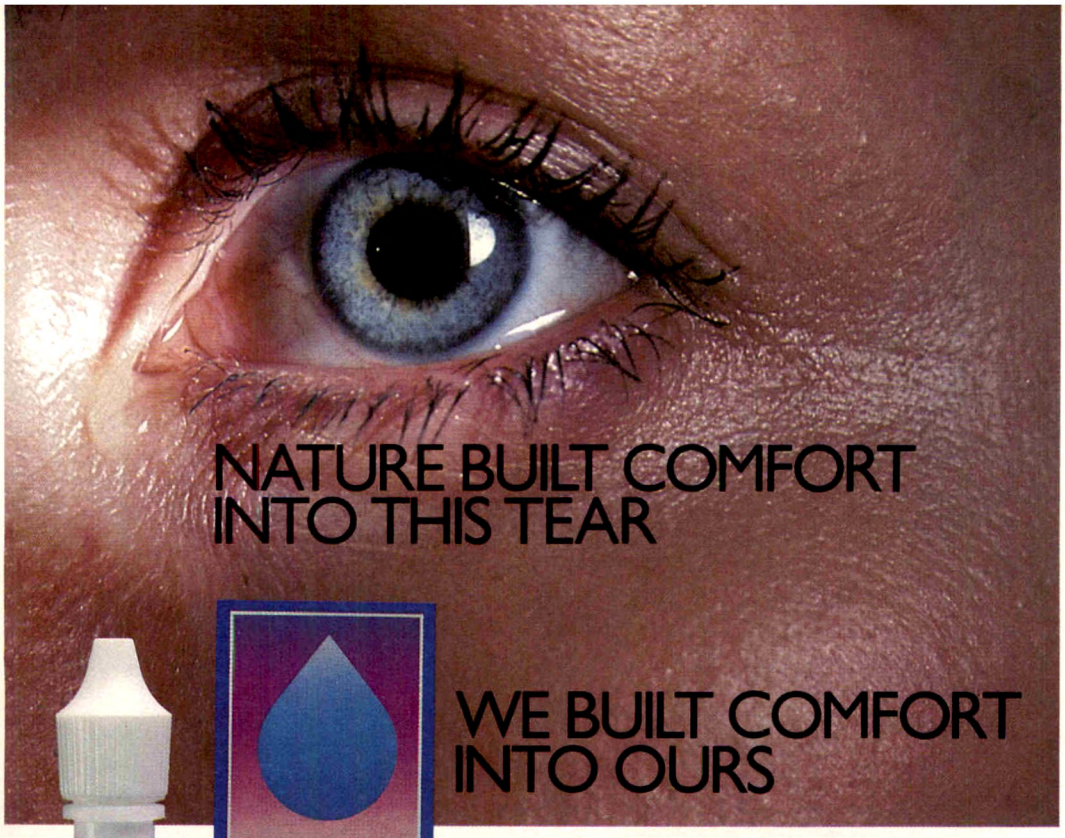
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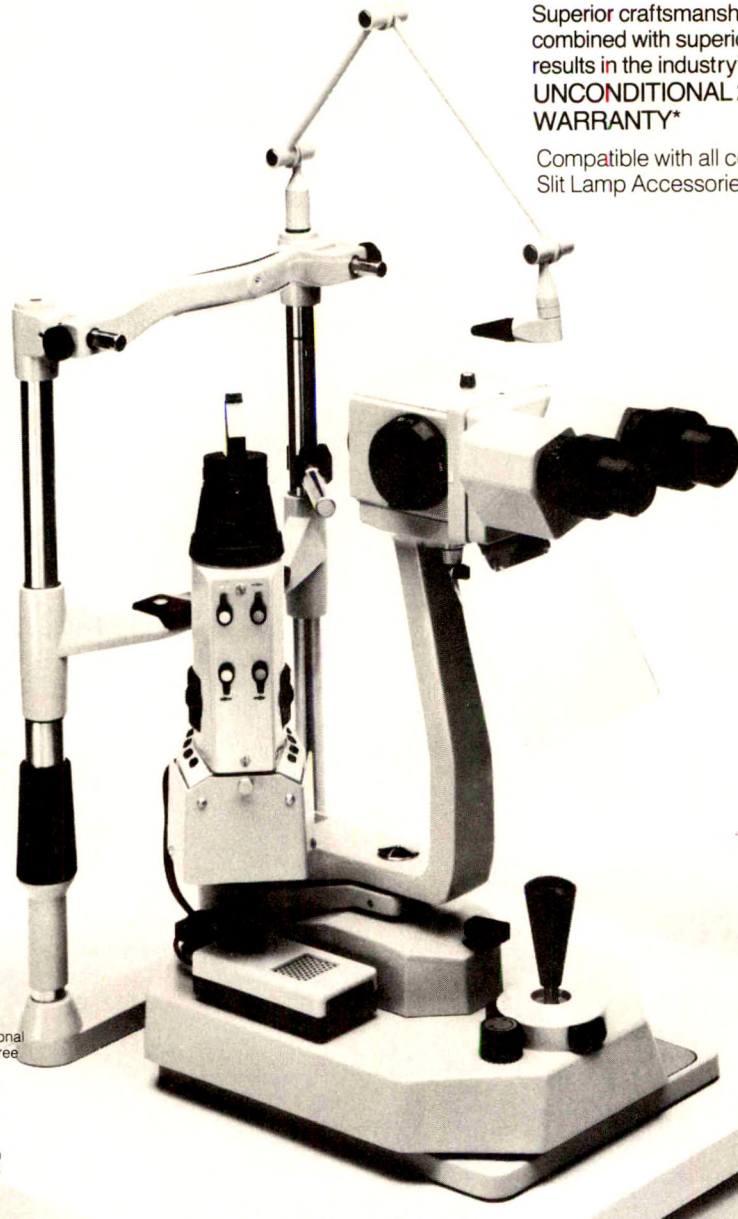
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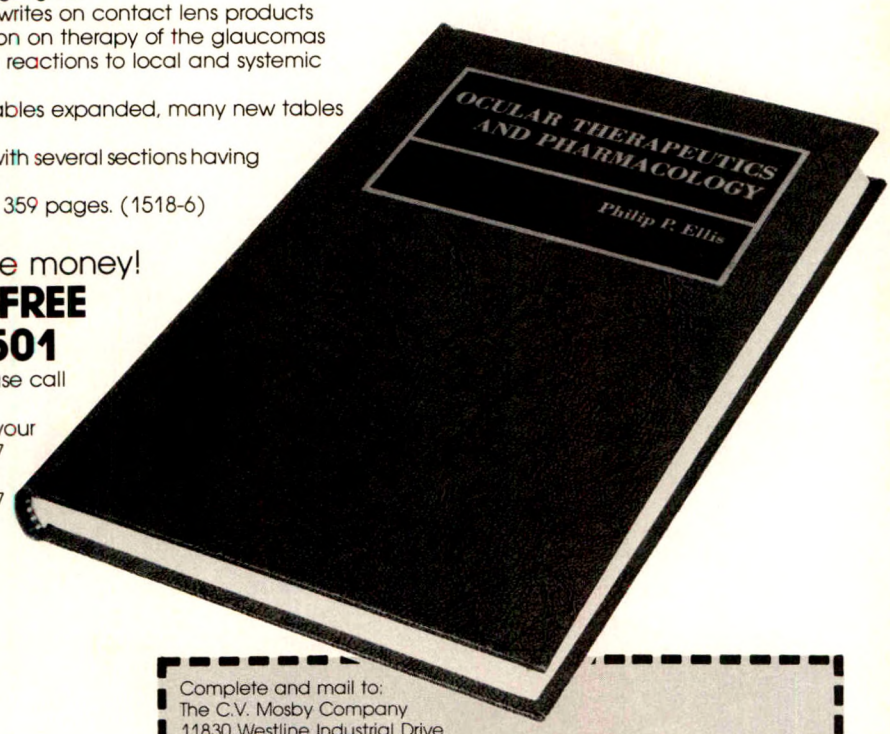
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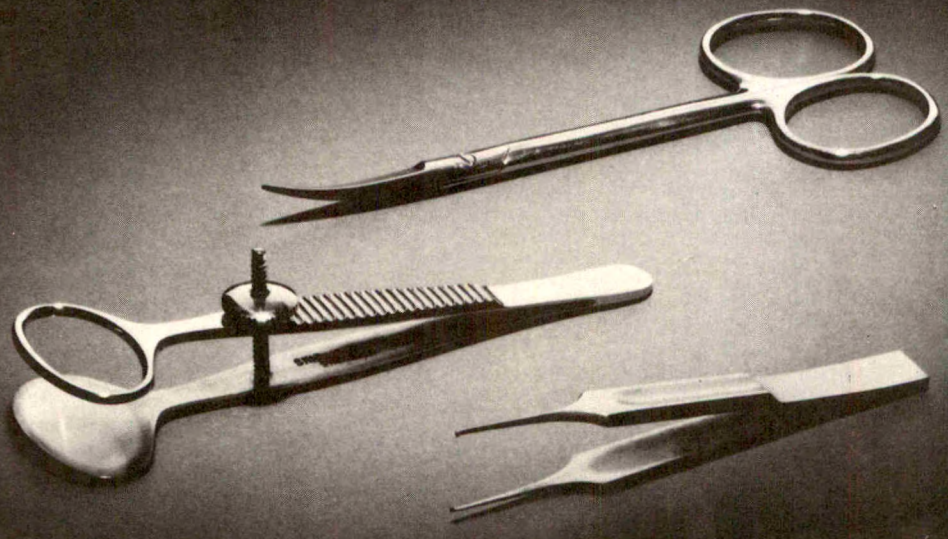
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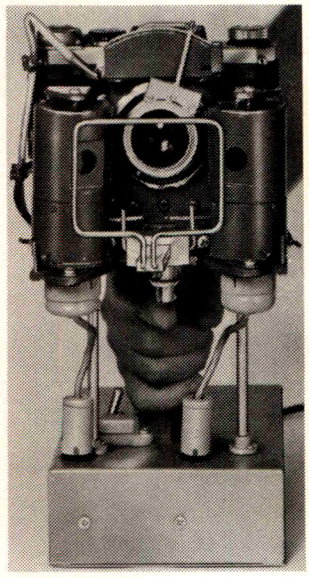
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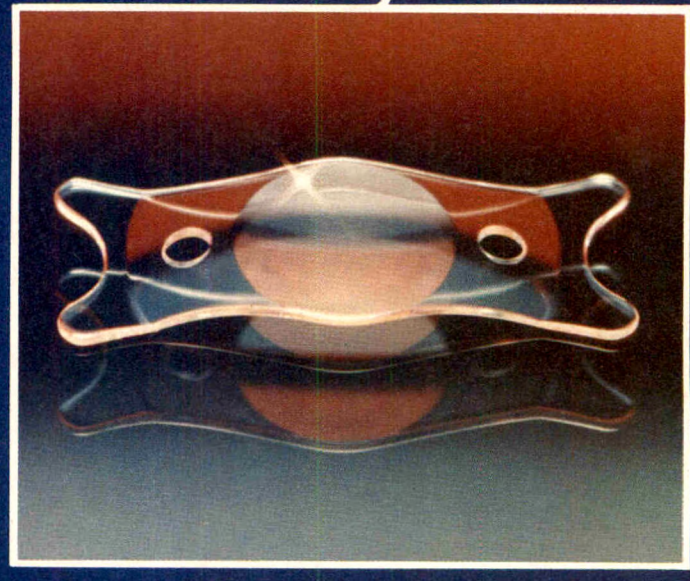
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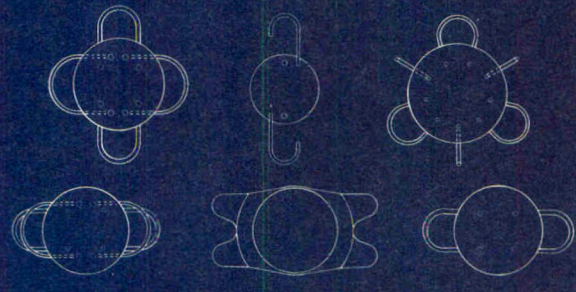
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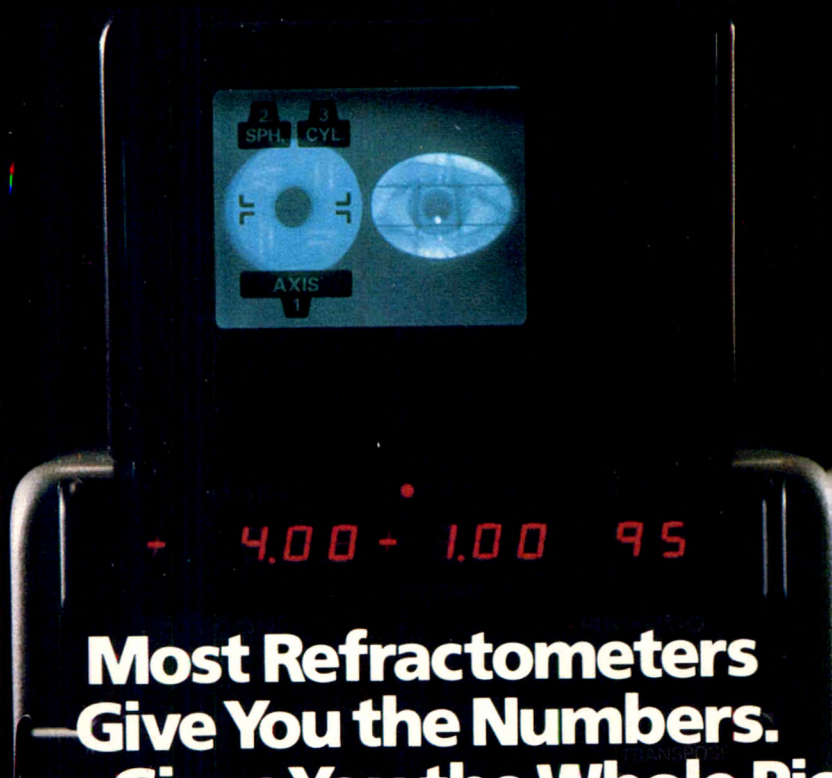


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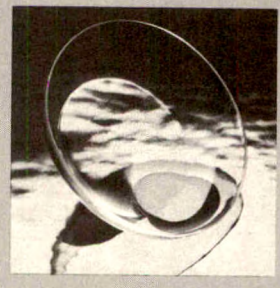
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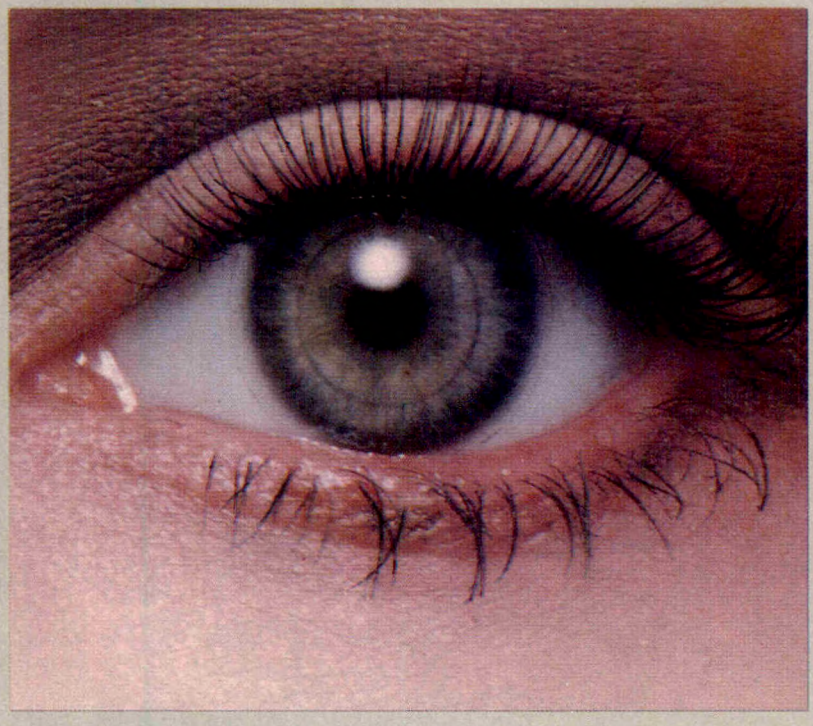
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[1]Clinical Data on file, Heyer-Schulte Medical Optics Center

[2]Cavanaugh, H.D., Bodner, B.I., Wilson, L.A. "Extended Wear Hydrogel Lenses, Long-Term Effectiveness and Costs," Ophthalmology, 1980, 87:9—871.

[3]Fatt, I. and Morris, J., "A Survey of Gas-Permeable Contact Lenses." The Optician, November 1977.

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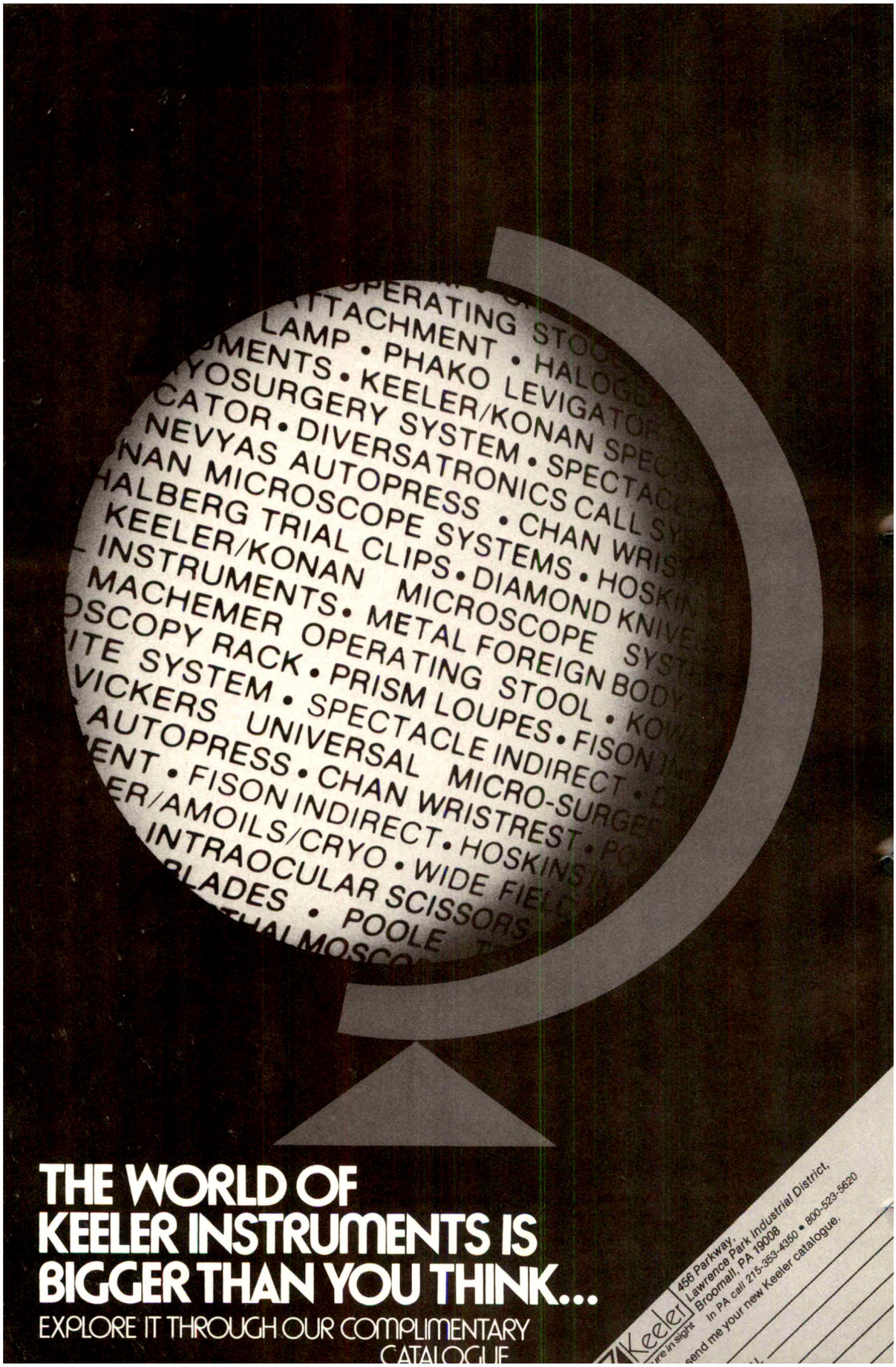
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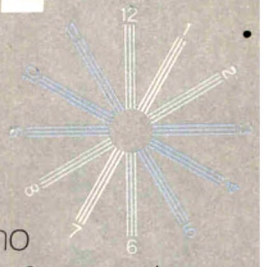
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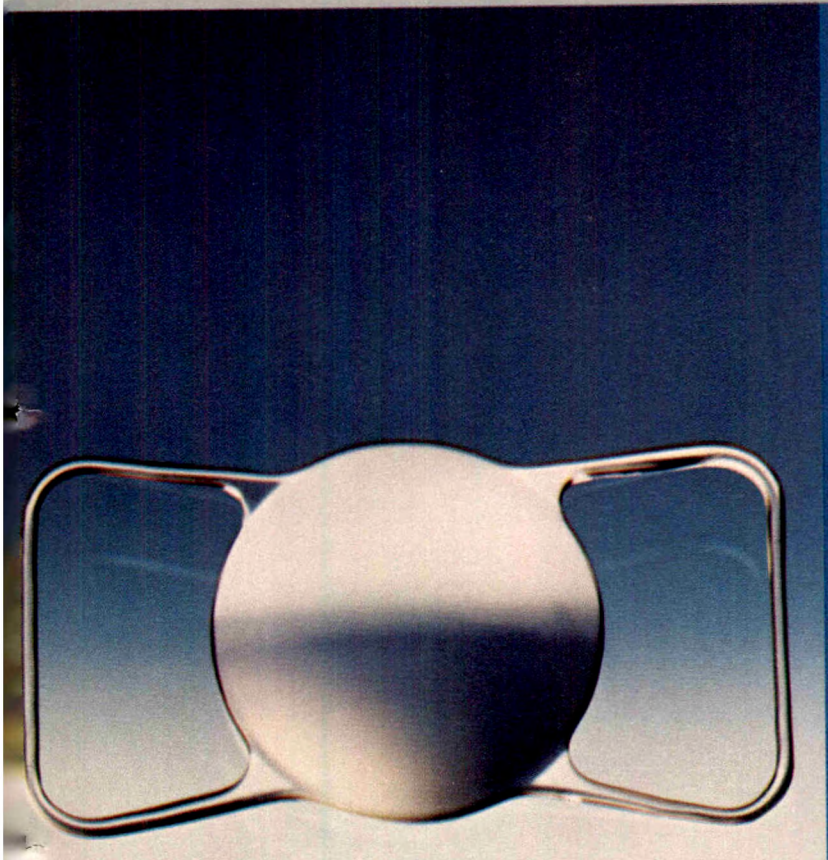
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1981 Apr. 24-25	St. Joseph Hospital Chicago, IL	Charles Vygantas, MD George Wyhinny, MD	Cindy Sandahl (312) 871-8444
May 6-9	University of Texas Medical San Antonio, TX	David Hunter, MD	Marilyn Rennels (512) 691-6295
May 22-23	Pacific Medical Center San Francisco, CA	Arthur W. Allen, MD	Sally Dunmeyer (415) 563-4321 ext. 2761
June 11-12	Georgetown University Washington, DC	Leonard Parver, MD	Mary J. Day (202) 625-2306
June 25-27	University of Iowa Iowa City, IA	Thomas Weingeist, MD	Terry Fanning (319) 356-2215
July 10-11	Hyatt Regency, Dearborn Detroit, MI	Howard Joondeph, MD	Marion Lynch (313) 882-4002
July 24-25	Truman Medical Center Kansas City, MO	Felix Sabates, MD L. Piebenga, MD	Harry Kuba (816) 556-3595
July 27-29	Sanatorio San Camilo Angel Gallardo 899 Buenos Aires, ARGENTINA	Dr. Alberto Zambrano Dr. Edgardo Manzitti Dr. Gustavo Mayorga Argañaraz	Delfor Horacio Cervo Uruguay 390-Piso 13-OF B 1015 Buenos Aires, Argentina Phone 45-3043
Aug. 3-7	Australian Society for Ophthalmology Melbourne or Sydney, AUSTRALIA	G. Waring, MD	
Aug. 7-8	Sheraton Plaza Hotel Rush Presbyterian Medical Center Chicago, IL	Thomas Bennett, MD	Barbara Trejo (312) 942-7095
Aug. 21-22	USC—Estelle Doheny Eye Foundation Los Angeles, CA	Kenneth R. Diddie, MD	Nancy Call (213) 224-7752
Sept. 4-5	The Broadmoor Colorado Springs, CO	Mylan Van Newkirk, MD	Delores Dailey (308) 635-3911
Sep. 18-19	Emory University Atlanta, GA	Travis Meredith, MD Henry Kaplan, MD	Debbie Seay (404) 329-4271
Oct. 1-3	Devers Eye Clinic Portland, OR	Richard Chenoweth, MD	Suzanne Meyers (503) 225-7881
Oct. 16-17	St. Louis Eye Hospital St. Louis, MO	Orion T. Ayer, MD Anwar Shah, MD	Karen Maroney (314) 644-6800
Nov. 19-20	Philadelphia, PA	Alexander J. Brucker, MD	Pat Shengle (215) 662-8675
1982 Feb. 12-13	University of North Carolina Chapel Hill, NC	David Eifrig, MD	Betty Neilson (919) 966-4161
Feb. 26-27	Emory University Atlanta, GA	Travis Meredith, MD Henry Kaplan, MD	Debbie Seay (404) 329-4271
Mar. 26-27	University of South Carolina Columbia, SC	Mark Hammer, MD	(803) 765-7356
Apr. 23-24	Jules Stein Eye Inst., UCLA Los Angeles, CA	Thomas Hanscom, MD	Lu Hendricks (213) 825-4617

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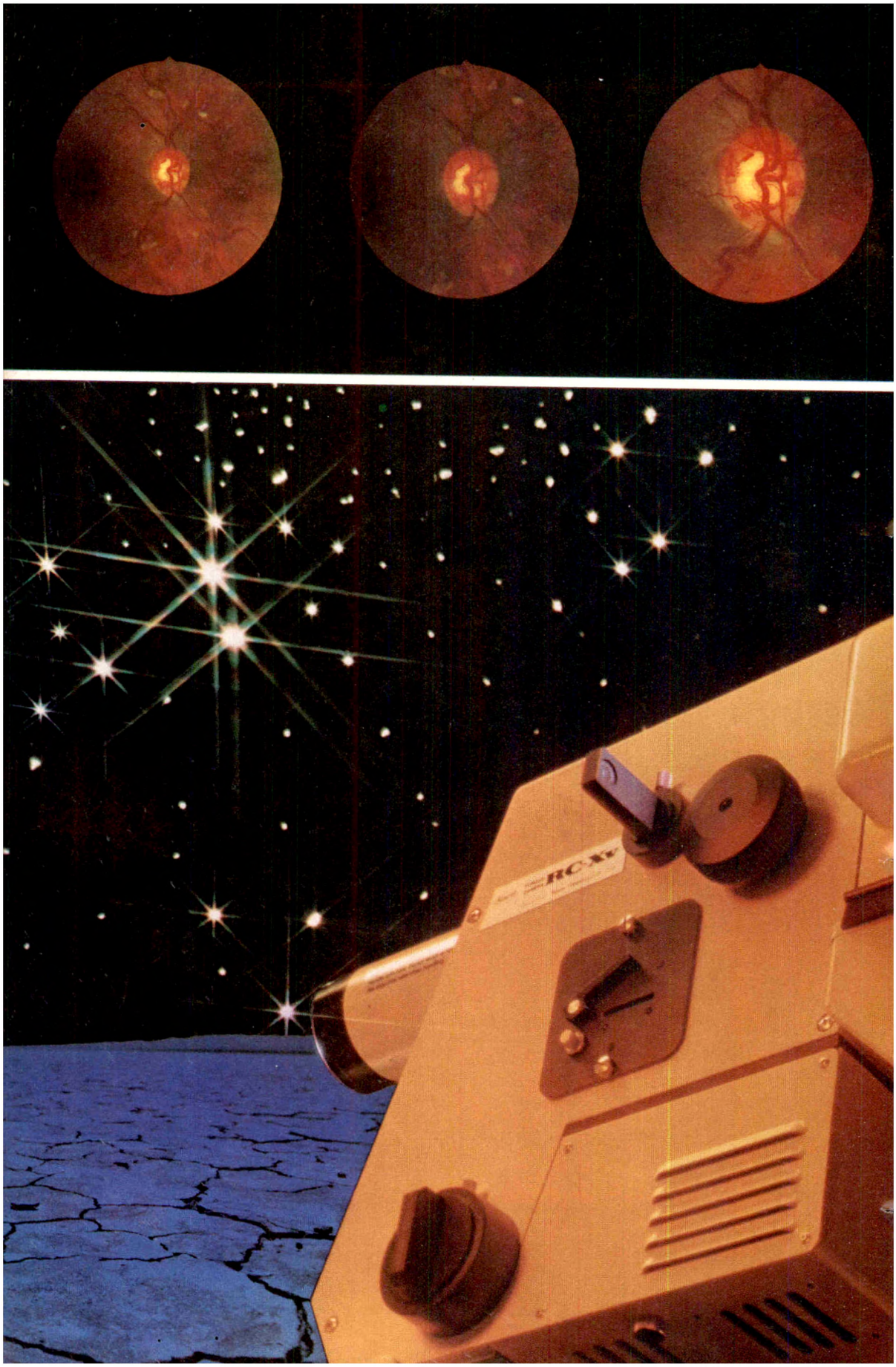
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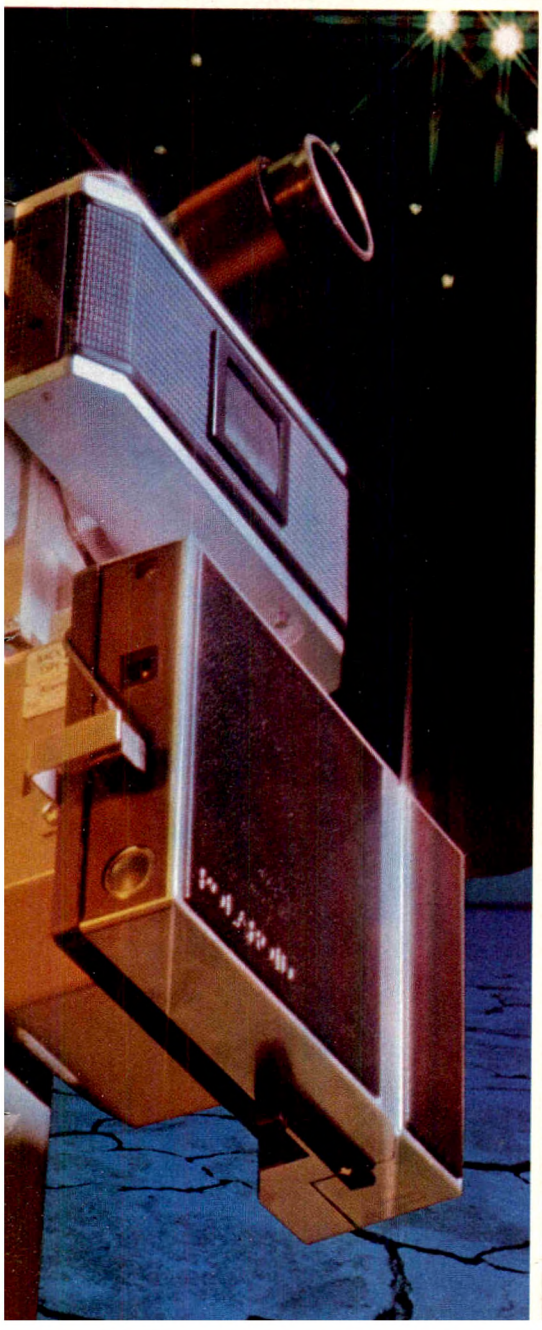
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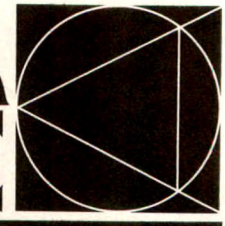


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ACTIONS: Sulfacetamide sodium at 10% concentration is a potent bacteriostatic agent (effective against a broad range of pathogens, including staphylococci). The prednisolone content (in microfine, non-irritating suspension) effectively counters the allergic and inflammatory manifestations of blepharitis. The phenylephrine component of Blephamide rapidly whitens the engorged vessels in the eye and lid.

INDICATIONS: Based on a review of this drug by the National Academy of Sciences — National Research Council and/or other information, FDA has classified the indications as follows: "Possibly" effective: Nonpurulent blepharitis and blepharoconjunctivitis (seborrheal, staphylococcal, allergic); nonpurulent conjunctivitis (allergic and bacterial). Final classification of the less-than-effective indications requires further investigation.

CONTRAINDICATIONS: Acute herpes simplex (dendritic keratitis), purulent untreated infections, vaccinia, varicella and most other viral diseases of the cornea and conjunctiva, ocular tuberculosis and fungal diseases of the eye. **WARNINGS:** 1. In diseases due to microorganisms, infection may be masked, enhanced or activated by the steroid. 2. Extended use may cause increased intraocular pressure in susceptible individuals. It is advisable that intraocular pressure be checked frequently. 3. In those diseases causing thinning of the cornea, perforation has been known to have occurred with the use of topical steroids. 4. Use with caution in patients with known or suspected sensitivity to sulfonamides — if sensitivity or other untoward reactions occur, discontinue medication. 5. Should be used with caution in the presence of narrow angle glaucoma. 6. Reports in the literature indicate that posterior subcapsular lenticular opacities have been reported to occur after heavy or protracted use of topical ophthalmic corticosteroids. **DOSAGE AND ADMINISTRATION:** Optimal dosage is 1 drop two to four times daily, depending upon the severity of the condition. In general, during early or acute stages of blepharitis, Blephamide produces results most rapidly — and most efficiently — with instillation directly into the eye, with the excess spread on the lid (Method I). When the condition is confined to the lid, however, Blephamide may be applied directly to the site of the lesions (Method II). **METHOD I: In the Eye and on the Lid.** 1. Wash hands carefully. Tilt head back and drop 1 drop into the eye. 2. Close the eye and spread the excess medication present after closing the eye over the full length of the upper and lower lids. 3. Do not wipe any of the medication off the lids. It will dry completely in 4 or 5 minutes to a clear film that remains on the lids for several hours — it cannot be seen by others, nor will it interfere with vision. 4. The medication should be washed off the lids once or twice a day. However, it should be reapplied after each washing. **METHOD II: On the Lid.** 1. Wash hands carefully. With head tilted back and eye closed, drop 1 drop onto the lid — preferably at the corner of the eye close to the nose. 2. Spread the medication over the full length of the upper and lower lids. 3. Do not wipe away any medication — it will dry in 4 to 5 minutes to a clear, invisible film which will remain on the lids for several hours. 4. The medication should be washed off the lids once or twice a day. However, it should be reapplied after each washing. **HOW SUPPLIED:** Blephamide sterile ophthalmic suspension is available in 5 ml and 10 ml plastic dropper bottles. To be dispensed on prescription only.

Reference:

1. Leibowitz HM, Kupferman A. Bioavailability and therapeutic effectiveness of topically administered corticosteroids. Trans Am Acad Ophthalmol Otolaryngol 1975 Jan/Feb; 79(1):OP78-OP88. The clinical significance of this animal data is unknown.

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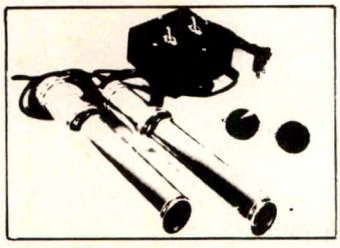
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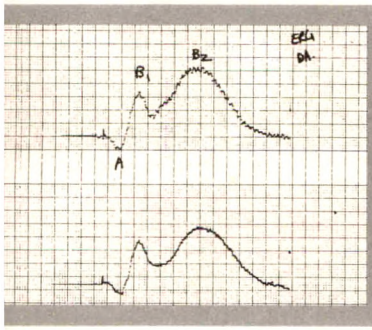
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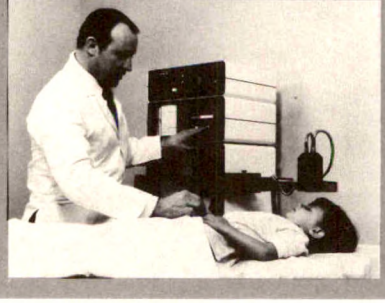


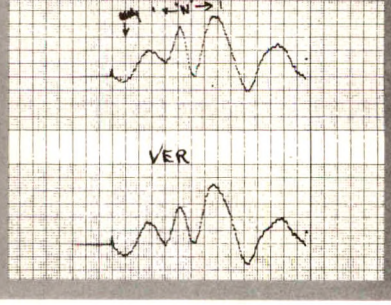
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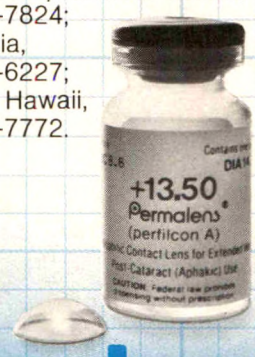
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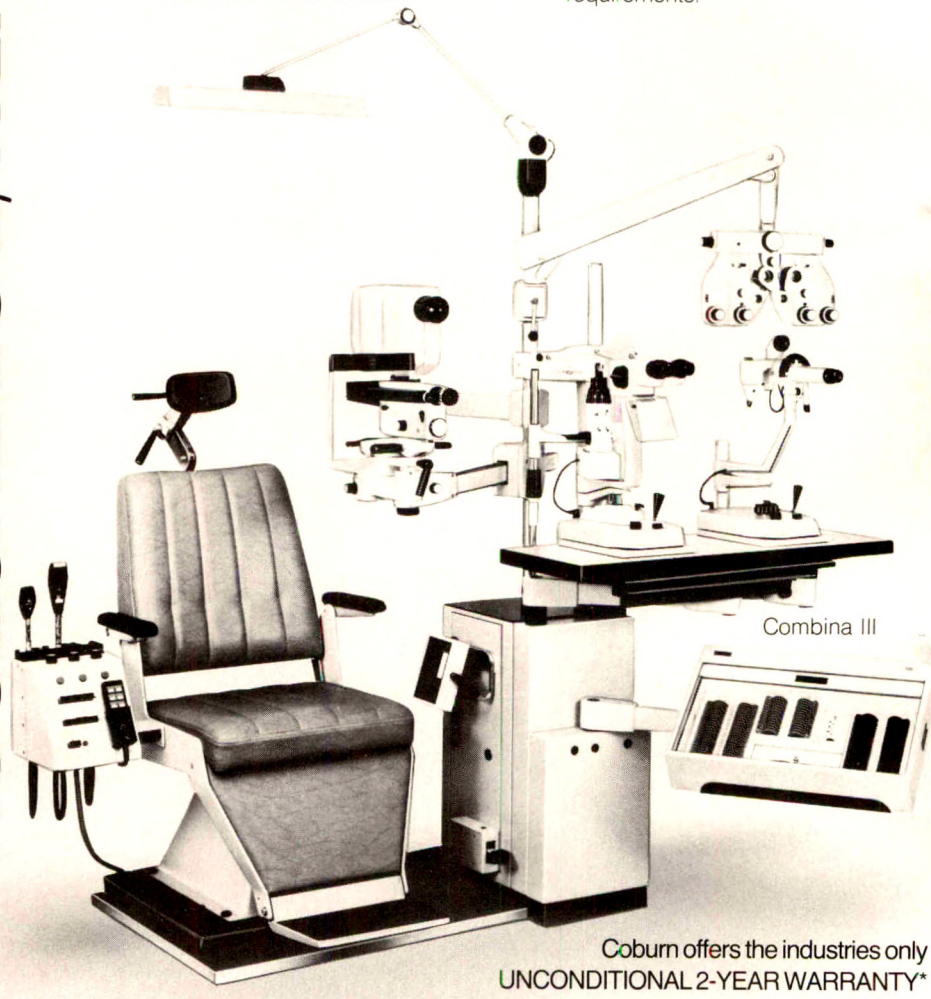
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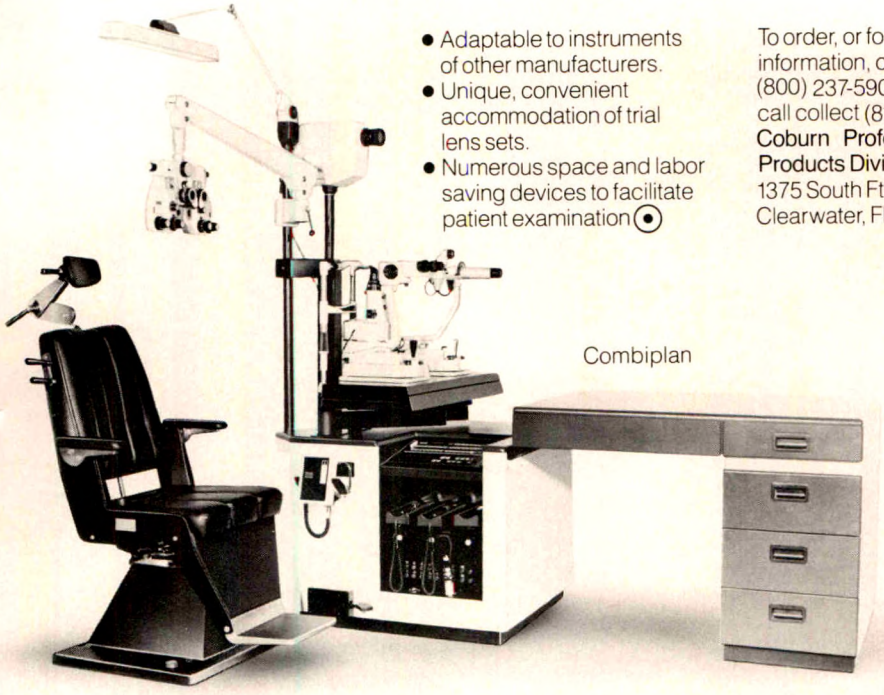


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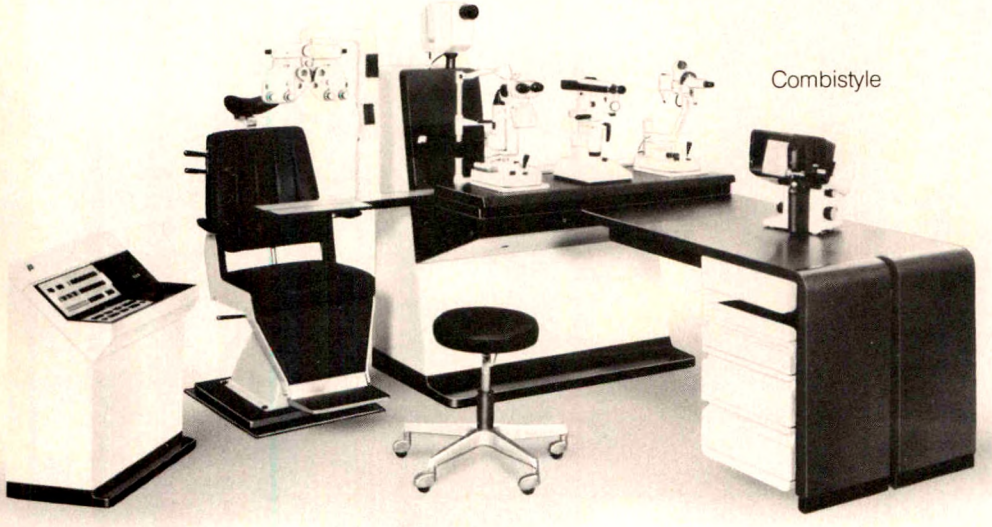
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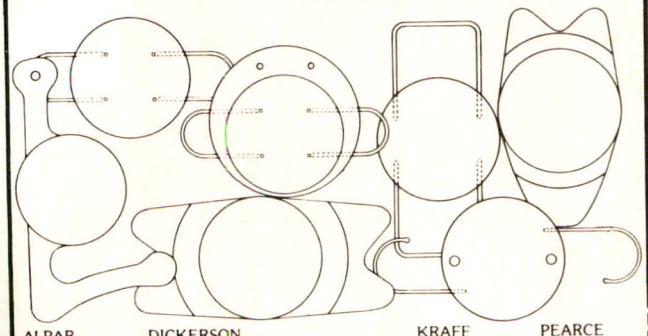
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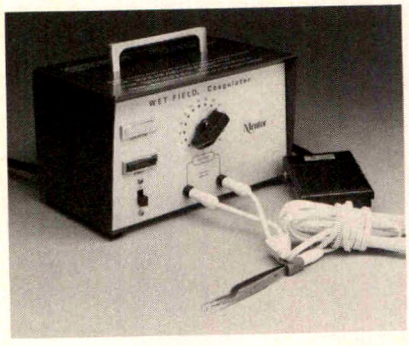
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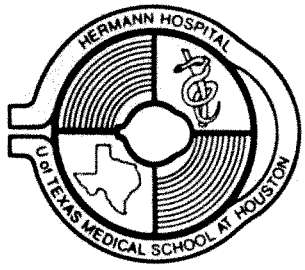
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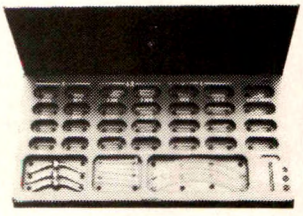
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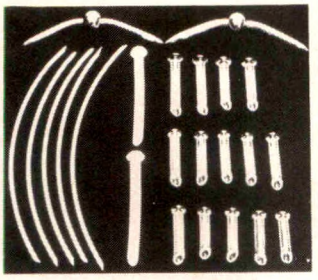
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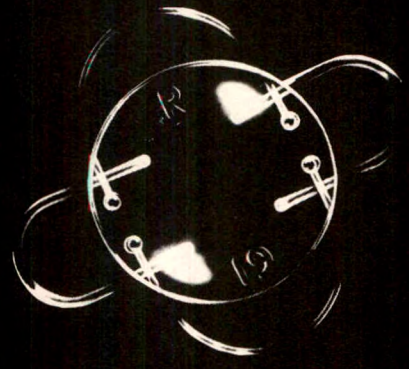
* L.T. Jones M.D., Dacryocystorhinostomy, American Journal of Ophthalmology, Volume 59, No. 5, May, 1965.
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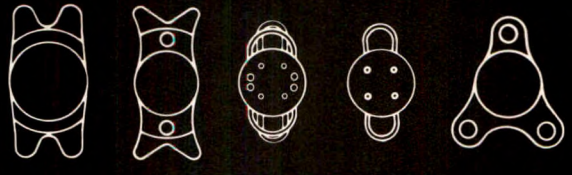
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Final classification of the less-than-effective indications requires further investigation.

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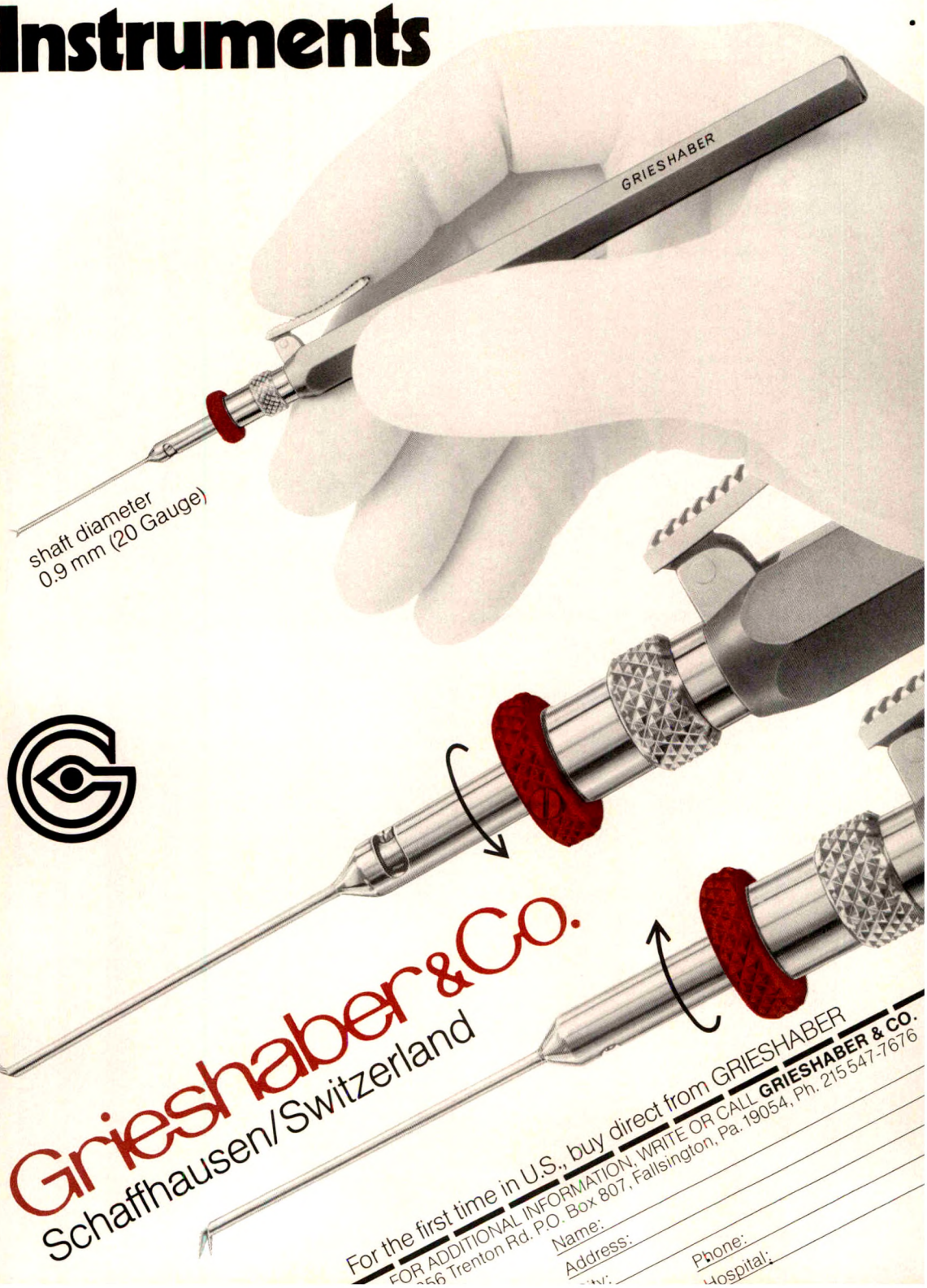
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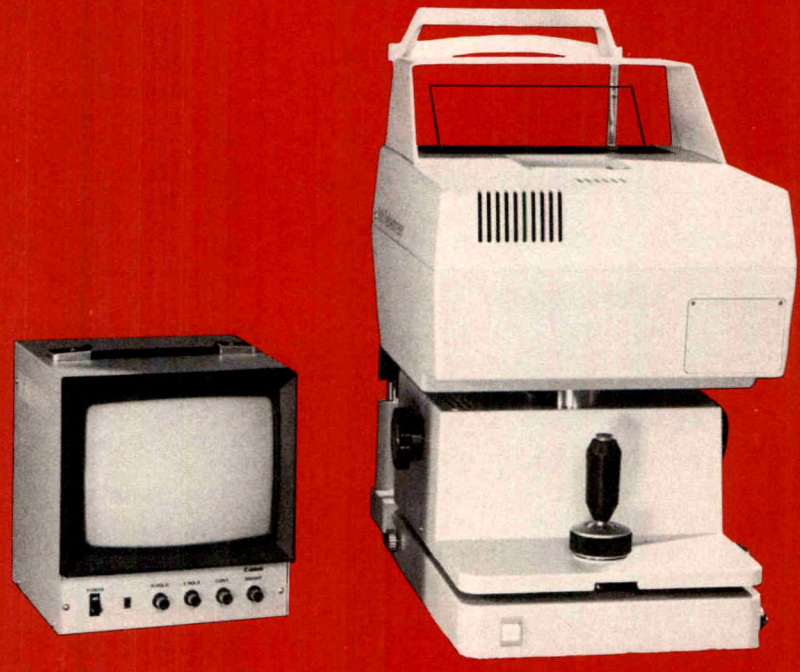
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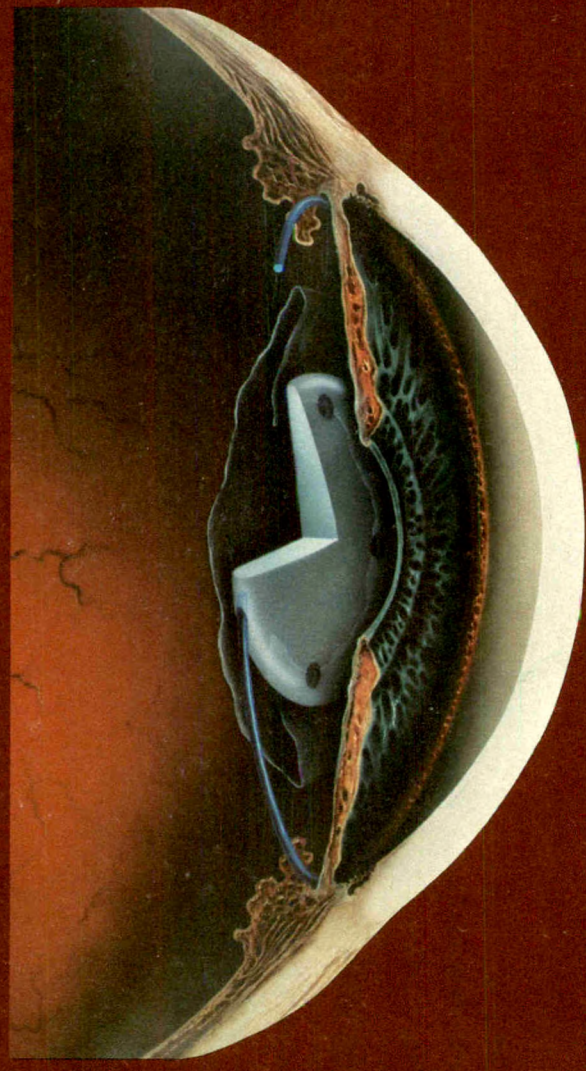
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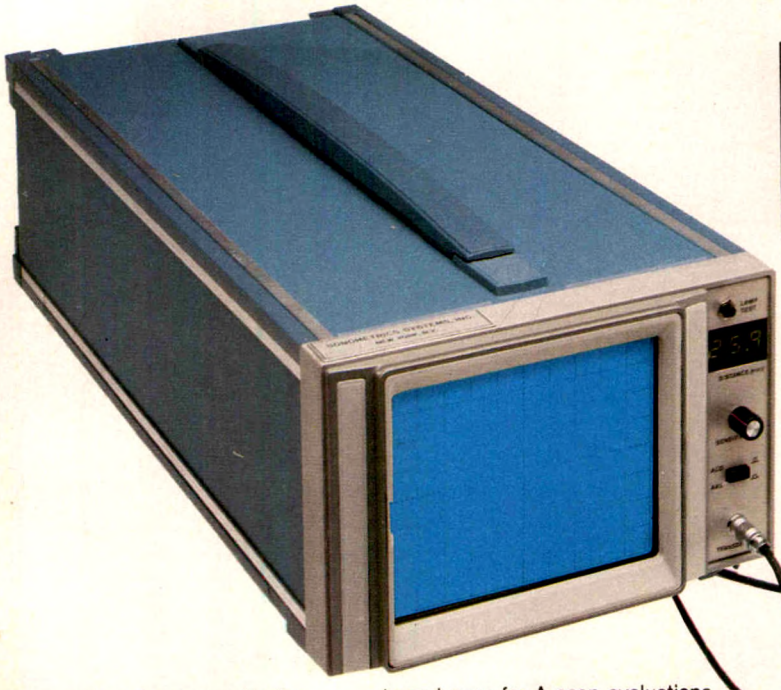
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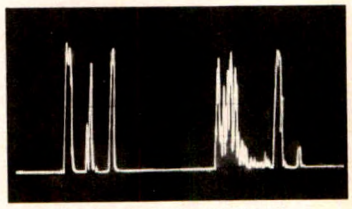
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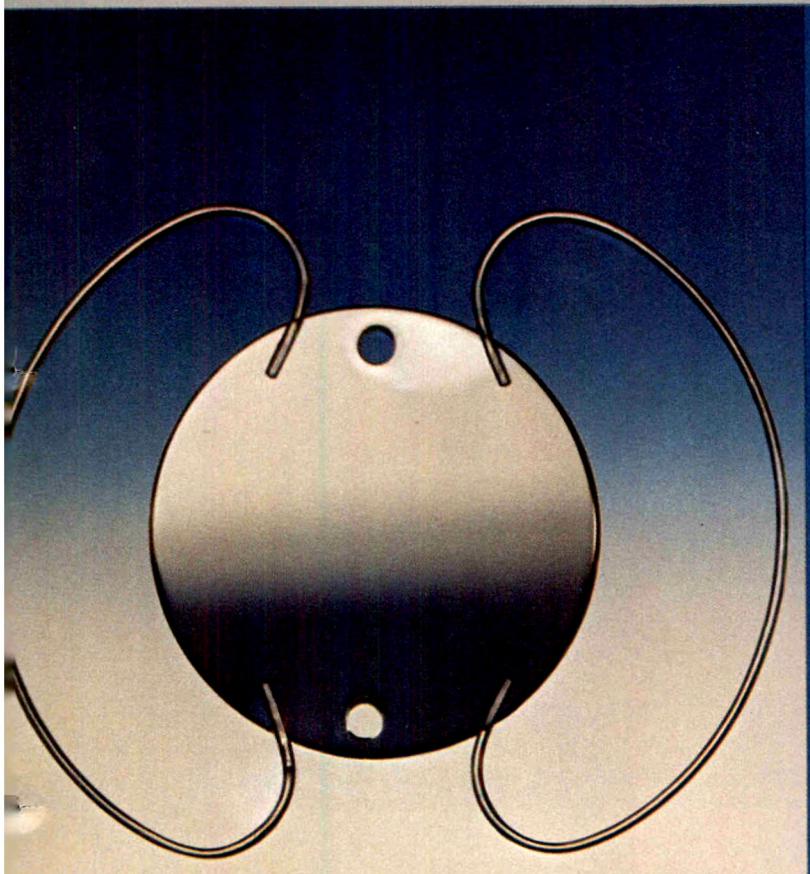
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The Eye of Horus
The origin of today's prescription symbol "Rx" goes back 5,000 years to the picture of the eye of the Egyptian god of healing—Horus.

DESCRIPTION: VIROPTIC® is the brand name for trifluridine (also known as trifluorothymidine, F_3TdR,F_3T), an antiviral drug for topical treatment of epithelial keratitis caused by Herpes simplex virus. The chemical name of trifluridine is 5-trifluoromethyl-2' -deoxyuridine.

VIROPTIC sterile opththalmic solution contains 1% trifluridine in an aqueous solution with acetic acid and sodium acetate (buffers), sodium chloride, and thimerosal 0.001% (added as a preservative).

CLINICAL PHARMACOLOGY: Trifluridine is a fluorinated pyrimidine nucleoside with *in vitro* and *in vivo* activity against Herpes simplex virus, types 1 and 2 and vacciniavirus. Some strains of Adenovirus are also inhibited *in vitro.*

Intraocular penetration of trifluridine occurs after topical instillation of VIROPTIC into human eyes. Decreased corneal integrity or stromal or uveal inflammation may enhance the penetration of trifluridine into the aqueous humor. Unlike the results of ocular penetration of trifluridine *in vitro,* 5-carboxy-2' -deoxyuridine was not found in detectable concentrations within the aqueous humor of the human eye.

INDICATIONS AND USAGE: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% is indicated for the treatment of primary keratoconjunctivitis and recurrent epithelial keratitis due to Herpes simplex virus, types 1 and 2. VIROPTIC is also effective in the treatment of epithelial keratitis that has not responded clinically to the topical administration of idoxuridine or when ocular toxicity or hypersensitivity to idoxuridine has occurred. In a smaller number of patients found to be resistant to topical vidarabine, VIROPTIC was also effective.

The clinical efficacy of VIROPTIC in the treatment of stromal keratitis and uveitis due to Herpes simplex or ophthalmic infections caused by vacciniavirus and Adenovirus has not been established by well-controlled clinical trials. VIROPTIC has not been shown to be effective in the prophylaxis of Herpes simplex virus keratoconjunctivitis and epithelial keratitis by well-controlled clinical trials. VIROPTIC is not effective against bacterial, fungal or chlamydial infections of the cornea or nonviral trophic lesions.

During controlled multicenter clinical trials, 92 of 97 (95%) patients (78 of 81 with dendritic and 14 of 16 with geographic ulcers) responded to VIROPTIC therapy as evidenced by complete corneal re-epithelialization within the 14 day therapy period. In these controlled studies, 56 of 75 (75%) patients (49 of 58 with dendritic and 7 of 17 with geographic ulcers) responded to idoxuridine therapy. The mean time to corneal re-epithelialization for dendritic ulcers (6 days) and geographic ulcers (7 days) was similar for both therapies. In other clinical studies, VIROPTIC was evaluated in the treatment of Herpes simplex virus keratitis in patients who were unresponsive or intolerant to the topical administration of idoxuridine or vidarabine. VIROPTIC was effective in 138 of 150 (92%) patients (109 of 114 with dendritic and 29 of 36 with geographic ulcers) as evidenced by corneal re-epithelialization. The mean time to corneal re-epithelialization was 6 days for patients with dendritic ulcers and 12 days for patients with geographic ulcers.

CONTRAINDICATIONS: VIROPTIC brand Trifluridine Ophthalmic Solution, 1%, is contraindicated for patients who develop hypersensitivity reactions or chemical intolerance to trifluridine.

WARNINGS: The recommended dosage and frequency of administration should not be exceeded (see Dosage and Administration).

PRECAUTIONS:

General: VIROPTIC brand Trifluridine Ophthalmic Solution, 1% should be prescribed only for patients who have a clinical diagnosis of herpetic keratitis.

VIROPTIC may cause mild local irritation of the conjunctiva and cornea when instilled but these effects are usually transient.

Although documented *in vitro* viral resistance to trifluridine has not been reported following multiple exposure to VIROPTIC, the possibility exists of viral resistance development.

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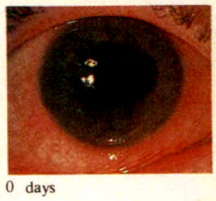
During controlled clinical trials 95% of patients (92 of 97) responded to VIROPTIC therapy. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 7 days, respectively.

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5 to 9 Drops Daily

One drop every 2 hours while awake until herpetic lesion has completely re-epithelialized, then one drop every 4 hours for 7 days post-re-epithelialization.



0 days



5 days after VIROPTIC therapy

Effective in 138 of 150 patients* Unresponsive or Intolerant to Idoxuridine or Vidarabine

In studies of patients unresponsive or intolerant to idoxuridine or vidarabine, VIROPTIC demonstrated 92% effectiveness. Dendritic and geographic ulcers were re-epithelialized in a mean time of 6 and 12 days, respectively.

*Data on file, Medical Division, Burroughs Wellcome Co.

Carcinogenesis, Mutagenesis, Impairment of Fertility: Mutagenic Potential. Trifluridine has been shown to exert mutagenic, DNA damaging and cell transforming activities in various standard *in vitro* test systems, and clastogenic activity in *Vicia faba* cells.

Although the significance of these test results is not clear or fully understood, there exists the possibility that mutagenic agents may cause genetic damage in humans.

Oncogenic Potential. The oncogenic potential of trifluridine is unknown at this time. The oncogenic potential of trifluridine in rodents is being evaluated.

Pregnancy: The drug should not be prescribed for pregnant women unless the potential benefits outweigh the potential risks.

Nursing Mothers: It is unlikely that trifluridine is excreted in human milk after ophthalmic instillation of VIROPTIC because of the relatively small dosage (≤ 5.0 mg/day), its dilution in body fluids and its extremely short half-life (approximately 12 minutes). The drug should not be prescribed for nursing mothers unless the potential benefits outweigh the potential risks.

ADVERSE REACTIONS: The most frequent adverse reactions reported during controlled clinical trials were mild, transient burning or stinging upon instillation (4.6%) and palpebral edema (2.8%). Other adverse reactions in decreasing order of reported frequency were superficial punctate keratopathy, epithelial keratopathy, hypersensitivity reaction, stromal edema, irritation, keratitis sicca, hyperemia, and increased intraocular pressure.

OVERDOSAGE: Overdosage by ocular instillation is unlikely because any excess solution should be quickly expelled from the conjunctival sac.

Acute overdosage by accidental oral ingestion of VIROPTIC has not occurred. However, should such ingestion occur, the 75 mg dosage of trifluridine in a 7.5 ml bottle of VIROPTIC is not likely to produce adverse effects. Single intravenous doses of 15-30 mg/kg/day in children and adults with neoplastic disease produce reversible bone marrow depression as the only potentially serious toxic effect and only after 3-5 courses of therapy. The acute oral LD_{50} in the mouse and rat was 4379 mg/kg or higher.

DOSAGE AND ADMINISTRATION: Instill one drop of VIROPTIC Ophthalmic Solution, 1% onto the cornea of the affected eye every two hours while awake for a maximum daily dosage of nine drops until the corneal ulcer has completely re-epithelialized. Following re-epithelialization, treatment for an additional seven days of one drop every four hours while awake for a minimum daily dosage of five drops is recommended.

If there are no signs of improvement after seven days of therapy, or complete re-epithelialization has not occurred after 14 days of therapy, other forms of therapy should be considered. Continuous administration of VIROPTIC for periods exceeding 21 days should be avoided because of potential ocular toxicity.

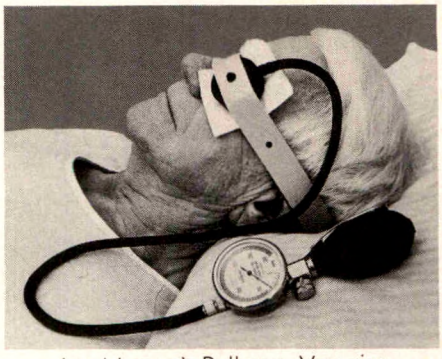
HOW SUPPLIED: VIROPTIC Ophthalmic Solution, 1% is supplied as a sterile ophthalmic solution in a plastic Drop-Dose® dispenser bottle of 7.5 ml. Store under refrigeration 2° to 8° C (36° to 46° F).

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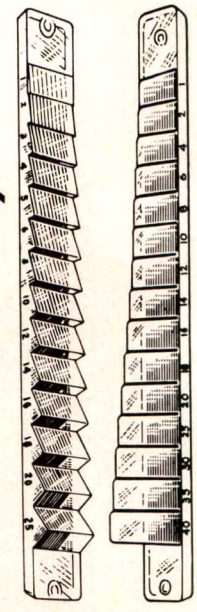
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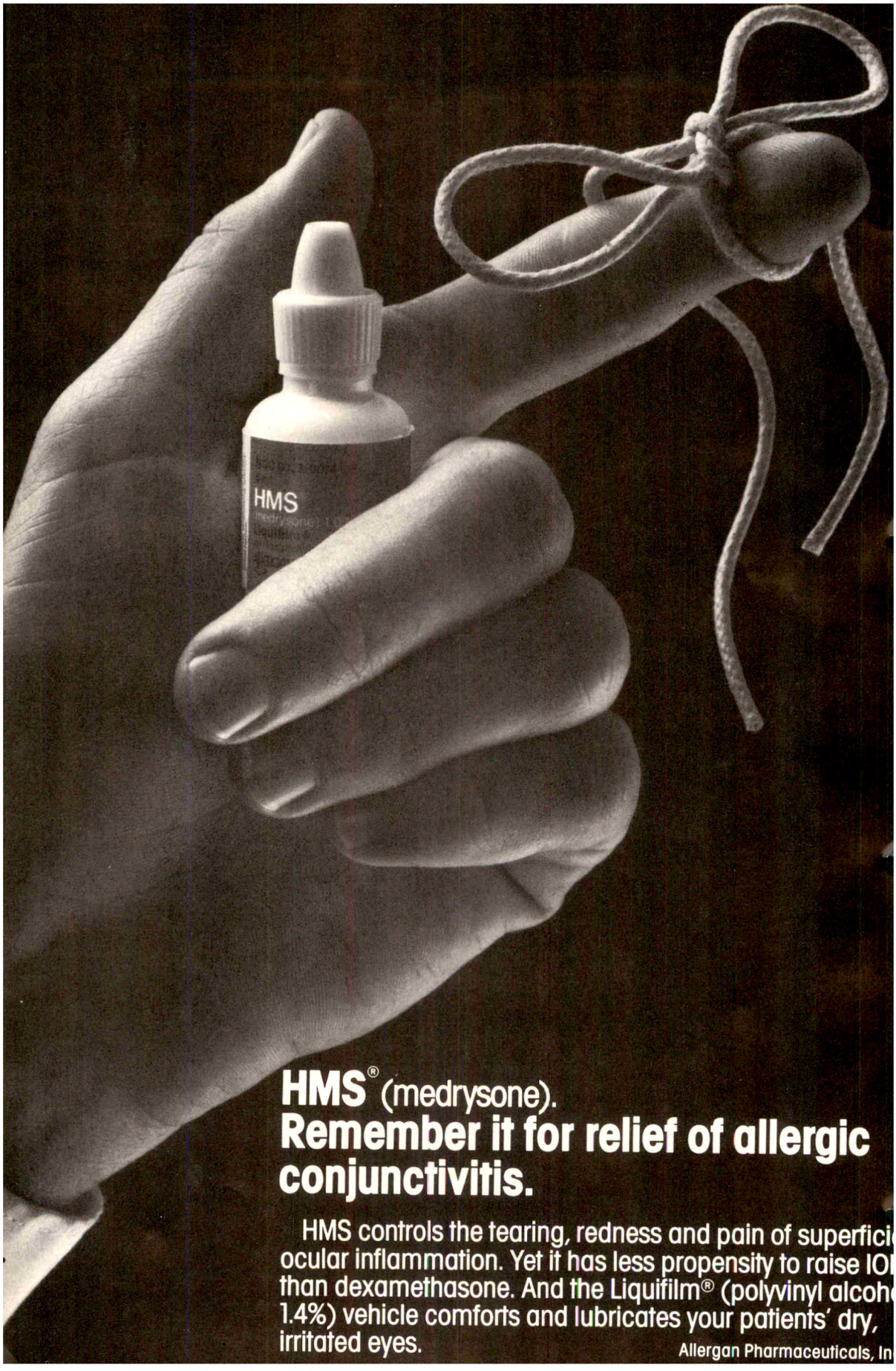


HMS®(medrysone)...memorable therapy.

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CONTAINS: medrysone®, 1.0%, with: Liquifilm (polyvinyl alcohol) 1.4%, benzalkonium chloride, edetate disodium, sodium chloride, potassium chloride, sodium phosphate monobasic, monohydrate, sodium phosphate dibasic, anhydrous, hydroxypropyl methylcellulose, purified water, and sodium hydroxide or hydrochloric acid if needed to adjust pH. ®Licensed under U.S. Patent 2,968,655. Medrysone (previously known as hydroxymesterone) belongs to the family of steroids, related structurally more to progesterone than to other available corticosteroids. It is a white crystalline powder, sparingly soluble in water, with a melting point of approximately 162°C, and empirical formula of $C_{22}H_{32}O_3$ and a molecular weight of 344.50. The specific rotation of a 1% solution of medrysone in chloroform is between +184° and +192°. It has a characteristic ultraviolet absorption maximum of 242±1 millimicrons in 95% ethanol solution. **Chemical Name:** 11β-Hydroxy-6α-methylpregn-4-ene-3, 20 dione. **ACTIONS:** HMS (medrysone) is a synthetic steroid with topical anti-inflammatory and anti-allergic activity. Steroids inhibit the inflammatory response to inciting agents of mechanical, chemical, or immunological nature of edema, fibrin deposition, capillary dilatation and leucocyte migration, capillary proliferation, deposition of collagen and scar formation. No generally accepted explanation of this steroid property has been advanced. HMS (medrysone) has less anti-inflammatory potency than 0.1% dexamethasone. Data from 2 uncontrolled studies[1-2] indicate that in patients with increased intraocular pressure and in those susceptible to a rise in intraocular pressure, there is less effect on pressure with HMS than with dexamethasone or betamethasone.
INDICATIONS: HMS (medrysone) is effective in the treatment of allergic conjunctivitis, vernal conjunctivitis, episcleritis, and epinephrine sensitivity.
CONTRAINDICATIONS: HMS (medrysone) is contraindicated in the following conditions: Acute superficial herpes simplex. Viral diseases of the conjunctiva and cornea. Ocular tuberculosis. Fungal diseases of the eye. Hypersensitivity to any of the components of the drug. **WARNINGS:** 1. Acute purulent untreated infections of the eye may be masked, enhanced or activated by the presence of steroid medication. 2. Corneal or scleral perforation occasionally has been reported with prolonged use of topical steroids. In high dosage they have been associated with corneal thinning. 3. Prolonged use of topical steroids may increase intraocular pressure, with possible resultant glaucoma, damage to the optic nerve, and defects in visual acuity and fields of vision. However, data from 2 uncontrolled studies[1-2] indicate that in patients with increased intraocular pressure and in those susceptible to a rise in intraocular pressure upon application of topical steroids, there is less effect on pressure with HMS than with dexamethasone or betamethasone. 4. Prolonged use of topical corticosteroids may rarely be associated with development of posterior subcapsular cataracts. 5. Systemic absorption and systemic side effects may result with the use of topical steroids. 6. HMS® (medrysone) is not recommended for use in iritis and uveitis as its therapeutic effectiveness has not been demonstrated in these conditions. 7. Steroid medication in the presence of stromal herpes simplex requires great caution; frequent slit lamp microscopy is suggested. 8. Prolonged use may aid in the establishment of secondary ocular infections from fungi and viruses liberated from ocular tissue. **Use In Pregnancy:** The use of topical steroids in pregnancy should be limited to conditions serious enough to warrant such treatment, so that possible risk to the fetus may be justified by the expected benefit to the mother. **PRECAUTIONS:** With prolonged use of HMS (medrysone) the intraocular pressure and lens should be examined periodically. In persistent corneal ulceration where a steroid has been used, or is in use, fungal infection should be suspected. **ADVERSE REACTIONS:** Occasional transient stinging and burning may occur on instillation. **DOSAGE AND ADMINISTRATION:** One drop instilled in the conjunctival sac up to every four hours. Shake well before using. DO NOT FREEZE. **HOW SUPPLIED:** As a sterile suspension in 5 ml. and 10 ml. plastic dropper bottles. On prescription only. NOTE: 5 ml bottle filled to 1/2 capacity and 10 ml bottle filled to 2/3 capacity for proper drop control.

REFERENCES: 1. Becker, B. and Kolker, A.E.: "Intraocular Pressure Response to Topical Corticosteroids" in Ocular Therapy, Complications and Management, Irving Leopold, Ed. St. Louis, C.V. Mosby, 1967, pp. 79-83. 2. Spaeth, G. Hydroxymethylprogesterone, Arch. Oph. 75: 783-787, 1966.

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AMERICAN JOURNAL OF OPHTHALMOLOGY™

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HISTOPATHOLOGIC AND CLINICAL ASSOCIATIONS OF SCLERITIS AND GLAUCOMA

KIRK R. WILHELMUS, M.D., IAN GRIERSON, PH.D., AND PETER G. WATSON, F.R.C.S.

London, England

Of 92 enucleated eyes with scleral inflammation and 114 eyes from 81 patients with scleritis, 45 (49%) and 20 (18%), respectively, showed evidence of increased intraocular pressure. Damage to the trabecular meshwork by iridocyclitis, overlying corneoscleral inflammation, or peripheral anterior synechiae was the most common cause of increased intraocular pressure. Other causes included topical corticosteroid use, angle neovascularization, and posterior scleritis with secondary angle closure.

Increased intraocular pressure may occur in eyes with scleritis as a consequence of the accompanying scleral edema and uveal inflammation. Glaucoma, particularly in combination with uveitis, is the most common reason for enucleation in uncontrolled scleritis. Fraunfelder and Watson[1] noted the histologic changes of glaucoma in 46% of 30 eyes enucleated because of scleritis. In a clinical survey of 301 eyes with scleritis, Watson and Hayreh[2] found glaucoma in 12%; McGavin and associates,[3] in another clinical series of 73 eyes, showed that 22% had increased intraocular pressures. In these two clinical investigations anti-inflammatory therapy alone was sufficient to control the intraocular pressure in 82% of the eyes; the remaining patients with increased intraocular pressures required long-term antiglaucoma medication or a filtering operation.

We investigated the histopathologic and clinical associations between scleritis and glaucoma to determine the causes of increased intraocular pressure in eyes with scleral disease.

SUBJECTS AND METHODS

Histologic survey—We reviewed 92 enucleated eyes with histopathologic evidence of scleritis. The patients ranged in age from 41 to 86 years (mean, 67 ± 12 years) at the time of enucleation. The female-to-male ratio was 1.8:1. We noted the following features: duration of corticosteroid treatment; the extent and location of the scleritis; the presence of scleral perforation at the time of enucleation; the presence of inflammatory cells in the trabecular meshwork, collector

From the Scleritis Clinic, Moorfields Eye Hospital (Drs. Wilhelmus and Watson); and the Department of Pathology (Dr. Grierson), Institute of Ophthalmology, London, England. This study was supported by a fellowship from the Friends of Moorfields Eye Hospital (Dr. Wilhelmus).

Reprint requests to Kirk R. Wilhelmus, M.D., Professorial Unit, Moorfields Eye Hospital, City Road, London EC1V 2PD, England.

vessels, and uvea; the extent of peripheral anterior synechiae; rubeosis iridis; vitreal hemorrhage; atrophy and fibrosis of the anterior uvea; excavation of the optic nerve; and degeneration of the ganglion cell nerve fiber layer of the retina.

Clinical review—We also performed a clinical review of 114 eyes with scleritis from 81 consecutive patients examined here. The patients' ages at the time of the initial diagnosis of scleritis ranged from 13 to 79 years (mean, 48 ± 12 years). The female-to-male ratio was 1.4:1. Features noted during observation periods of one to five years included the following: type and location of the scleral inflammation; the presence of anterior uveitis; the extent and location of scleral transparency, thinning, staphyloma, or perforation; a history of topical or oral corticosteroid therapy; the highest recorded intraocular pressure; and the configuration of the anterior chamber angle. Selected patients underwent scleral fluorescein angiographic and A-scan and B-scan ultrasonographic tests. Corticosteroid-induced ocular hypertension was judged to be present if the intraocular pressure increased to more than 25 mm Hg on each occasion that a patient with a gonioscopically normal angle used a topical corticosteroid.

RESULTS

Histologic survey—Of the 92 enucleated eyes with scleritis, 33 had scleritis limited to a position anterior to the equator of the globe, 52 eyes had combined anterior and posterior scleritis, and seven had posterior scleritis only (Table 1).

Forty-five (49%) of the 92 enucleated eyes had glaucoma (Table 2). Of the glaucomatous eyes, 36 (80%) had a focus of scleritis overlying the outflow system (Fig. 1), whereas similar foci were found in only 25 (53%) of the 47 nonglaucomatous eyes ($P < .005$). Additionally, anterior uveitis was present in 38 (84%) of the glaucomatous eyes and 21 (45%) of the eyes without evidence of glaucoma ($P < .005$). Of the perforated eyes, eight had glaucoma and 12 did not.

Fourteen eyes had open-angle glaucoma; nine of these 14 had anterior uveitis with inflammatory cells within the trabecular meshwork and around the collector channels (Fig. 2). Four eyes showed no evidence of active trabecular inflammation but did have uveal perivasculitis and inflammatory cuffing around the corneoscleral limbal collector vessels (Fig. 3). One case was further complicated by the presence of pseudoexfoliation material.

TABLE 1

SUMMARY OF CLINICAL FEATURES OF EYES WITH SCLERITIS

Patient Data	Histologic Survey (92 Eyes)		Clinical Review (114 Eyes)	
	No.	%	No.	%
Age (yrs, mean ± 1 S.D.)	67 ± 12		48 ± 12	
Sex (female:male ratio)	1.8:1		1.4:1	
Location of scleritis				
Anterior	33	36	100	88
Anterior and posterior	52	57	10	9
Posterior	7	7	4	3
Corneoscleral limbal inflammation	61	66	65	57
Anterior uveitis	59	64	29	25
Perforation	20	22	4	3
Increased intraocular pressure	45	49	20	18

TABLE 2

STATUS OF ANTERIOR CHAMBER ANGLE IN EYES WITH SCLERITIS AND INCREASED INTRAOCULAR PRESSURE

Angle	Histologic Survey		Clinical Review	
	No.	%	No.	%
Open	14	15	18	16
Closed	17	18	2	2
Neovascular	14	15	0	0

A total of 17 eyes had partial or total angle closure (Figs. 4 and 5). Of these, 15 had accompanying anterior uveitis, and seven had evidence of posterior synechiae with fibrous development between the iris and lens.

A neovascular membrane occluding the angle was present in 14 of the glaucomatous eyes. One of these eyes had endothelialization of the anterior chamber angle and anterior surface of the iris.[4]

Six globes (four from women and two from men) had glaucoma and posterior scleritis. Of these six eyes, two had open angles, two had angle closure, and two had neovascularization of the angle.

Clinical review—Of the 114 eyes with scleritis, the focus of scleral inflammation was located in the anterior sclera alone in 100, both anterior and posterior to the ora serrata in ten, and behind the ora in four. The corneoscleral limbus was involved in 65 of the patients with anterior loci of scleritis.

Twenty-nine (25%) of the eyes with scleritis had had at least one episode of anterior uveitis. Scleral translucency or thinning was found in 31 eyes (27%). Four eyes, all with increased intraocular pressures, developed marked staphyloma (Fig. 6). One of these eyes had become perforated as a result of ocular trauma. Scleral perforation occurred in three eyes without increased intraocular pressures; in two the perforation occurred spontaneously in an area of inflammatory necrotizing scleritis and in one it was caused by trauma.

Twenty eyes of 14 patients had increased intraocular pressures (Tables 2 and 3). The highest recorded intraocular

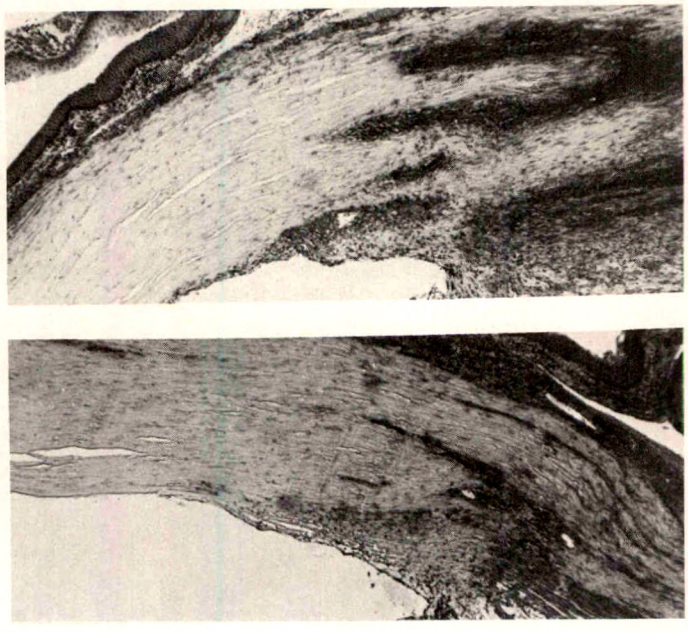


Fig. 1 (Wilhelmus, Grierson, and Watson). Diffuse scleral inflammation involving the corneoscleral limbus in two eyes with open-angle glaucoma. Top, Lymphocytic cuffing around Schlemm's canal and hyalinization of the trabecular meshwork (×45). Bottom, Mononuclear cells have obliterated Schlemm's canal and extend around the intrascleral vascular channels (×45).

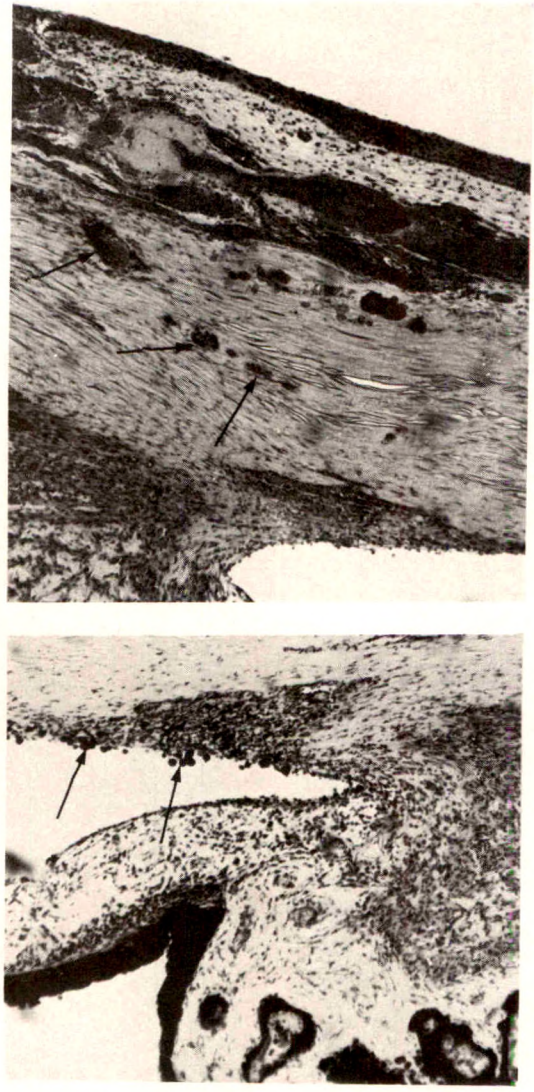


Fig. 2 (Wilhelmus, Grierson, and Watson). Top, Open anterior chamber angle with occluded intrascleral vessels (arrows) in a glaucomatous eye (×45). Bottom, Trabecular meshwork is occluded by numerous lymphocytes and large mononuclear cells (arrows) (×60).

pressures ranged from 26 to 54 mm Hg. One patient developed bilateral closed angles associated with posterior scleritis (Fig. 7). Eighteen eyes had increased intraocular pressures with open angles. In these eyes, the increased intraocular pressure was related to the use of a topical corticosteroid in seven, caused by anterior uveitis in six, and associated with perilimbal scleral edema and vascular engorgement with gonioscopically normal angles in five (Fig. 8). We did not detect any cases of neovascular glaucoma.

The increased intraocular pressures of the patients were controlled, although glaucomatous visual field loss occurred in two eyes. The seven eyes with corticosteroid-associated ocular hypertension returned to normal after the topical corticosteroid was discontinued. The intraocular pressures of the six eyes in which the increased pressure was caused by anterior chamber and trabecular inflammation were controlled with a combination of oral and topical corticosteroids.

In the five eyes with circumferential corneoscleral inflammation and open angles, the intraocular pressure was returned to normal in one by oral indomethacin, in one by oral prednisolone, in one by oral prednisolone and cyclophosphamide, and in one by oral prednisolone, azathioprine, and acetazolamide, and topical echothiophate. The intraocular pressure of one patient with perilimbal scleral inflammation could not be controlled, and she later underwent a trabeculectomy (Case 13).

CLASSIFICATION—Although scleritis occurs in only 0.5% of enucleated eyes,[5] increased intraocular pressure is common.[6] From the appearance of pathologic specimens in this series, glaucoma appeared to be an ominous sign in the course of scleritis.

We classified the types of glaucoma by morphologic findings in the anterior chamber angle. Of the 92 enucleated eyes with scleritis, 14 (15%) had open-angle glaucoma, 17 (18%) had closed-angle glaucoma, and 14 (15%) had neovascular glaucoma. In the clinical review of 114 eyes, 18 (16%) had open-angle and two (2%) had acute closed-angle hypertension.

Open-angle glaucoma—In the clinic population studied by Watson and Hay-



Fig. 3 (Wilhelmus, Grierson, and Watson). Intrascleral and deep scleral vessels are narrowed with perivascular lymphocytic cuffing (×120).

reh,[2] 12% of the eyes with scleritis had open-angle glaucoma. They differentiated primary open-angle glaucoma from secondary glaucoma by gonioscopically normal angles, a persistently increased intraocular pressure despite anti-inflammatory therapy, and a response to miotics and acetazolamide. Increased intraocular pressure may occur because of a preexisting abnormal outflow system that is further impaired by edema of the corneoscleral limbus and inflammation of the angle structures.[7]

Active uveal inflammation with occlusion of the trabecular meshwork by inflammatory cells was the principal mechanism of increased intraocular pressure in most of the eyes with scleritis that we studied. Pseudoexfoliation was present in one eye, and four eyes had open angles without trabecular inflammation, suggesting a resolved or secondary form of inflammatory glaucoma.

In the 18 eyes in the clinical series with open-angle hypertension, the increased intraocular pressure appeared to be caused by a secondary form of open-angle hypertension from anterior uveitis, topical corticosteroid use, or perilimbal inflammation and edema. Topical and oral anti-inflammatory treatment of eyes with hypertensive uveitis controlled the increased intraocular pressure. Discontinuation of the topical corticosteroid led to control of the intraocular pressure in those eyes with corticosteroid-induced ocular hypertension.

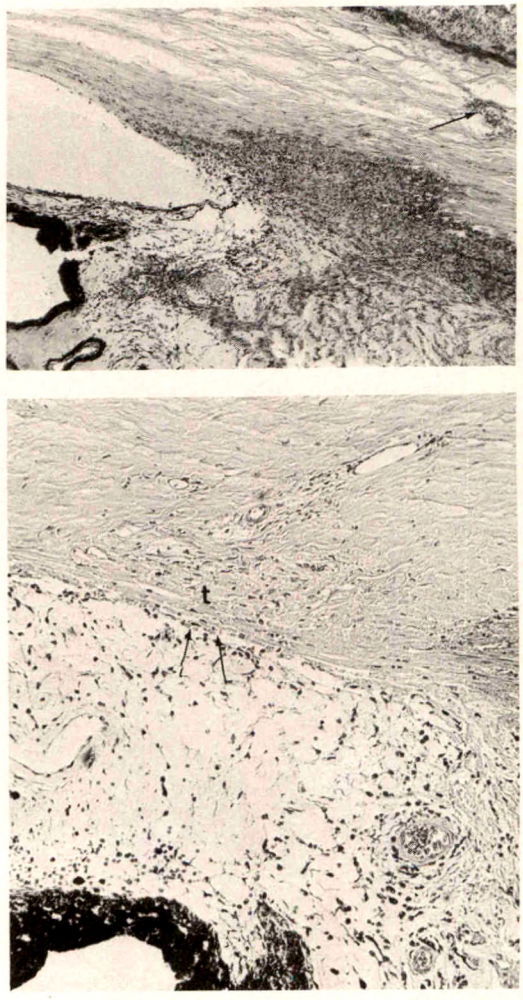


Fig. 4 (Wilhelmus, Grierson, and Watson). Top, The anterior chamber angle is only partially open and inflammatory cells abound in the trabecular meshwork and around an intrascleral vessel (arrow) (×60). Bottom, Mononuclear cells (arrows) are present at the site of contact between iris and cornea. The trabecular meshwork (t) is thickened and hypocellular, and Schlemm's canal is obliterated (×200).

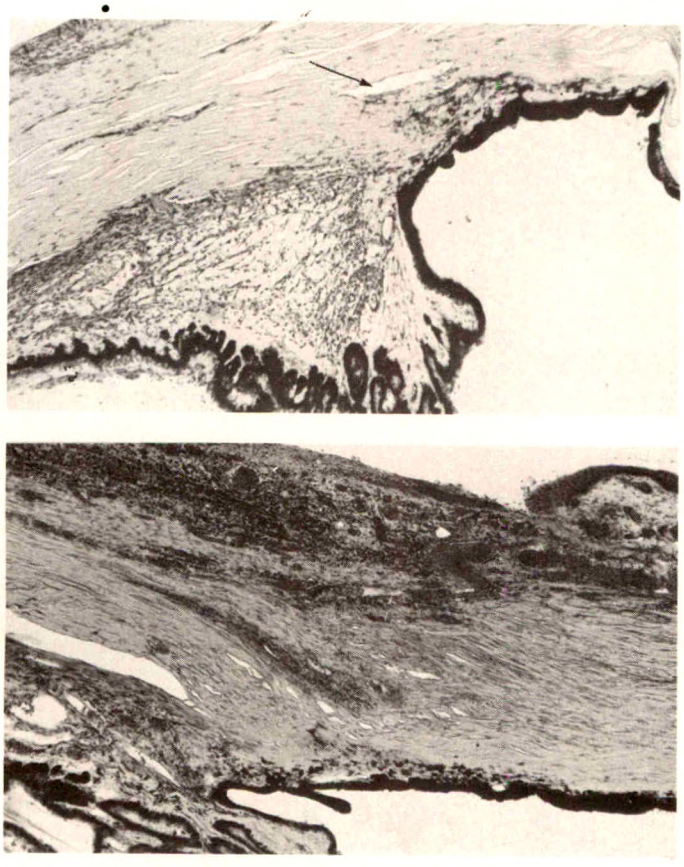


Fig. 5 (Wilhelmus, Grierson, and Watson). Complete angle closure with a thin and atrophic iris. Top, Peripheral iridocorneal adhesion with obliteration of the trabecular meshwork although Schlemm's canal can be identified (arrow) (×45). Bottom, Complete occlusion of the angle with hyalinization of the outflow channels (×45).

Systemic corticosteroid and immunosuppressive therapy[8] was administered to four patients with severe corneoscleral inflammation and open-angle hypertension. Two of these patients subsequently developed glaucomatous visual field loss; one received supplementary antiglaucoma medication and the other underwent a trabeculectomy.

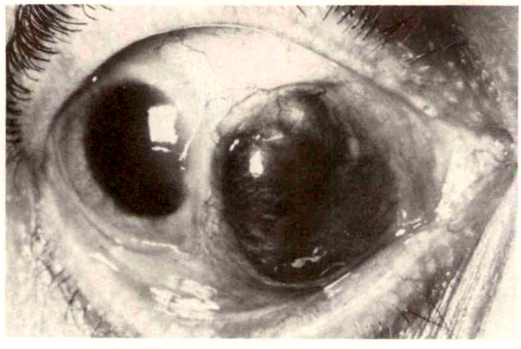


Fig. 6 (Wilhelmus, Grierson, and Watson). Case 13. A 53-year-old woman with rheumatoid arthritis and necrotizing scleritis developed a staphyloma of the medial sclera with a persistently increased intraocular pressure of 50 mm Hg. The pressure could not be controlled, and she underwent a successful trabeculectomy.

Closed-angle glaucoma—Angle closure may occur in association with scleritis because of iridocyclitis with anterior or posterior synechiae or ciliary body edema.[9] Peripheral anterior synechiae and pupillary block with iridolenticular adhesions from an uncontrolled intraocular inflammatory reaction appeared to be the major reason for chronic closed-angle glaucoma in the 17 eyes with scleritis. Rarely, acute angle closure may occur in association with posterior scleritis; an annular choroidal detachment may result in an anterior rotation of the ciliary body at the scleral spur with subsequent closed-angle glaucoma.[10]

TABLE 3

CLINICAL FEATURES OF 14 PATIENTS (20 EYES) WITH SCLERITIS AND OCULAR HYPERTENSION

Patient, Sex, Age (yrs)	Involved Eye	Type of Scleritis	Uveitis	Staphyloma	Type of Ocular Hypertension
1, F, 48	Both	Posterior	−	−	Closed-angle
2, F, 53	Both	Anterior diffuse	+	−	Open-angle, inflammatory
3, F, 51	Left	Anterior diffuse	+	−	Open-angle, inflammatory
4, M, 55	Right	Anterior diffuse	+	+	Open-angle, inflammatory
5, M, 44	Both	Necrotizing	+	−	Open-angle, inflammatory
6, M, 62	Left	Anterior diffuse	−	−	Open-angle, corticosteroid-induced
7, M, 41	Right	Anterior diffuse	−	−	Open-angle, corticosteroid-induced
8, M, 55	Left	Anterior diffuse	−	−	Open-angle, corticosteroid-induced
9, M, 43	Left	Anterior diffuse	−	+P*	Open-angle, corticosteroid-induced
10, M, 34	Both	Anterior diffuse	+	−	Open-angle, corticosteroid-induced
11, F, 49	Right	Necrotizing	−	−	Open-angle, corticosteroid-induced
12, M, 33	Both	Anterior diffuse	−	−	Open-angle, perilimbal scleritis
13, F, 53	Right	Necrotizing	−	+	Open-angle, perilimbal scleritis
14, F, 54	Both	Necrotizing	−	+/−	Open-angle, perilimbal scleritis

*P, perforation.

Neovascular glaucoma—Neovascular glaucoma has not been observed clinically in patients with scleritis. In the 14 eyes in this group, it was not possible to determine the origin of the neovascularization. However, most of these eyes had had intraocular inflammations that may have predisposed them to persistently increased intraocular pressure with subsequent retinal ischemia. In two eyes a focus of juxtapapillary scleral inflammation suggested a direct extension of the inflammatory reaction to the central retinal vessels. The high incidence of neovascular glaucoma in enucleated eyes with scleritis should lead us to suspect vascular occlusion and scleritis as a cause of intractable glaucoma.

DISCUSSION

Anterior uveitis was associated with 84% of the glaucomatous enucleated eyes and 33% of the clinical cases with open-angle hypertension. Iridocyclitis may contribute directly to ocular hypertension by direct blockage of the anterior chamber angle and by peripheral anterior synechiae formation. The damage to the trabecular meshwork caused by uveal and corneoscleral inflammation was the most common cause of increased intraocular pressure in eyes with scleritis.

Scleral inflammation overlying the anterior chamber angle may have a direct inhibitory effect on the aqueous outflow system. Increased intraocular pressure may occur when the scleral inflammation extends around the entire corneoscleral limbal circumference. Ocular hypertension caused by this is more difficult to control, particularly in those eyes with necrotizing scleritis.

A related mechanism appeared to be present in four enucleated eyes with open-angle glaucoma but without demonstrable trabecular meshwork abnormality. Histopathologically each of these eyes had lymphocytic cuffing around the intrascleral outflow channels with perivasculitis of the anterior uvea. While these eyes may have had primary open-angle glaucoma, corticosteroid-induced glaucoma, or a previous inflammatory glaucoma with microstructural damage to the meshwork, the inflammation around the outflow channels may have resulted in an increased resistance to aqueous outflow. Cases of increased intraocular pressure without uveitis but with gonioscopically

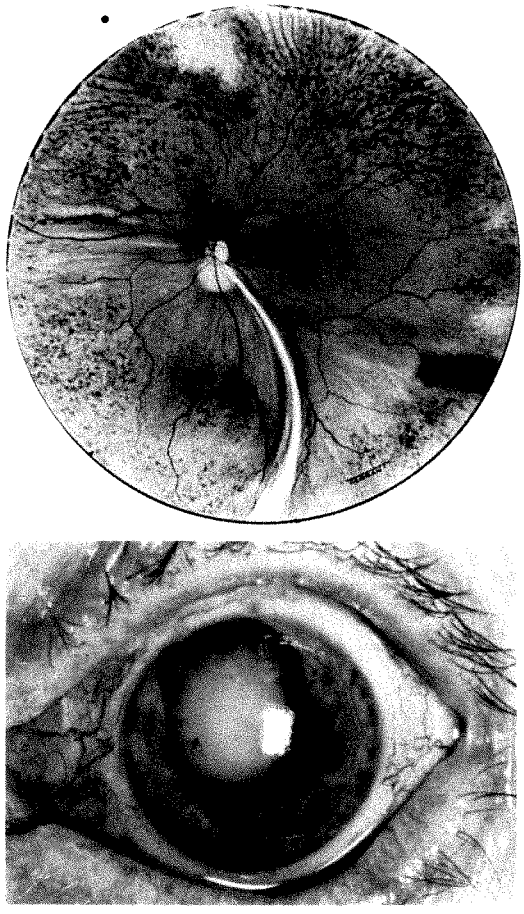


Fig. 7 (Wilhelmus, Grierson, and Watson). Case 1. A 53-year-old woman developed bilateral retinal detachments caused by posterior scleritis with secondary angle closure and an intraocular pressure of 35 mm Hg. Top, Fundus drawing of the left eye shows an inferonasal choroidal mass with overlying retinal detachment, peripheral pigment mottling, and choroidal folds. A- and B-scan ultrasonographic tracings demonstrated a shallow anterior chamber of 1.5 mm, a thickened posterior sclera of 2 mm with retrobulbar edema, and a peripheral choroidal detachment. Bottom, A sector iridectomy was performed on the left eye; diffuse anterior scleritis is present temporally and nasally. The detachment flattened, the anterior chamber deepened, and the intraocular pressure returned to 16 ± 2 mm Hg after treatment with 80 mg of oral prednisolone and 400 mg of oxyphenbutazone daily.

normal angles that respond poorly to miotics[11] may be caused by an outflow facility impaired by collector channel inflammation.

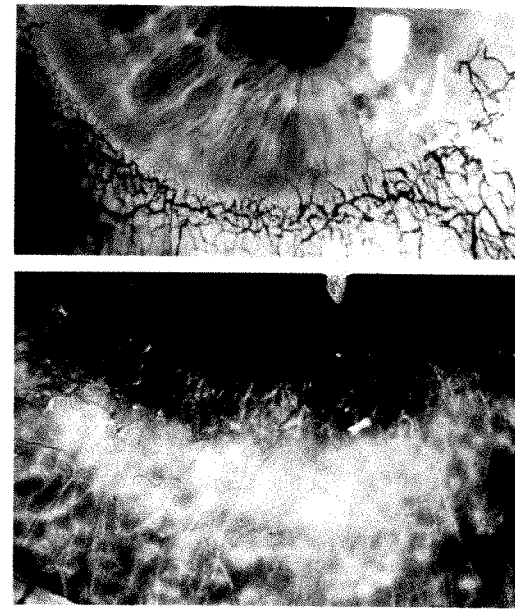


Fig. 8 (Wilhelmus, Grierson, and Watson). Case 4. A 55-year-old man with rheumatoid arthritis and anterior scleritis had intermittent episodes when ocular hypertension increased to 55 mm Hg. Top, Slit-lamp photograph of the right eye shows circumferential scleral inflammation of the corneoscleral limbus. Bottom, Scleral fluorescein angiography shows persistent staining of the perilimbal sclera.

Scleritis that is limited to the area posterior to the equator may be associated with open-angle, closed-angle, or neovascular glaucoma. An associated anterior uveitis with inflammatory debris in the angle was present in those eyes with open angles. Angle closure occurred either because of inflammatory angle synechiae or because of an annular choroidal detachment with anterior displacement of the iris-lens plane and anterior chamber shallowing. It is important to recognize this cause of acute closed-angle glaucoma, because treatment of the posterior scleritis with systemic anti-inflammatory medication may result in deepening of the anterior chamber. Angle neovascularization may also occur in association with posterior scleritis. Rubeosis iridis and angle neovascularization may have occurred by a direct extension of the posterior scleral

inflammation to the optic nerve and central retinal vessels. The clinical findings of optic disk edema and venous dilatation occasionally observed in patients with posterior scleritis may occur by a similar mechanism.[12,13]

Other causes of increased intraocular pressure with scleritis have been suggested. Because the serial sections needed to examine the complete vascular system were not available in this series, we could not substantiate the role of periphlebitis[14] or compression[15] of the vortex veins from an adjacent focus of scleritis in the development of glaucoma.

For a similar reason, staphyloma formation could not be studied on the selected histologic sections. In the clinical cases, however, large anterior staphylomas only occurred in those eyes in which the intraocular pressure was more than 35 mm Hg. Conversely, rupture of the globe was more likely to be caused by mechanical trauma to sclera already thinned and weakened by a necrogranulomatous reaction than by ocular hypertension.[16]

Of the histopathologic and clinical associations we noted between scleritis and increased intraocular pressure in this investigation, trabecular meshwork damage by inflammatory cells and tissue edema appeared to be the most important factor in the development of ocular hypertension. Scleral inflammation with secondary ocular hypertension that is not treated adequately with anti-inflammatory medication may result in the development of irreversible glaucomatous damage.

The finding of increased intraocular pressures in 45 (49%) of the enucleated eyes but in only 20 (18%) eyes of clinic patients suggested that uncontrolled inflammation is an important cause of ocular morbidity in the course of scleritis.

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PSEUDOMONAS AERUGINOSA SCLERITIS

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In two patients *Pseudomonas aeruginosa* scleral infection led to ocular perforation. In one patient, a scleral abscess was identified anteriorly. A scleral perforation occurred at a more posterior focus, but the eye was salvaged with minimal residual visual function. In the other patient, perforation at the corneoscleral limbus occurred after initial corneal improvement with antibiotic therapy; histopathologic examination of the enucleated globe disclosed an abscess extending from the corneoscleral limbus to the equator superiorly.

Although many species of *Pseudomonas* can cause eye infections, *P. aeruginosa* is the most common and most pathogenic of these organisms.[1] The frequently devastating ocular infection caused by this organism is related to its production of proteases, partially explaining the acute onset and rapid progression of the infection, its relative resistance to many commonly used antibiotics, and the tendency for the infection to recur after apparent remission, especially with the use of corticosteroids.[2-5] Corneal ulceration, with or without perforation, is the most frequent ocular infection caused by *P. aeruginosa*; endophthalmitis may occur after corneal perforation.[2]

Exogenous *Pseudomonas* scleritis occurs rarely and is usually caused by limbal-scleral extension of a corneal focus.[6,7] We treated two patients with *P. aeruginosa* scleral abscesses; in one patient, the abscess appeared to be isolated from the corneal zone of involvement.

CASE REPORTS

Case 1—A 91-year-old man, in good health except for medically controlled systemic hypertension, complained to his ophthalmologist of pain, photophobia, redness, and a moderate discharge in his left eye of ten days' duration. There was no history of trauma. His primary physician had treated him with a mixture of polymyxin B sulfate, neomycin sulfate, and hydrocortisone (Cortisporin Ophthalmic Solution) in the left eye four times daily. During the previous three days he had used chloramphenicol 0.5% drops four times daily.

An ocular examination disclosed a visual acuity of counting fingers in the left eye. The temporal bulbar conjunctiva was chemotic and dense white corneal stromal infiltrates were present between the 2 and 3 o'clock and the 4 and 6 o'clock positions with marked thickening of the adjacent sclera. The uninvolved cornea appeared clear. There was a moderate inflammatory reaction in the anterior chamber with the presence of posterior synechiae. Cultures were negative for bacteria and scrapings showed polymorphonuclear leukocytes and eosinophils. The patient was treated with 1 drop of gentamicin sulfate 0.3% and 1 drop of sodium sulfacetamide 10% in the left eye every hour, atropine sulfate 1% drops four times daily, and 500 mg of ampicillin, taken orally four times daily. Two days later he was referred here because of increasing scleral swelling and continued discomfort.

The patient was hospitalized. His visual acuity was R.E.: 6/22 (20/70) and L.E.: 6/240 (5/200). The bulbar conjunctiva was injected and chemotic and a firm, tender, elevated scleral nodule was present at the 5 o'clock position, 4 mm from the corneoscleral limbus (Fig. 1). A dense white peripheral stromal corneal infiltrate extended from the 10 to 6 o'clock positions

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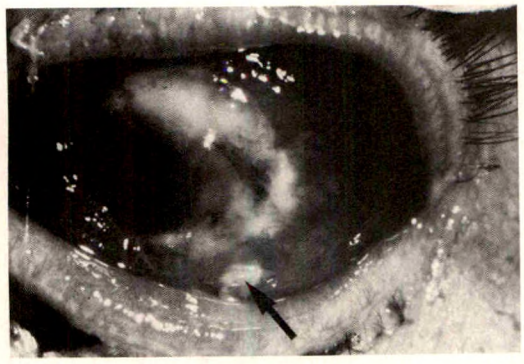


Fig. 1 (Codère, Brownstein, and Jackson). Case 1. Left eye shows a peripheral corneal infiltrate extending from the 10 to the 6 o'clock positions. An elevated scleral nodule (arrow) is present 4 mm from the corneoscleral limbus at the 5 o'clock position. It is not in direct communication with the peripheral corneal infiltrate.

with marginal ulceration from the 2 to 4 o'clock positions. There was no evidence of any communication between this peripheral corneal infiltrate and the elevated scleral nodule at the 5 o'clock position. The rest of the cornea showed mild stromal and epithelial edema, and a moderate iritis was present. Corneal and conjunctival scrapings displayed a few polymorphonuclear leukocytes and occasional gram-positive cocci in pairs. Conjunctival and corneal cultures disclosed a heavy growth of *P. aeruginosa* that was sensitive to gentamicin, colistin sulfate, and carbenicillin (using the antimicrobial disk sensitivity test with an agar overlay).

The patient was initially treated with 1 drop of atropine 1% and 1 drop of phenylephrine HCl 10% in the left eye twice daily, 1 drop each of prednisolone sodium phosphate 0.125%, gentamicin 0.3%, and acetylcysteine 20% in the left eye four times daily, and 500 mg of cloxacillin sodium, given intravenously every six hours. Once *P. aeruginosa* was cultured, the therapy was changed to 1 drop each of gentamicin sulfate 0.9%, carbenicillin 0.6%, and polymyxin B sulfate 0.2% in the left eye every hour, and 2 g of intravenous carbenicillin every four hours. Subconjunctival injections of 30 mg of gentamicin and 100 mg of carbenicillin were given on alternate days. Treatment with topical acetylcysteine, atropine, and phenylephrine HCl was continued.

Three days after the patient entered the hospital, the scleral nodule at the 5 o'clock position, which had become more elevated, was incised. Purulent material, from which *P. aeruginosa* was cultured, continued to drain from this abscess for two weeks. During the next week, the corneal stromal infiltrate spread to involve the entire temporal half of the cornea as well as the nasal limbal region. The polymyxin B sulfate was discontinued and the acetylcysteine 20% was increased to 1 drop every three hours; tobramycin sulfate 0.3%, 1 drop every hour, was added.

Eight days after the patient entered the hospital, we noted a small hyphema and iridodonesis. B-scan ultrasonography, which had given normal results at the time of admission, disclosed a total retinal detachment. The globe appeared to have undergone spontaneous perforation posteriorly. The cornea was thinned superiorly, almost to the point of perforation. After three weeks of therapy, the corneal infiltrate started to resolve and the thin peripheral superior peripheral groove began to vascularize. Cultures of the cornea, scleral abscess, and conjunctiva showed no growth of organisms on the culture media on the fourth day of hospitalization and only a light growth of *Staphylococcus epidermidis* during the third and fourth weeks after admission.

Five months later, the patient had no discomfort but his visual acuity was only light perception. The central cornea remained clear with a superior and temporal pannus and the area of the scleral abscess was pigmented. The anterior chamber was deep; rubeosis iridis and a dense cataract were evident. The intraocular pressures were R.E.: 12 mm Hg and L.E.: 18 mm Hg.

Case 2—A 77-year-old woman had experienced pain and redness in her right eye for several weeks. One week before she was hospitalized, she had noted that her vision was poor in that eye. She had been taking tolbutamide (250 mg) orally twice daily for long-standing adult-onset diabetes mellitus, methyldopa (250 mg, four times daily) for hypertension, and digoxin (0.125 mg daily), furosemide (40 mg daily), and potassium chloride (600 mg daily) for congestive heart failure. She also had neovascular glaucoma in her right eye. Her left leg had been amputated seven months previously because of gangrene. On the evening before she entered the hospital, she had received 1 drop of gentamicin sulfate 0.3% in the right eye every hour.

Her visual acuity was R.E.: no light perception and L.E.: 6/12 (20/40). The right eye had a purulent discharge from the chemotic and markedly injected conjunctiva under which two limbal-scleral nodules were present superiorly. A central corneal ulcer with a dense corneal infiltrate was noted and a hypopyon occupied 10% of the anterior chamber. A Giemsa-stained corneal scraping displayed numerous polymorphonuclear leukocytes and a Gram-stained corneal smear disclosed gram-negative rods; the culture revealed *P. aeruginosa* that was sensitive to gentamicin, carbenicillin, and colistin sulfate (using the antimicrobial disk sensitivity test with an agar overlay).

Treatment was started with 1 drop of gentamicin 1.4% hourly and bacitracin ointment (500 units/g) every two hours in the right eye. She received 2 g of intravenous carbenicillin every four hours for nine days; this was discontinued because of an allergic reaction. Subconjunctival injections of 20 mg of gentamicin and 100 mg of carbenicillin were given daily for eight days. After the eighth day conjunctival scarring made the injections impossible. The hypopyon and the central ulcer progressively disappeared with the treatment, but the limbal-scleral nodules

increased in size for five days and then remained unchanged.

On the seventh day of her hospitalization, corneal cultures showed a light growth of both *S. epidermidis* and *P. aeruginosa*; ten days later a culture from the right eye showed no growth. Three weeks after the patient entered the hospital, her right eye became indurated and a large staphyloma perforated superiorly; one hour later the globe was enucleated. A culture taken at the beginning of the operation disclosed a heavy growth of *P. aeruginosa*. The postoperative course was uneventful.

Pathologic findings—The right globe measured 33 × 21 × 25 mm; the opaque cornea measured 13 mm horizontally. Superiorly, an irregular corneoscleral limbal perforation measuring 14 × 9 mm contained the prolapsed hemorrhagic intraocular contents (Figs. 2 to 4).

Microscopic examination disclosed two superior confluent abscesses, one involving the full-thickness sclera near the equator and the other at the corneoscleral limbus at the site of the rupture (Figs. 4 and 5). Hemorrhagic intraocular tissues, including necrotic uvea, ruptured lens, vitreous, and retina, had prolapsed through the superior corneoscleral limbal wound. The cornea showed disorganization of the architecture of the thickened epithelium with bulla formation, a fibrous scar replacing most of Bowman's membrane, a moderate acute inflammatory cell infiltrate of the stroma, and absence of most of the endothelium. No definite microorganisms could be identified in sections stained for bacteria.

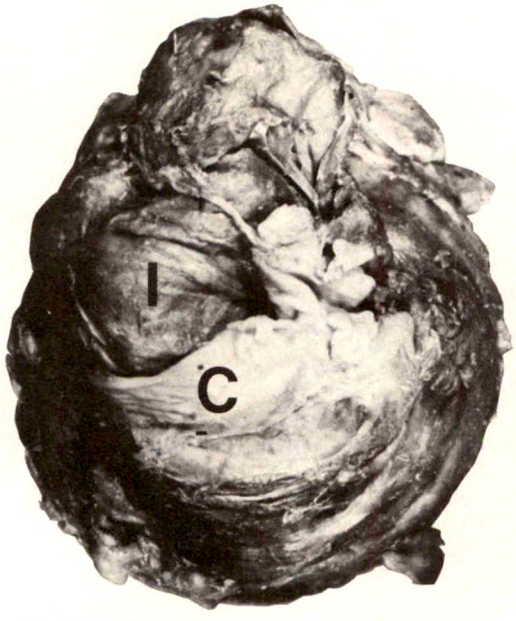


Fig. 2 (Codère, Brownstein, and Jackson). Case 2. Right globe displays a superior corneoscleral limbal rupture with the cornea (C) displaced inferiorly by the prolapsed hemorrhagic intraocular contents (I).

Discussion

Ocular infection with *P. aeruginosa* often occurs in a compromised host.[4] Our Patient 2 was a diabetic with neovascular glaucoma in her infected eye. Both of our patients were elderly (91 and 77 years old). Both showed a slow progression of the infective process in the cornea with extension toward the periphery and into the sclera instead of the rapid deepening of a central corneal ulcer, which is the usual site of perforation in external ocular *Pseudomonas* infections.[8]

The first case was unusual in that a scleral abscess developed in an area isolated from any adjacent corneal or corneoscleral limbal ulcer. This isolated scleral abscess, which to our knowledge has not been previously described with *P. aeruginosa* infections except after previous scleral trauma, may cause diagnostic difficulties.[9] Our Case 1, for example, was interpreted as nodular scleritis with sclerokeratitis. This lesion, therefore, should be included in the differential diagnosis of nodular scleritis. The use of topical antibiotic-corticosteroid drops during the initial week of infection may explain the unusual progression in this case. In our second patient, the scleral abscess represented an extension of the



Fig. 3 (Codère, Brownstein, and Jackson). Case 2. Right globe shows prolapsed intraocular contents at the superior limbal rupture. The thick white scleral nodule superiorly (arrow) corresponds to the confluent abscesses noted in Figures 4 and 5.

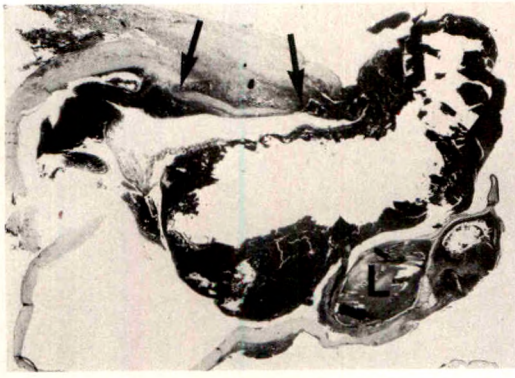


Fig. 4 (Codère, Brownstein, and Jackson). Case 2. Prolapsed hemorrhagic intraocular tissue at superior corneoscleral limbal rupture with lens (L) dislocated anteriorly and inferiorly. Confluent superior scleral abscesses (arrows) are evident at the corneoscleral limbus and near the equator (hematoxylin and eosin, ×3.5).

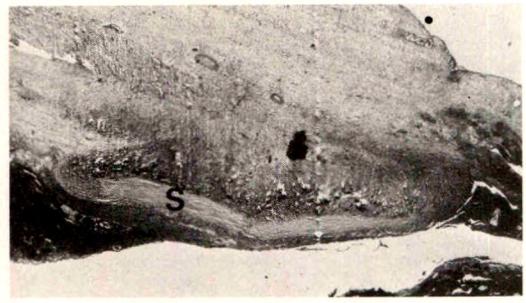


Fig. 5 (Codère, Brownstein, and Jackson). Case 2. Higher-power view of the two confluent full-thickness scleral abscesses shown in Figure 4. A sequestrum of sclera (S) is surrounded by the inflammatory reaction (hematoxylin and eosin, ×40).

corneal and corneoscleral limbal infection; the necrotic abscess extended to the equator, with a sequestrum of sclera, despite prolonged antimicrobial therapy.

Successful treatment of *P. aeruginosa* infections requires early diagnosis (by scrapings and cultures) and prolonged administration of antibiotics to which the organisms are sensitive, even after there is clinical improvement.[3,4,8] A partially controlled infection may recur or become exacerbated weeks after the initiation of treatment, even without corticosteroid therapy.[4,5,8] In our second case, the clinical findings and the negative bacterial culture reports suggested that the disease had been controlled. However, perforation of the globe during the third week of hospitalization and a culture positive for *P. aeruginosa* at the time of surgery indicated that organisms were present in the scleral stroma and that the previous negative culture was probably related to the antibiotics administered. Prolonged intensive antibiotic therapy may save the globe, as occurred in our first patient, even in the presence of a presumed posterior perforation, manifested in this case by iridodonesis, hyphema, and retinal detachment.[3,5] Penetrating keratoplasty offers some hope of salvaging the globe in cases unresponsive to medical therapy.[10] Raber and associates[11] emphasized that once *Pseudomonas* corneal infection extends into the sclera, medical treatment is not effective; four of their patients with these findings eventually required enucleation or evisceration. However, Eiferman[7] recently reported three cases of perforated *Pseudomonas* corneal ulcers with scleral extension, all of which showed dramatic improvement after keratoplasty and cryotherapy to the remaining cornea and corneoscleral limbus. Since *Pseudomonas* organisms appear to be susceptible to cryotherapy in vivo, we should consider this mode of therapy as an adjunct to medical and surgical treatment of *Pseudomonas* scleral abscesses.

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Fifty years ago this month in The Journal

The best method of securing anesthesia is also a slight modification of the usual practice. Cocaine four percent instilled four or five times and adrenalin once or twice will give ample anesthesia for incising and undermining the conjunctiva and exposing the muscle and tendon. If one is ready to begin and cocaine has been instilled twice only, time can be saved by applying a tiny wad of cotton, soaked in ten percent cocaine, just over the muscle and tendon and then closing the lids carefully over it for three minutes.

After the muscle has been exposed and before it has been taken up on the hook, novocaine two percent and adrenalin 1 to 10,000 are injected along the muscle back of the tarsoorbital fascia.

Walter B. Lancaster: Operation to shorten a rectus muscle with buried sutures. Am. J. Ophthalmol. 14:483, 1931

GENETIC BACKGROUND OF ACUTE ANTERIOR UVEITIS*

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To determine the hereditary pattern of acute anterior uveitis, we examined seven families—one family with four cases, one family with three cases, and four families with two cases of acute anterior uveitis. A total of 16 patients with uveitis and 28 relatives underwent ocular examinations and serologic determination of HLA antigens, protease inhibitor α_1-antitrypsin typing, and X-ray examination of sacroiliac joints.

Sacroiliitis was found in 11 of 16 patients (68.8%) and incomplete Reiter's disease in seven of 16 (43.8%). HLA-B27 antigen was found in all patients with uveitis and in six of 21 (28.6%) relatives. HLA-Cw1 antigen was found in nine of 14 (64.3%) patients with uveitis, often in the same haplotype with HLA-B27 antigen. HLA-DR4 antigen was found in seven of 11 (63.6%) patients with uveitis. There was no significant association of the α_1-antitrypsin variant MZ with uveitis.

We believe that a pleiotropic gene associated with HLA-B27 antigen with autosomal dominant inheritance, incomplete penetrance, and variable expressivity may determine susceptibility to acute anterior uveitis in linkage disequilibrium with HLA-Cw1 antigen.

Several investigators[1-3] have found a significant association between HLA-B27 antigen and acute anterior uveitis. The modes of inheritance for HLA-B27 and acute anterior uveitis have been described in 18 families.[4-11] In one of these families three cases of acute anterior uveitis occurred[10]; no more than two cases occurred in any other family.[4-9,11] We studied six other families with more than one patient, including one family with four cases and one family with three cases. In addition to the HLA-A, HLA-B, and HLA-C antigens determined in earlier studies, we also determined HLA-DR antigens in this study.

Brewerton and associates,[12] working in England, reported a strong association between the protease inhibitor α_1-antitrypsin phenotype MZ and acute an-

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terior uveitis, but Brown and associates[13] did not find this association in patients with acute anterior uveitis in North America. Because of these conflicting findings, we included an analysis of the association between protease inhibitor phenotype MZ and acute anterior uveitis in our study.

Subjects and methods

We asked 224 patients who had been treated here for acute anterior uveitis since 1964 to return for follow-up examinations. During the acute phase of their illness, all patients had had redness, photophobia, and pain; some had experienced reduced vision and increased lacrimation in the inflamed eye, with ciliary injection, cells, and flare in the aqueous humor, small keratic precipitates, and exudates in the aqueous humor and posterior synechiae.

Of the 224 patients, 141 (62.9%) returned and provided details about their relatives. Of the 137 unrelated patients, 64 (46.7%) reported that others in their families had rheumatic diseases. In 19 cases (13.9%) at least one other family member had had iritis. From these 19 families, we selected five that included 13 cases of acute anterior uveitis for the present study. We also included two families, one with two cases of acute anterior uveitis and one with one case of acute anterior uveitis and one case of recurrent episcleritis, that were not in the original series.

A total of 16 patients with confirmed acute anterior uveitis and 28 family members underwent routine ocular examinations. Using the two-stage microcytotoxicity method,[14] we determined HLA-A and HLA-B antigens in all 16 patients and in 21 relatives and HLA-C antigens in 14 patients and 21 relatives. We determined HLA-DR antigens in four families (11 patients and 19 relatives) with the Eighth International HLA Workshop serum panel. In 15 patients and 20 relatives, we typed the protease inhibitor α_1-antitrypsin with separator isoelectric focusing,[15] allowing reliable identification of protease inhibitor subtypes, including the Z variant.[16] We used 120 healthy volunteers from the same geographic area as controls for α_1-antitrypsin typing.

Sixteen patients with acute anterior uveitis and 19 family members underwent X-ray examination of the sacroiliac joints. Alterations leading to the radiologic diagnosis of sacroiliitis were the same as those used in earlier studies.[7] We used the New York clinical criteria for ankylosing spondylitis.[17]

Case reports

Family 1—The 74-year-old father (Fig. 1, I-1) had had severe acute polyarthritis when he was 20 years old, and ankylosing spondylitis ever since. He had had four episodes of acute anterior uveitis between the ages of 31 and 50 years. His 70-year-old wife (Fig. 1, I-2) had had osteoarthritis in the hip, knee, and ankle joints since the age of 55 years. She had no ocular symptoms.

The eldest child (Fig. 1, II-1), a 38-year-old woman, had had relapsing polyarthritis since the age of 15 years, ankylosing spondylitis since the age of 21 years, and frequent episodes of acute anterior uveitis in the left eye with development of posterior synechiae since the age of 24 years. She had undergone peripheral iridectomy when she was 30 years old because of seclusion of the pupil of the left eye. Since then, she had had five episodes of acute anterior uveitis in the right eye, and frequent urinary tract infections. Her visual acuity was 6/6 (20/20) in the right eye. There were old pigmented keratic precipitates, and permanent posterior synechiae at the 6 o'clock position. Her visual acuity was 6/9 (20/30) in the left eye. We found old richly pigmented keratic precipitates, seclusion and partial occlusion of the pupil, anterior peripheral synechiae, severe atrophy of the iris stroma, a peripheral iridectomy hole at the 12 o'clock position, and shallow cupping of the temporal part of the optic disk. Intraocular pressure was R.E.: 12 mm Hg and L.E.: 14 mm Hg.

The proband (Fig. 1, II-2), a 37-year-old woman had had acute gastroenteritis with diarrhea, fever, nausea, and myalgia at the age of 26 years and symptoms of ankylosing spondylitis ever since. At the age of 34 years, she had had acute anterior uveitis with fibrinous exudation, posterior synechiae, and vitreous opacities in her right eye. The inflammatory signs had resolved completely in six weeks with cycloplegic and systemic and topical corticosteroid treatment. Seven months later she had had acute anterior uveitis, with inflammatory signs, in her left

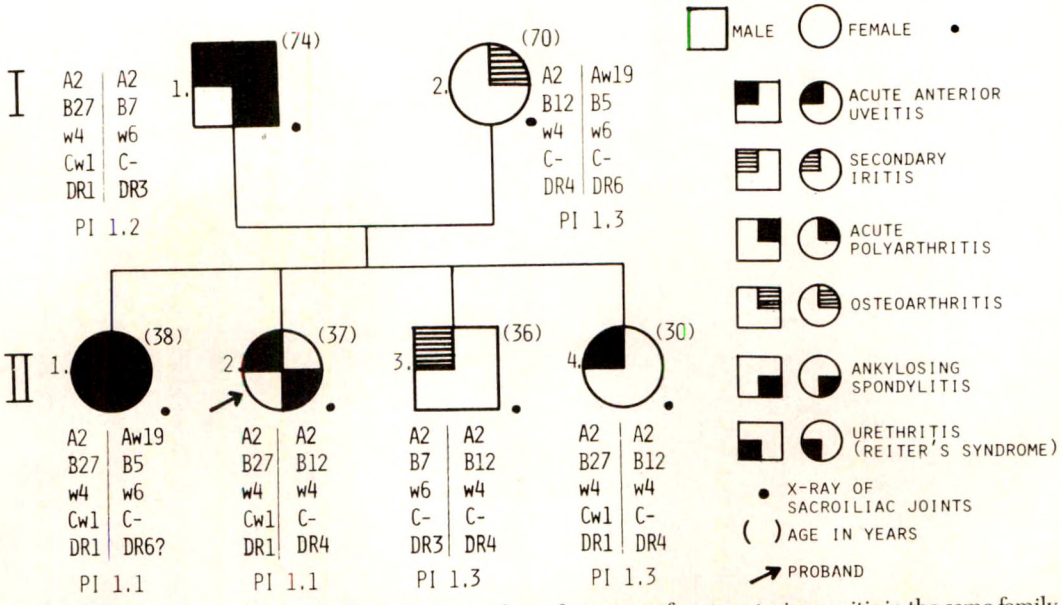


Fig. 1 (Saari and associates). Family 1. Pedigree shows four cases of acute anterior uveitis in the same family, occurrence of rheumatic diseases, and segregation of HLA antigens and protease inhibitor α_1-antitrypsin Pi M subtypes.

eye. This had subsided in four weeks with topical corticosteroid and cycloplegic treatment. Visual acuity was 6/6 (20/20) in both eyes.

Her 36-year-old brother (Fig. 1, II-3) had had slight secondary iritis for a few days in the right eye caused by a corneal foreign body that had remained in situ for three days. He also had occasional slight conjunctival irritation.

The youngest child, a 30-year-old woman (Fig. 1, II-4), had had acute anterior uveitis in the right eye at the age of 24 years and two episodes of recurrent iritis in the same eye one year and three years later. During the acute attacks, she had had a heavy ciliary injection, aqueous flare and cells, small keratic precipitates, posterior synechiae, vitritis, and slight macular edema. These inflammatory signs had subsided in five weeks with topical corticosteroid and cycloplegic treatment. Visual acuity was 6/5 (20/16) in both eyes.

Family 2—The 73-year-old grandmother (Fig. 2, I-2) had had episodes of iritis for years. At the age of 69 years, she had had acute anterior uveitis in both eyes for five weeks. Her visual acuity was R.E.: 6/15 (20/50) −1.75 and L.E.: 6/6 (20/20) −2.0. There was a posterior cortical cataract in the right eye, and pseudoexfoliation of the lens capsule in the left eye. Both eyes showed persistent posterior synechiae and medullated nerve fibers in the retina above the optic disk. X-ray films showed sacroiliitis.

A 42-year-old mother (Fig. 2, II-5) of the proband had had ankylosing spondylitis and frequent episodes of urethritis since the age of 29 years. At the age of 34 years, she had had hypertrophic cervicitis, urethritis, and acute salpingitis with headache, nausea, vomiting, abdominal pains, and temperatures as high as 40C followed by acute polyarthritis. Seven months later she had had acute anterior uveitis in the left eye that lasted for five weeks. One year later she had had iritis in the left eye. Two years later she again had had iritis in the left eye. On this occasion the vitritis and macular edema had responded to cycloplegic and systemic and topical corticosteroid treatment in five weeks. Visual acuity was 6/5 (20/16) in both eyes. The left eye showed remnants of the pigmented layer of the iris on the anterior surface of the lens; otherwise the eyes were normal.

Her 38-year-old sister (Fig. 2, II-6) had had occasional episodes of bilateral conjunctivitis with photophobia, pain, redness, and increased lacrimation since the age of 34 years.

The proband, a 19-year-old man (Fig. 2, III-2), had had acute anterior uveitis in the right eye for four weeks at the age of 15 years. Two years later he had had arthritis in the left knee and ankle joints. He had occasional episodes of conjunctivitis with redness and a smarting sensation. Visual acuity was 6/5 (20/16) in both eyes. The right eye showed pigment remnants on the anterior surface of the lens. X-ray films showed sacroiliitis.

Family 3—The 55-year-old proband (Fig. 3, II-10) had had acute anterior uveitis in the right eye with severe ciliary injection, aqueous flare and cells, hypopyon, and posterior synechiae when he was 49 years old. The inflammatory signs had subsided in five weeks with topical cycloplegic and corticosteroid treatment. Four years later he had had recurrent exudative iritis that lasted for four weeks in the same eye. Visual acuity was 6/5 (20/16) in both eyes.

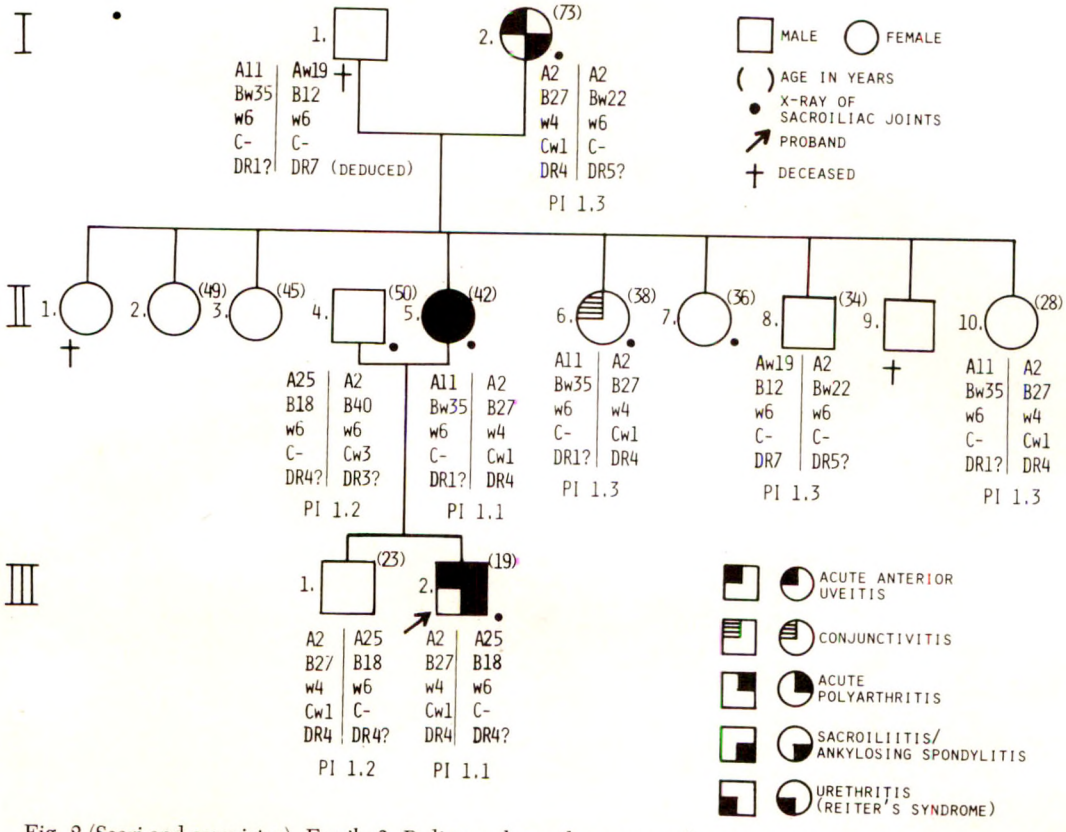


Fig. 2 (Saari and associates). Family 2. Pedigree shows three cases of acute anterior uveitis with rheumatic diseases in the same family, HLA antigen haplotypes, and segregation of protease inhibitor α_1-antitrypsin Pi M subtypes.

The 53-year-old brother (Fig. 3, II-11) of the proband had had ankylosing spondylitis since the age of 20 years. He had had acute anterior uveitis in the left eye at the age of 46 years and four episodes of acute anterior uveitis with posterior synechiae and macular edema in the right eye during the next six years. His visual acuity was 6/6 (20/20) in both eyes. There were permanent posterior synechiae in the inferior part of the pupil in the right eye and pigment remnants on the anterior surface of the lens in both eyes.

The 64-year-old brother (Fig. 3, II-4) of the proband had bronchial asthma, allergic conjunctivitis, chronic allergic rhinitis, and osteoarthritis.

The 59-year-old brother (Fig. 3, II-8) had had rheumatic fever with reactive arthritis and erythema nodosum at the age of 42 years. His youngest child (Fig. 3, III-3) had atopic eczema.

The 57-year-old brother (Fig. 3, II-9) of the proband had allergic conjunctivitis, and the youngest brother (Fig. 3, II-12), who had chronic allergic rhinitis, had had carditis and reactive arthritis after rheumatic fever.

Family 4—The mother (Fig. 4, I-1) had had urethritis and relapsing polyarthritis but no ocular inflammation. Her oldest child (Fig. 4, II-1) had had paresis of the left facial and abducens nerves at the age of 34 years and balanitis four years later. He had lower back pain and X-ray films showed unilateral grade 2 sacroiliitis.

The proband (Fig. 4, II-2), a 39-year-old man, had had acute anterior uveitis in the right eye at the age of 27 years. Two years later he had had mild conjunctivitis for a few days in both eyes. At the age of 38 years, he had had acute anterior uveitis with severe pericorneal injection, aqueous flare and cells, small keratic precipitates, posterior synechiae, vitritis, and papilledema in his left eye. The inflammatory signs had subsided in six weeks with cycloplegic and systemic and topical corticosteroid treatment.

His 32-year-old sister (Fig. 4, II-3) had had acute anterior uveitis in the left eye at the age of 22 years. Her visual acuity was 6/5 (20/16) in both eyes. The left eye showed pigment remnants on the anterior surface of the lens.

Family 5—The proband (Fig. 5, top, I-1) had had ankylosing spondylitis since the age of 23 years. He had urethritis, prostatitis, and an erythrocyte sedimentation rate as high as 70 mm/hr (Westergren). He

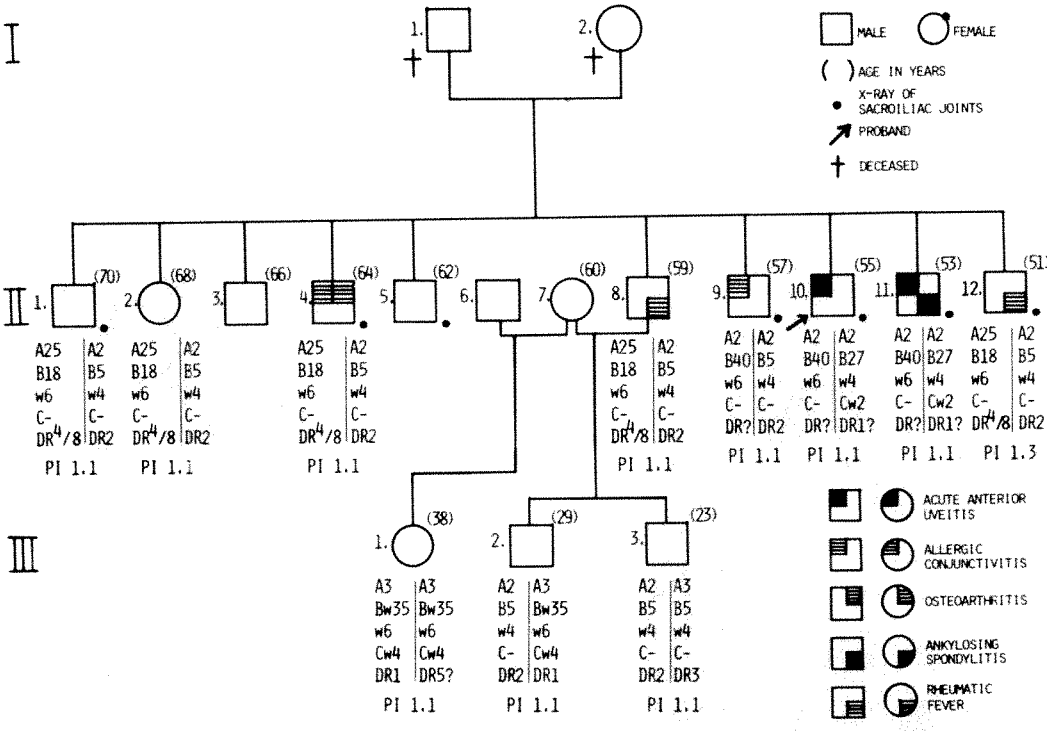


Fig. 3 (Saari and associates). Family 3. Pedigree shows two cases of acute anterior uveitis and different allergic and rheumatic diseases in the same family, HLA antigen haplotypes, and segregation of protease inhibitor α_1-antitrypsin Pi M subtypes.

had had acute anterior uveitis in the right eye when he was 30 and 35 years old and in the left eye when he was 47 and 50 years old. His oldest son (Fig. 5, top, II-1) had had seronegative, asymmetric polyarthritis at the age of 19 years.

Three siblings (Fig. 5, top, II-2, II-3, and II-4) had lower back pain, and X-ray films showed sacroiliitis. One of them (Fig. 5, top, II-3) had had acute anterior uveitis in the left eye when he was 13, 14, and 19 years old, and circinate erosive balanitis at the age of 17 years.

Family 6—The 45-year-old proband (Fig. 5, bottom, III-8) had had ankylosing spondylitis since the age of 33 years. He had had acute anterior uveitis in

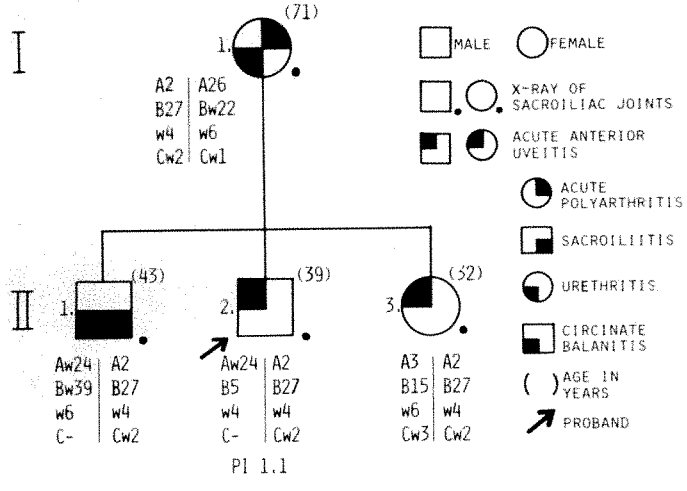


Fig. 4 (Saari and associates). Family 4. Pedigree shows HLA antigen haplotypes and two cases of acute anterior uveitis and rheumatic diseases in the same family.

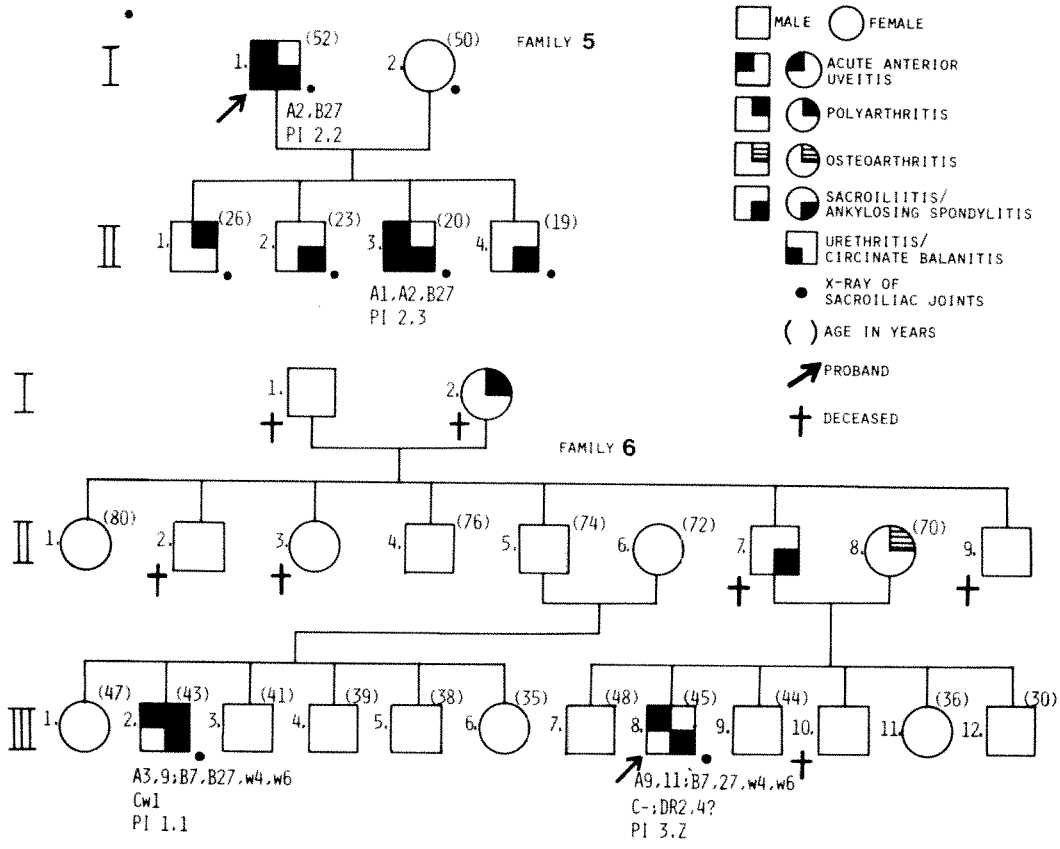


Fig. 5 (Saari and associates). HLA antigen phenotypes and protease inhibitor α_1-antitrypsin Pi M subtypes in Families 5 (at top) and 6 (at bottom), including two cases of acute anterior uveitis in the same family.

the left eye at the ages of 35 and 38 years and in the right eye at the ages of 43 and 44 years. His grandmother (Fig. 5, bottom, I-2) had been bedridden for 20 years because of chronic arthritis, his father (Fig. 5, bottom, II-7) had ankylosing spondylitis, his mother (Fig. 5, bottom, II-8) had osteoarthritis, and two siblings (Fig. 5, bottom, III-7 and III-11) complained of lower back pain.

The 43-year-old cousin of the proband (Fig. 5, bottom, III-2) had had acute anterior uveitis with fibrinous exudates in the aqueous humor and posterior synechiae in the right eye at the age of 38 years. One year later he had had acute gastroenteritis with diarrhea and melena that lasted for one month, reactive polyarthritis, aching and stiffness of the back, and an erythrocyte sedimentation rate as high as 83 mm/hr (Westergren), followed three months later by acute anterior uveitis in the left eye. One year later he had had acute anterior uveitis in the right eye, and X-ray films confirmed that he had ankylosing spondylitis.

Family 7—The proband (Fig. 6, II-2) had had bilateral acute anterior uveitis for five weeks at the age of 28 years. Intraocular pressure had been R.E.: 10 mm Hg and L.E.: 8 mm Hg after the onset of the iritis but four weeks later, during treatment with topical corticosteroids, it had increased to R.E.: 39 mm Hg and L.E.: 41 mm Hg. Treatment with acetazolamide, 500 mg orally three times a day, had decreased the intraocular pressure to normal, but she had to stop taking the drug because of side effects. Two weeks later, the intraocular pressure had increased to R.E.: 38 mm Hg and L.E.: 34 mm Hg. One day of treatment with timolol maleate 0.25% eyedrops, used twice daily, had decreased the intraocular pressure to normal.

Her 60-year-old father (Fig. 6, I-1) had had seven episodes of recurrent episcleritis since the age of 43 years.

RESULTS

We found HLA-B27 in all 16 patients (seven probands) with acute anterior uveitis (100%) and in six (28.6%) of 21 tested relatives, whereas it is found in only 14% of healthy controls.[18] The increased

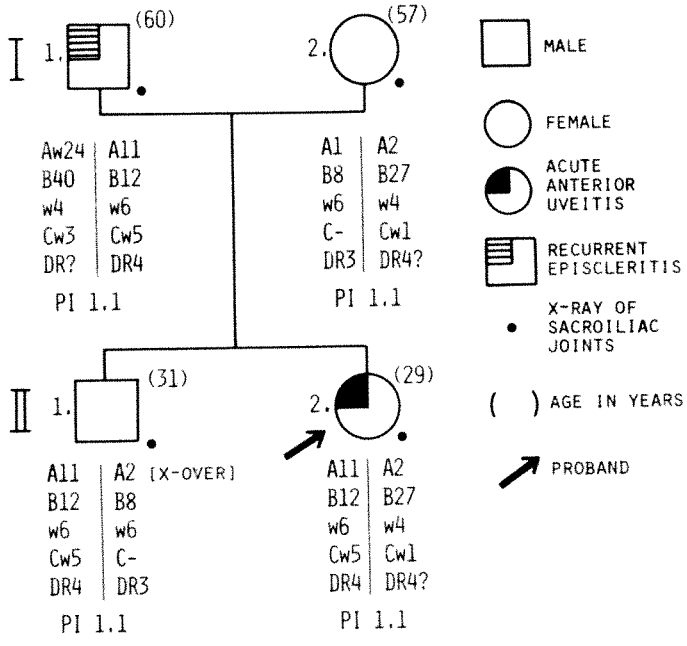


Fig. 6 (Saari and associates). Family 7. Pedigree shows HLA antigen haplotypes, segregation of protease inhibitor α_1-antitrypsin Pi M subtypes, one case of acute anterior uveitis, and one case of recurrent episcleritis.

frequency of HLA-B27 antigen in the probands with acute anterior uveitis compared with the controls was highly significant ($P < .0001$). Of the 14 patients with acute anterior uveitis tested for HLA-C locus antigens, nine (64.3%) had the antigen HLA-Cw1. We found HLA-Cw1 antigen in three of six (50%) tested probands and in five of 21 (23.8%) tested relatives, but it is found in only 14% of normal controls.[18] The increased frequency of HLA-Cw1 antigen in the probands compared with our controls was not significant ($\chi^2 = 3.512$; $P < .1$). Both HLA-Cw1 and HLA-B27 antigens were found in eight of 12 patients (66.7%) and in four of six (66.7%) relatives positive for HLA-B27 antigen whose haplotypes could be derived.

In the 11 patients with acute anterior uveitis who were tested for HLA-DR locus antigens, HLA-DR1, HLA-DR2, and HLA-DR4 antigens were found seven (63.6%), one (9.1%), and seven (63.6%) times, respectively. We found HLA-DR1 antigen in two (40%) and HLA-DR4 antigen in four (80%) of the five probands tested for HLA-DR locus antigens. We found HLA-DR1 antigen in 25 (30.1%) and HLA-DR4 antigen in 18 (21.7%) of our 83 normal controls. The increased frequency of HLA-DR4 antigen in the probands compared with the controls was almost significant ($\chi^2 = 5.725$; $P < .025$); however, the corrected P value[19] showed that the increased frequency of HLA-DR4 antigen in the probands was not significant. Of the ten patients tested for HLA-DR antigen whose haplotypes could be derived, six (60%) had HLA-DR1 antigen and four (40%) had HLA-DR4 in the same haplotype as HLA-B27 antigen.

We found protease inhibitor α_1-antitrypsin variant Z in one of 15 (6.7%) tested patients (Fig. 6, bottom, III-8), in none of 20 tested relatives without uveitis, and in five of 120 (4.2%) control subjects. The difference between MZ frequencies in the iritis patients and in the controls was not significant ($\chi^2 = 0.049$; $P < .9$).

Sacroiliitis and ankylosing spondylitis occurred in 11 of the 16 (68.8%) patients with acute anterior uveitis, and in three

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of 19 (15.8%) family members studied with X-rays.

Discussion

In this study, 19 of the 137 (13.9%) unrelated patients with acute anterior uveitis had relatives with the same disease. This is the first study to report four cases of acute anterior uveitis in the same family (Fig. 1). We described also one family with three cases (Fig. 2) and four families with two cases of acute anterior uveitis. These findings indicated that acute anterior uveitis can be familial.[4-11]

In none of our six families with more than one case did the acute anterior uveitis remain monosymptomatic. Ankylosing spondylitis was observed in 69% of patients with iritis and in 16% of their relatives who were studied by X-rays. Seven patients with acute anterior uveitis (43.8%) and four (14.3%) of 28 relatives had manifestations of complete or incomplete Reiter's disease.

These results suggested that a pleiotropic gene determines the susceptibility to acute anterior uveitis, ankylosing spondylitis, reactive arthritis, and Reiter's disease; in our series the 100% association with HLA-B27 antigen suggested a close linkage between the two loci. In Family 1 (Fig. 1), three of four patients with acute anterior uveitis had ankylosing spondylitis and two also had manifestations of Reiter's disease; all of them had HLA-B27 antigen. In Family 2 (Fig. 2), all three uveitis patients had ankylosing spondylitis and two of them also had complete or incomplete Reiter's disease; all were positive for HLA-B27 antigen. This unusual family with acute anterior uveitis in three successive generations in both sexes demonstrated almost conclusively the autosomal dominant inheritance of susceptibility to acute anterior uveitis. Of the 11 HLA-typed members of Family 3, only two (Fig. 3, II-10 and II-11) were positive for HLA-B27 antigen. Both of them had acute anterior uveitis and one also had ankylosing spondylitis. All the members of Family 4 (Fig. 4) had HLA-B27 antigen and rheumatic diseases. In Family 5 (Fig. 5, top), the father had acute anterior uveitis, ankylosing spondylitis, incomplete Reiter's disease, and HLA-B27 antigen, and all his children had rheumatic diseases.

Every patient in this study with acute anterior uveitis and every tested relative with sacroiliitis and ankylosing spondylitis or Reiter's disease had HLA-B27 antigen.

In patients positive for HLA-B27 antigen, conjunctivitis may occur after *Yersinia*,[11] *Salmonella*,[20] and *Campylobacter*[21] infections as a symptom of Reiter's disease or as an independent reactive disorder. In this study, three patients positive for HLA-B27 antigen had conjunctivitis; one of them (Fig. 2, III-2) also had acute anterior uveitis, arthritis, and ankylosing spondylitis, one (Fig. 4, II-2) had intermittent conjunctivitis with acute anterior uveitis, and one (Fig. 2, II-6) had conjunctivitis as a monosymptomatic disease. These findings and our earlier results[11,20] suggested that the iritis-arthritis susceptibility gene may possess variable expressivity, appearing in the eyes as reactive conjunctivitis or acute anterior uveitis, in the joints as reactive arthralgia or arthritis, and in the lumbosacral spine as sacroiliitis or ankylosing spondylitis. It was obvious, however, that allergic conjunctivitis (Fig. 3, II-4 and II-9), secondary iritis (Fig. 1, II-3), episcleritis (Fig. 6, I-1),[9,22] infective conjunctivitis, arthritis associated with rheumatic fever (Fig. 3, II-8 and II-12),[23] and osteoarthritis (Fig. 1, I-2, and Fig. 3, II-4) were not associated with the HLA-B27 antigen except coincidentally.

In Family 7, the proband (Fig. 6, II-2) had a common sporadic case of acute anterior uveitis. She had inherited the HLA-B27-bearing risk haplotype from her healthy mother who represented

HLA-B27-positive skipped generation. Obviously the mother was not exposed to the same environmental infectious factors that seem to operate in concert with HLA-B27 antigen to produce uveitis.[11] The brother of the proband (Fig. 6, II-1) showed the only crossover in this study. He had inherited from his mother only the HLA-A locus of the HLA-B27-bearing risk haplotype and he was healthy.

In this study and in our earlier study,[7] we found that HLA-Cw1 antigen often appeared in the same haplotype as HLA-B27 antigen. This seems to occur in the normal Finnish population. These results suggested that in Finns the pleiotropic gene associated with HLA-B27 antigen that controls susceptibility to acute anterior uveitis is in linkage disequilibrium with HLA-Cw1 antigen.

This was the first study to show the inheritance of acute anterior uveitis and HLA-DR antigens. The results showed an almost significant association of acute anterior uveitis with HLA-DR4 antigen, but in probands the corrected P value was not significant. The course, severity, and number of recurrences of iritis did not differ between patients with HLA-DR4 antigen and those without HLA-DR4 antigen. However, the frequency of HLA-DR4 antigen was not increased in patients with ankylosing spondylitis.[24,25] More extensive studies are needed to decide if there is any association between acute anterior uveitis and HLA-DR4 antigen. Curiously, one patient with recurrent scleritis (Fig. 6, I-1) had HLA-DR4 antigen, the frequency of which increases in patients with rheumatoid arthritis.[26]

This study supported the finding of Brown and associates[13] that the reported association of acute anterior uveitis and heterozygous α_1-antitrypsin deficiency[12] is not universal. The results of this study show that α_1-antitrypsin phenotype MZ is not in close linkage with the iritis susceptibility gene in Finns.

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OPHTHALMIC MINIATURE

The number of references cited in the literature list (be always scrupulously careful to observe the house style) should be that which is sufficient and necessary; it may be a symptom of scientmanship (see Chapter 6) to quote references from journals published so long ago that librarians desperate for space have long since had them stashed away in the galleries of disused mines. Due homage and justice to one's predecessors are criteria to keep in mind, although some names are so great and some ideas so familiar that omission is homage greater than citation. Nice judgment is needed, though; one man's compliment may be another's source of grievance.

P. B. Medawar, *Advice to a Young Scientist*
New York, Harper and Row, 1979

TRANSPLANTATION OF LACERATED CORNEAS

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We performed penetrating keratoplasties in eight eyes with opaque corneas from healed corneal lacerations that extended into the corneoscleral limbus. We found dense fibrovascular membranes that extended from the laceration site over the surface of the iris into the angle in several cases and posteriorly to involve the ciliary body in all cases. In seven eyes moderate to severe increases of intraocular pressure occurred postoperatively. Additionally, five eyes had retinal detachments which were either present at the time of surgery or which developed postoperatively. The corneal grafts remained transparent in all eight eyes and visual acuity improved in four.

The visual prognosis for eyes with corneal lacerations has dramatically improved within the last several years.[1-9] This is probably related to improvements in microsurgical instruments and suture materials and especially to the widespread use of the operating microscope.[5-9] Nevertheless, in many eyes satisfactory vision is not obtained after repair of the laceration because of severe central corneal scarring and irregular astigmatism. Other sight-reducing complications in such patients include anterior segment fibrosis, cataract, and retinal detachment.[10] We studied the complications and results of penetrating keratoplasty in eyes with healed corneal lacerations that extended through the corneoscleral limbus.

MATERIAL AND METHODS

Eight patients, ranging in age from 5 to 43 years, with healed corneal lacerations that extended into the corneoscleral limbus underwent penetrating keratoplasty between 1974 and 1979. In seven eyes, the laceration extended 2 to 3 mm beyond the corneoscleral limbus into the sclera. Three of the eyes had previously undergone cataract extraction; in one the lens had been expulsed at the time of the trauma. Preoperative intraocular pressures were within normal limits in six eyes, and were less than 5 mm Hg in the other two. Adequate visualization of the fundus was impossible in any of these eyes. B-scan ultrasonography disclosed an inferior retinal detachment in one eye and was equivocal for a retinal detachment in another.

Corneal transplantation technique—The host cornea was trephined to a depth of two thirds of the thickness of the cornea. The anterior chamber was entered at a point away from the laceration where anterior chamber elements could be seen. In all eyes dense fibrovascular connective tissue extended from the laceration, often in front of the iris, into the chamber angle and always behind the iris or iris remnants to the ciliary body. The surgeon dissected the iris remnants away from the retrocorneal connective tissue which he excised as far behind the iris as he could see. He was careful to avoid traction on this membrane during its ex-

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cision. He performed anterior vitrectomy when it was necessary, and sutured the corneal donor tissue in place with eight interrupted 9-0 nylon sutures followed by a continuous 10-0 polypropylene suture. The polypropylene suture was pulled up and tied; he then removed the 9-0 sutures and injected a balanced saline solution into the anterior chamber. In five of eight corneas, the healed laceration resulted in obvious flattening of the corneal curvature. In these eyes a donor button 8 mm in diameter was placed into a 7.5-mm bed.

Preoperative and postoperative intraocular pressures were measured with a pneumotonometer.

We have selected one representative case for a detailed discussion.

Case Report

Case 1—A 26-year-old man suffered a penetrating laceration of his left cornea that involved the corneoscleral limbus and extended about 2 mm into the sclera (Table). He was referred to us approximately four years after the injury for evaluation of a dense corneal leukoma. The lens was extracted during the repair of the laceration.

The initial examination disclosed a visual acuity of light perception with projection in the involved eye. A dense corneal leukoma and associated retrocorneal membrane was present (Fig. 1). The anterior chamber was shallow and the intraocular pressure was 16 mm Hg. B-scan ultrasonography showed no evidence of any posterior segment abnormality.

A penetrating keratoplasty was performed with excision of a dense retrocorneal membrane that involved the entire surface of the iris and extended into the posterior chamber (Figs. 2 and 3). Shortly after the surgery, the intraocular pressure increased to more than 50 mm Hg. This was treated with cyclocryotherapy. Since then, medical therapy has maintained the intraocular pressure at less than 27 mm Hg.

The retina has remained attached, and the graft has remained clear for three years with the exception of a small area of opacification secondary to a rejection in the first year. The best corrected visual acuity is 6/15 (20/50) with a soft contact lens.

Results

Although the intraocular pressures were usually in the normal range or below normal during the first few postopera-

TABLE

Clinical summary of eight patients

Case, Sex, Age (yr)	Surgical Procedure	Intraocular Pressure		Postoperative Retinal Detachment	Graft Status
		Preoperative	Postoperative		
1, M, 26	Laceration repaired; cataract extracted	Normal	Controlled with cyclocryotherapy	No	Transparent centrally; 3 yr after surgery, visual acuity was 6/15 (20/50)
2, F, 42	Laceration repaired; cataract extracted	Normal	Controlled medically	Yes	Transparent
3, F, 12	Laceration repaired; cataract extracted	Normal	Controlled medically	Yes (reattached)	Transparent; 3 yr after surgery, visual acuity was 6/30 (20/100)
4 F, 8	Laceration repaired; cataract extracted	Normal	< 5 mm Hg	Yes	Transparent
5, M, 4	Laceration repaired	< 5 mm Hg	Controlled medically	Yes	Transparent
6, M, 15	Laceration repaired	Normal	Controlled with cyclocryotherapy	No	Transparent; 5 yr after surgery, visual acuity was 6/60 (20/200)
7, M, 42	Laceration repaired	Normal	Controlled medically	No	Transparent; 20 mo after surgery, visual acuity was 6/12 (20/40)
8, F, 4	Laceration repaired; lens expulsed at time of trauma	< 5 mm Hg	Controlled medically	Yes	Transparent

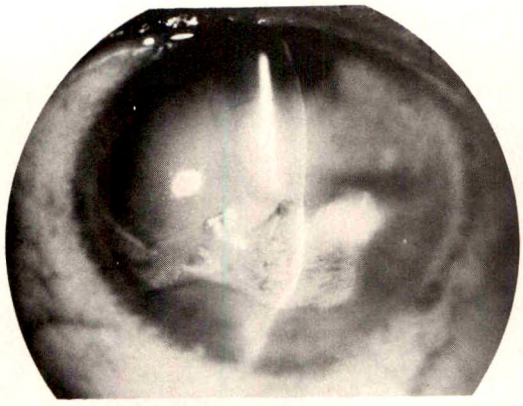


Fig. 1 (Sharkey and Brown). Case 1. Preoperative appearance of healed lacerated cornea that involved the corneoscleral limbus at the 8 o'clock position.

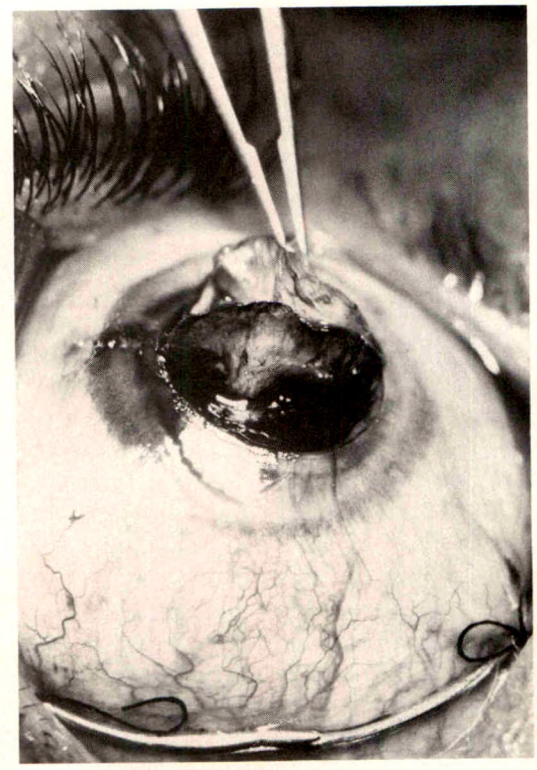


Fig. 2 (Sharkey and Brown). Case 1. Host cornea partially excised and held in forceps. A thick connective tissue membrane was dissected away from the surface of the iris. Its extension through the pupil is visible.

tive days, seven of the eight eyes developed pressures higher than 35 mm Hg by the third postoperative week. In five eyes the intraocular pressures were controlled with medical therapy (Cases 2, 3, 5, 7, and 8), but in the remaining two (Cases 1 and 6) cyclocryotherapy was necessary.

The one eye that did not develop increased intraocular pressure (Case 4) had had a preoperative traction detachment of the inferior retina that progressed to a total detachment. In four eyes (Cases 2, 3, 5, and 8) retinal detachment was present preoperatively or developed postoperatively. In two of these eyes (Cases 2 and 3) the diagnosis of retinal detachment was made at the operating table; in the other two (Cases 5 and 8) retinal detachment was first observed postoperatively. In these latter four eyes, we observed transvitreal traction bands, extensive epiretinal and subretinal fibrosis, or both. Retinal reattachment surgery was successful in one of two eyes (Case 3). Surgery was not considered in one eye (Case 8) because of advanced optic atrophy. A fourth eye (Case 5) may undergo retinal surgery in the near future. In Case 4, in which B-scan ultrasonography had disclosed a retinal detachment preoperatively, the detachment was judged to be inoperable because the retina was folded and incarcerated in the scleral laceration. In three of the eight eyes (Cases 1, 6, and 7), the retina remained attached throughout the follow-up period.

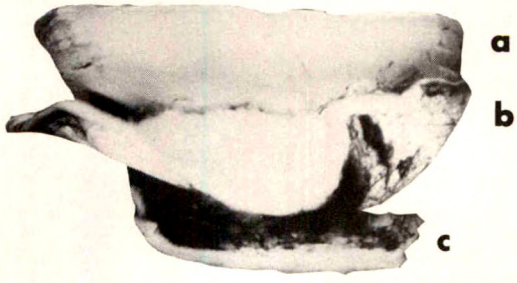


Fig. 3 (Sharkey and Brown). Case 1. Formaldehyde-fixed excised cornea (a) and connective tissue membrane (b) that covered the anterior iris and extended posterior to the iris to the ciliary body (c).

Discussion

All eight eyes showed moderate to marked obliteration of the anterior chamber. This was the result of adherence of the iris to the corneal scar and envelopment of the iris by a fibrous ingrowth that extended anterior to the iris into the angle in most eyes and behind the iris to the ciliary body in all eyes. This ingrowth was so thick and tough that in some eyes a straight suture scissors was necessary to excise it. Although all of the eyes had normal to low intraocular pressure preoperatively, seven eyes developed markedly increased intraocular pressure between the first and fourth postoperative weeks. This is unusual, since it has been our experience that increased intraocular pressure after corneal transplantation is usually associated with preoperative glaucoma (S. I. Brown, unpublished data). We assumed that the intraocular pressure increased postoperatively because a large portion of the chamber angles were occluded preoperatively by fibrous ingrowth and synechiae, although the intraocular pressure was controlled by a ciliochoroidal detachment caused by contraction of the cyclitic portion of the ingrowth. Postoperatively, however, when the ingrowth was excised, the traction-induced ciliochoroidal detachment was relieved, and the effect of the impaired outflow became apparent.

Five of the eight eyes had retinal detachments, only one of which was diagnosed preoperatively by ultrasonography. In this patient (Case 4), corneal surgery was performed so that the retinal detachment could be seen and repaired. In the other four eyes, the postoperative examination disclosed extensive retinal traction associated with retinal detachment. The presence of these well-established fibrotic bands so soon after surgery indicated that traction, with or without detachment, had been present preoperatively. In two instances the traction band originated in the scleral extension of the laceration. In both eyes the retinal traction apparently increased after surgery, leading to overt detachment in one (Case 5) and to a larger detachment in the other (Case 2).

In eyes with lacerations of the cornea that involve the corneoscleral limbus and 1 to 3 mm of sclera, with associated trauma to the lens and ciliary body, corneal transplantation is complicated by a thick fibrous ingrowth that incorporates the angle, iris, lens remnants, and ciliary body. Because of the membrane's firmness and bulk, it is difficult to avoid some pull during its excision; this pull may be transmitted to the retina via the traction bands that are often present. Furthermore, surgical disturbance of the anterior vitreous, as well as postoperative contraction of the remaining unexcised ingrowth, may also lead to retinal traction. This series suggests that anterior lacerations of the type described have retinal complications similar to those described in posterior lacerations.[11-13]

Marked late increases in the intraocular pressure were rather consistent despite the use of donor buttons larger than the host bed. We used oversized donor buttons to overcome the corneal flattening caused by the lacerations.

We do not know the details of the initial examination or the original reparative surgery of these patients. We believe, as do others, that avoiding incarceration of the iris and vitreous and removing the lacerated lens probably decrease the extent and incidence of fibrous ingrowth. However, a study of vitrectomy incisions indicated that the fibrous ingrowth originating in the lacerated ciliary body cannot be eliminated completely.[14]

Despite these complications, corneal transplantation, even in the presence of retinal detachment, can be worthwhile in these eyes, because we achieved trans-

parent grafts and control of intraocular pressure in all eight eyes, and functional to good vision in four.

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FACTORS IN THE SUCCESSFUL SURGICAL MANAGEMENT OF BASAL CELL CARCINOMA OF THE EYELIDS

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We studied the clinical and histopathologic features of 165 basal cell carcinomas of the eyelids and classified the tumors into four types (nodular, ulcerative, morphea, and multicentric) that differ in incidence, results of incomplete excision, aggressiveness, and recurrence.

The value of frozen-section monitoring of the surgical margins was evident. In 39 cases excised with monitoring of margins by frozen sections, there were no recurrences. In 126 cases treated by excisional biopsy without frozen-section monitoring, 34 (26.9%) were incompletely excised and seven (5.5%) recurred.

The surgical treatment of basal cell carcinoma should always include the use of frozen-section monitoring of the surgical margins. This is especially important in the morphea, multicentric, and ulcerative types, in which the invasive extent of the tumor is often not apparent clinically. These forms are also more aggressive.

Basal cell carcinoma accounts for 85% to 95% of all malignant epithelial tumors of the eyelids.[1,2] In managing this tumor, it is important to recognize the morphea, ulcerative, and multicentric types of basal cell carcinoma as aggressive infiltrating tumors and to use frozen sections to monitor the margins of surgical specimens.[3]

We evaluated the factors involved in the successful management of basal cell carcinoma. The basic goal is the complete initial excision of the tumor, while sacrificing the least amount of normal eyelid tissue, so that eyelid reconstruction will be as simple as possible.

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MATERIAL AND METHODS

We studied 185 consecutive basal cell carcinomas from the eyelid with regard to tumor location, clinical features, preoperative diagnosis, surgical technique, frozen-section monitoring of margins, histopathologic features, and tumor recurrence. These carcinomas were submitted to the pathology laboratory here between Jan. 1, 1968, and December 1977.

Of the 185 carcinomas, we eliminated 17 from the study because of inadequate follow-up information. Three other cases were biopsy specimens before cryotherapy and were not included. Of the 165 remaining cases reviewed, 126 cases had been managed by excisional biopsy; in the other 39 cases the margins of the excision had been monitored by study of frozen sections.

Because most excised specimens with basal cell carcinoma are elliptical in

shape, we used and recommend the following method. If the long axis of the specimen was less than 10 mm, the specimen was bisected at its narrowest width through the center of the tumor. We prepared full-thickness sections for study from a plane parallel to that cut. This method was used to check three margins (two skin margins at the narrow part of the specimen, and the deep margin). If the ellipse was larger than 10 mm, the specimen was cruciated (Fig. 1). A 3- to 4-mm central portion was labeled A, and the two end portions were labeled B and C. We processed the B and C portions separately, and prepared sections from the plane perpendicular to the A specimen. We checked five surgical margins by this method. While obtaining the excisional biopsy specimen, the surgeon marked one end of the elliptical tissue with a suture to indicate its orientation (superior, inferior, nasal, or temporal). Thereafter, care was taken to ensure that the histologic cuts were clean and that the sections included the full thickness of the specimens.

In doing the excision the surgeon sought to make a definitive resection of the tumor, with 2 to 3 mm of surrounding normal full-thickness of skin and subepithelial tissue. The orientation of the suture was noted on the surgical pathology requisition sheet, along with all other pertinent clinical information. If frozen-section monitoring was performed, the surgeon also submitted a separate and labeled full-thickness sliver of skin and subcutaneous tissue from the area outside the definitive resection or from a site causing some concern because of the operative findings (Fig. 2). The additional specimen was frozen-sectioned at three levels, approximately 200 μ apart. If tumor was found at any of the three levels, a further excisional resection was performed, after which an additional sliver of skin and subepithelial tissue was submitted for frozen-section monitoring. This sequence was continued until no further tumor was identified. Specimens used for frozen-section monitoring were also fixed, processed, and sectioned for permanent sections and served as controls for the frozen sections.

If, on frozen sections, the tumor was multicentric or had a morphea pattern, the surgeon was notified that the margin of tumor might not be evident clinically.

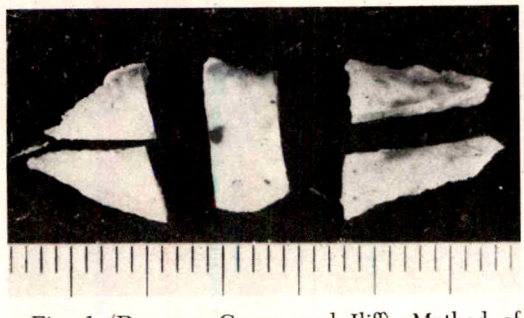


Fig. 1 (Doxanas, Green, and Iliff). Method of preparing an elliptical specimen measuring more than 10 mm in its longest dimension. A central 3- to 4-mm section is removed and labeled A. The two remaining end-portions are labeled B and C and then bisected perpendicular to A. Specimens A, B, and C are submitted separately.

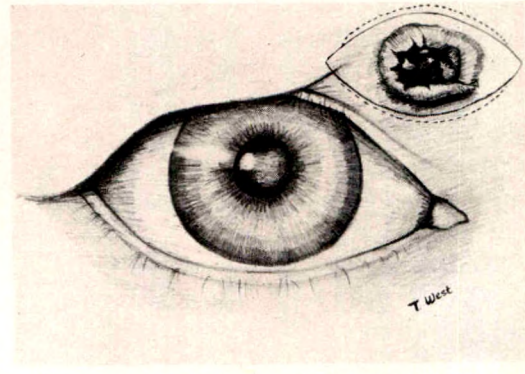


Fig. 2 (Doxanas, Green, and Iliff). Basal cell tumor of the upper eyelid. En bloc elliptical excision of the tumor (solid line). Additional slivers of tissue (dotted lines) were submitted for frozen sections.

RESULTS

Of the 165 tumors studied, 111 (67.2%) were in the lower eyelid, 24 (14.5%) were in the upper eyelid, 23 (13.9%) were at the inner canthus, and 7 (4.2%) were at the outer canthus.

The patients ranged in age from 19 to 88 years. Tumors occurring in the seventh and eighth decades of life accounted for 56.0% of the cases. No sexual predilection was noted; 77 women and 73 men were affected. The predilection for whites was striking. Only two of the 165 basal cell carcinomas were from blacks.

The follow-up period ranged from two to 12 years (mean, 6.4 years; median, 7 years).

A total of 47 surgeons excised the 165 basal cell carcinomas. One of us (C. E. I.) was the surgeon in 59 cases; another surgeon submitted eight specimens, 22 resident surgeons submitted 38 specimens, and 23 different surgeons submitted from one to three specimens. All but one surgeon were on staff here. The one outside surgeon contributed two cases.

The basal cell carcinomas had been incompletely excised in 34 (20.6%) of the 165 cases. All of these incomplete excisions were in the group of 126 cases in which frozen-section monitoring was not done; the occurrence of incomplete excision in this group was thus 34 of 126 cases (26.9%).

There were no incomplete excisions in the 39 cases monitored by frozen sections; this was confirmed by the permanent sections of the tissue used for frozen-section monitoring.

The tumors recurred in seven of 165 cases (4.2%) All seven were in the incomplete excisional biopsy group. In eight cases in the excisional biopsy group, the tumor margins were close to the surgical margin but there was no recurrence.

Among the 34 cases of incomplete excisional biopsy, there was no recurrence in 16 cases, seven of the 34 tumors were re-excised within one to two months, seven of the 34 recurred, and four were treated by irradiation. The radiation therapy ranged from 4,000 to 4,800 rads. There was no recurrence after radiation therapy.

Of the seven cases that were re-excised within one to two months after the initial incomplete excision, two showed no evidence of residual tumor and five had residual tumor.

Seven of the 34 patients (20.5%) in the incomplete excisional biopsy group had recurrence of tumor within one month to one year after excision. All recurrences were re-excised completely—four with frozen-section controls. Two cases were exenterations of the orbit.

We compared the preoperative clinical diagnoses in this series of excised basal cell carcinomas with the adequacy of surgical management, judged by the histopathologic verification of complete or incomplete excision. This analysis gave similar findings for completely and incompletely excised lesions. Evaluation of both groups of lesions together disclosed a correct preoperative clinical diagnosis in 61.2% of the cases. Analysis of incompletely and completely excised lesions disclosed a correct preoperative clinical diagnosis of basal cell carcinoma in 58.9% and 61.8% of these two groups, respectively. The most common misdiagnoses were papilloma and epithelial inclusion cyst.

The nodular type was present in 99 cases (60.0%), the ulcerative type in 35 cases (21.2%), the morphea type in 25 cases (15.1%), and the multicentric type in six cases (3.6%). Of the 34 incompletely excised cases, 17 (50.0%) were nodular, seven (20.5%) were ulcerative, nine (26.4%) were the morphea type, and one (3.0%) was multicentric. Of the seven recurring tumors, three were morphea, three ulcerative, and one nodular.

Discussion

The surgical management of basal cell carcinoma should be directed toward complete initial excision of the tumor. Inadequate or incomplete surgical excision of the basal cell carcinoma is obviously not desirable. One study,[4] for example, noted a recurrence in 34.8% of 66 incompletely excised basal cell carcinomas, most of them from the face. The recurrence rate of basal cell carcinoma of the eyelids has been reported as 0 to 10%.[5-8] Einaugler and Henkind[9] reported a 50% incidence of incompletely excised basal cell carcinoma in the periocular region. Studies by Older, Quickert, and Beard[3] and by Chalfin and Putterman[5] reported no recurrences of basal cell carcinomas when frozen-section monitoring of surgical margins was performed in 53 cases.

Our data confirm that frozen-section monitoring of surgical margins has an essential role in the management of basal cell carcinoma. Of the 165 cases, 39 (23.6%) were monitored by frozen sections. Permanent sections of the tissue that had been used for frozen sections confirmed in each instance the preliminary findings made with frozen sections.

Of the 165 cases, 126 (76.3%) were treated with excisional biopsy with no frozen-section monitoring of the margins. Of those 126 cases, 34 (26.9%) were incompletely excised. Eighteen of those 34 lesions were either re-excised or treated by radiation therapy. Patients whose tumors were removed by excisional biopsy without frozen-section monitoring required additional therapy in 14.3% of the cases.

The frozen-section monitoring of the 39 cases used 197 margin specimens (an average of 5.0 per case). The number of excised specimens used for frozen sections in specific cases varied. There were two specimens monitored in seven patients, three in 12, four in 11, five in one, six in two, ten in three, and 12, 18, and 26 specimens in one patient each.

Morphologic patterns of basal cell carcinoma refer to the microscopic configuration and extent of dermal invasion by the tumor. There are four basic morphologic patterns of basal cell carcinoma. The classification we used is a modification of the one proposed by Lund.[10] These categories are nodular, ulcerative, morphea, and multicentric.

Nodular pattern—This form of basal cell carcinoma appears clinically as a raised, firm, pearly nodule (Fig. 3, top left) with superficial telangiectatic vessels on its surface (Fig. 3, top right). The tumor consists of a discrete proliferation of cells that resemble the basal layer of the epithelium and undergo peripheral palisading (Fig. 3, bottom left and bottom right). Little connective tissue proliferation accompanies the tumor. Slow, gradual enlargement of the tumor may produce a surrounding connective-tissue pseudocapsule. Dermal involvement is usually confined to the area of apparent epithelial involvement. The tumor should be excised with 2 to 3 mm of apparently normal tissue to insure adequate excision. If the margins are doubtful, frozen sections should be used to monitor the surgical margins.

The nodular type of basal cell carcinoma may have various histologic features. It may be composed mainly of a solid proliferation of basaloid cells that have a characteristic peripheral palisading. (An artifactitious contraction of the tumor during the fixation process can produce a separation of the tumor from the surrounding connective tissue.) The nodular type may also have an adenoid or cystic appearance. The presence of an eccrine or an apocrine histologic differentiation may reflect the pluripotential nature of the basaloid cells composing the tumor.[11]

The presence of one or more cysts in a

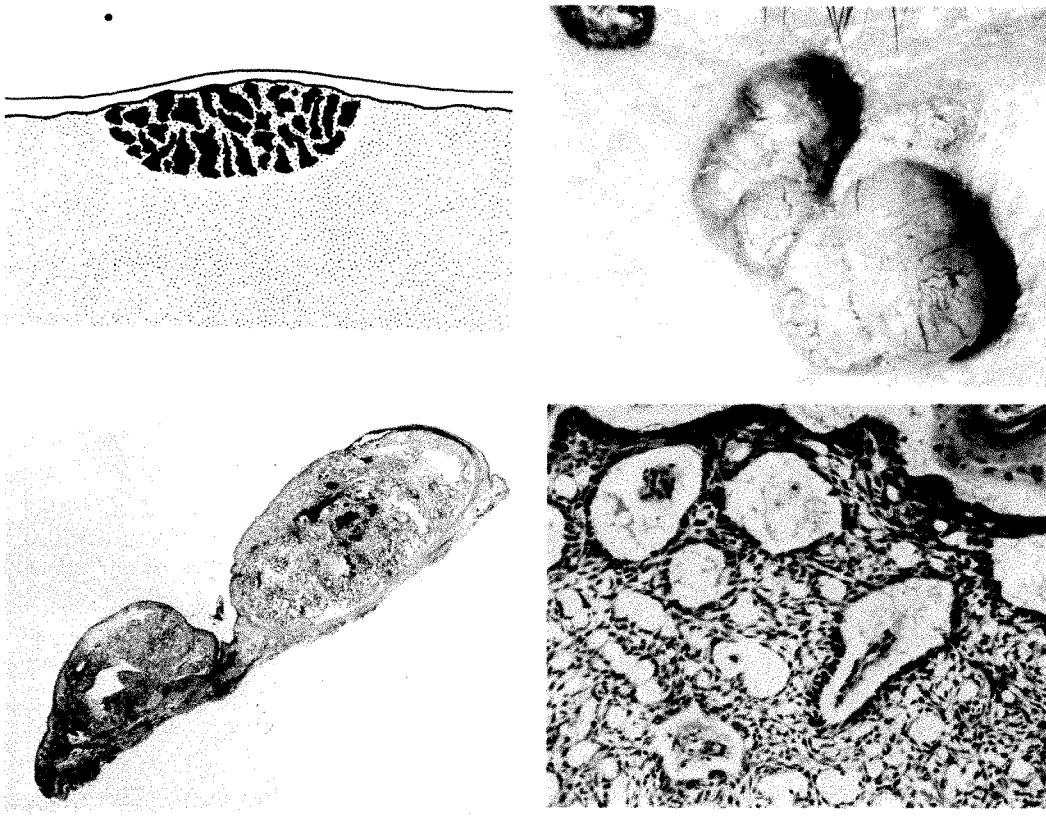


Fig. 3 (Doxanas, Green, and Iliff). Nodular basal cell carcinoma. Top left, Drawing shows a well-circumscribed tumor with pseudocapsule. Top right, Clinical appearance of a firm pearly nodule with superficial telangiectatic vessels. Bottom left, Histopathologic section of a nodular tumor. Despite the large size, the tumor does not extend deeply into the dermis or ulcerate the epithelium (hematoxylin and eosin, ×8). Bottom right, Higher-power view shows cystic tumor composed of basaloid cells (hematoxylin and eosin, ×250).

basal cell carcinoma is probably a result of the tumor's outgrowing its vascular supply, producing central necrosis. When the cystic component is a prominent feature of the tumor, clinical differentiation from an epithelial inclusion cyst becomes difficult.[12] Basal cell carcinomas may also have extensive hyperkeratosis or be raised from the surface epithelium, simulating a papilloma or seborrheic keratosis. Basal cell carcinoma may also occur at the base of a cutaneous horn.[13] In our series of cases, there was no differentiation toward sebaceous cells with foamy vacuolation or holocrine secretion. Because of the various clinical appearances of basal cell carcinoma, the preoperative clinical diagnosis was correct in only 60.9% of cases (Table).

Ulcerative pattern—This form of basal cell carcinoma derives its name from its clinical appearance (Fig. 4, top left and top right). A tumor with a true ulcer crater, with a raised pearly epithelial margin, is a characteristic of this form of basal cell carcinoma. This pattern also suggests a deeper, more infiltrative dermal component to the tumor (Fig. 4, bottom left). A pseudocapsule of surrounding connective tissue is not usually a

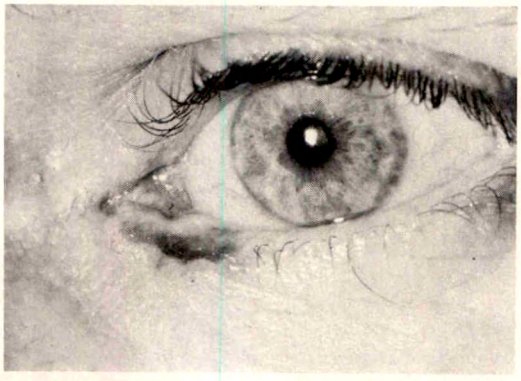
TABLE

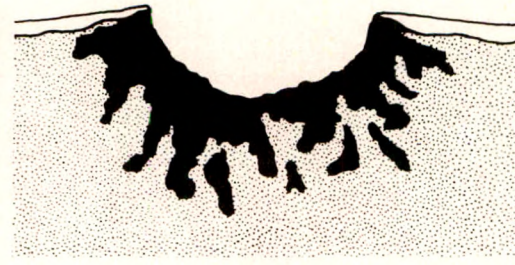
CLINICAL DIAGNOSIS AND DEGREE OF EXCISION OF 165 BASAL CELL CARCINOMAS

Clinical Diagnosis	Completely Excised		Incompletely Excised		Total	
	No.	%	No.	%	No.	%
Basal cell carcinoma	81	61.8	20	58.8	101	61.2
Papilloma	12	9.2	4	11.7	16	9.6
Epithelial inclusion cyst	6	4.6	5	14.7	11	6.7
Chronic inflammation	4	3.0	1	2.9	5	3.0
Nevus	3	3.0	—	—	3	1.8
Squamous cell carcinoma	1	0.07	—	—	1	0.06
No diagnosis given	24	18.3	4	11.7	28	16.9
Total	131	79.3	34	20.7	165	100.0

feature of ulcerative basal cell carcinoma. A greater inflammatory component may accompany areas of dermal involvement.

Morphea pattern—This form of basal cell carcinoma often appears clinically as a pale undurated plaque (Fig. 5, top left and top right). This type is also called sclerosing basal cell carcinoma. The connective tissue component is much greater in this form of basal cell carcinoma. Small islands of basaloid cells are embedded within a dense connective tissue frame-





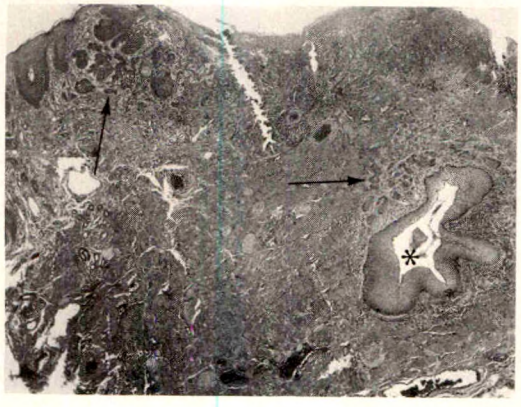


Fig. 4 (Doxanas, Green, and Iliff). Ulcerative basal cell carcinoma. Top left, Erosion of medial aspect of the left lower eyelid. Eyelashes are absent in involved area. Top right, Drawing shows loss of epithelium centrally and invasion into the dermis. Bottom left, Histopathologic section of ulcerative tumor shown at top left. The basaloid proliferation of cells infiltrates the dermis and underlying connective tissue (arrows), with some inflammatory reaction. The canaliculus (asterisk) is partially surrounded by the tumor (hematoxylin and eosin, ×30).

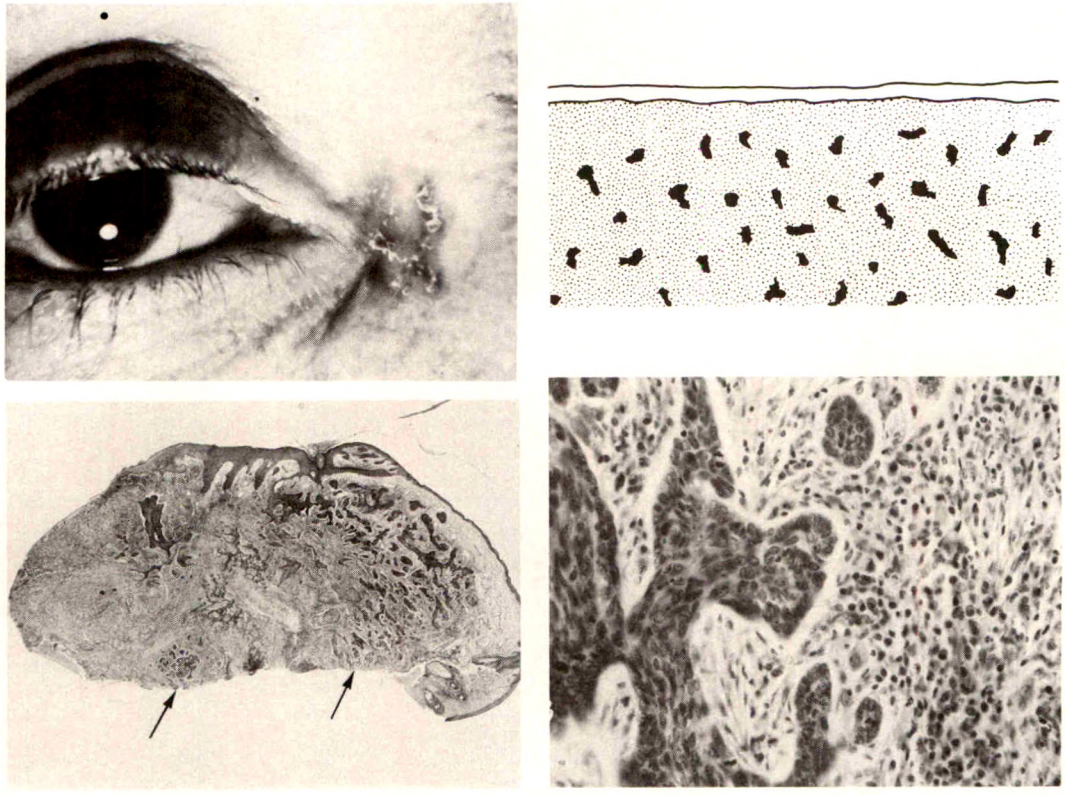


Fig. 5 (Doxanas, Green, and Iliff). Morphea type of basal cell carcinoma. Top left, Clinical appearance is that of a pale indurated plaque in the inner canthal region. Top right, Beneath an intact epithelium, discrete islands of tumor cells are encased in a dense connective tissue network. Bottom left, Histologically, the morphea type shows widespread dermal and connective tissue involvement beneath an intact epithelium. Note the presence of tumor at the surgical margins (arrows) (hematoxylin and eosin, ×18). Bottom right, Higher-power view shows cords of tumor invading deeper structures of the eyelid (hematoxylin and eosin, ×350).

work. This form of basal cell carcinoma is aggressive,[14-17] often extending deep into the dermis and connective tissue (Fig. 5, bottom left and bottom right), beyond the area of suspected clinical involvement. With time, the tumor may erode and disfigure the orbit (Fig. 6).

Multicentric pattern—This type of basal cell carcinoma has numerous lobules of tumor at the base of the epithelium or in the superficial corium (Fig. 7). The margin of the lesion is often beyond the area of suspected clinical involvement.

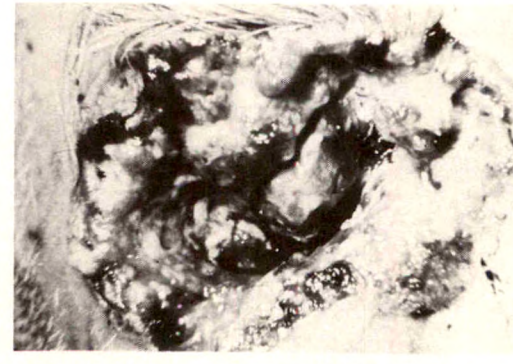


Fig. 6 (Doxanas, Green, and Iliff). Morphea type has eroded and disfigured orbital tissues. The globe could not be identified on clinical examination.

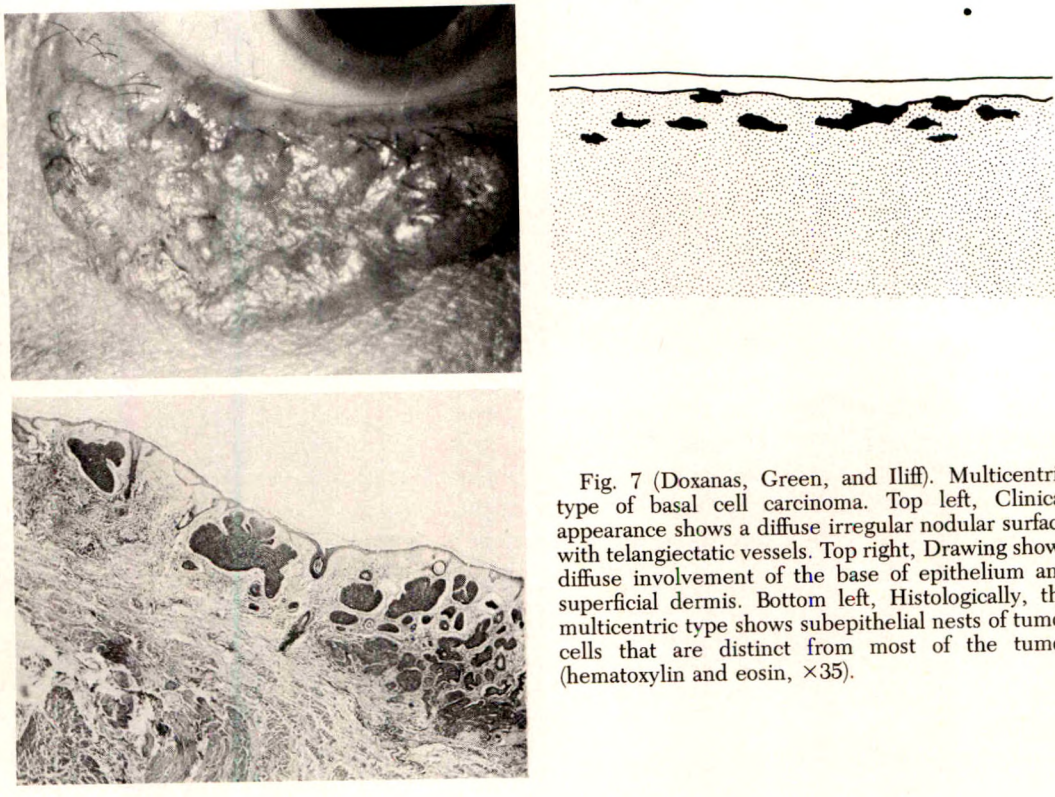


Fig. 7 (Doxanas, Green, and Iliff). Multicentric type of basal cell carcinoma. Top left, Clinical appearance shows a diffuse irregular nodular surface with telangiectatic vessels. Top right, Drawing shows diffuse involvement of the base of epithelium and superficial dermis. Bottom left, Histologically, the multicentric type shows subepithelial nests of tumor cells that are distinct from most of the tumor (hematoxylin and eosin, ×35).

The ulcerative, the multicentric, and the morphea types often extend beyond the clinically evident area. This is caused by the dermal involvement of the tumor, in areas without changes in the overlying epithelium. Excisional biopsy of basal cell carcinoma of these types is likely to result in incomplete removal of the tumor in a considerable percentage of cases, and we strongly recommend that surgical margins of the tumor be monitored by examination of frozen sections.

The Table summarizes the morphologic types of basal cell carcinoma in relation to the adequacy of surgical excision.

The nodular type occurred in 99 instances, and in 17 cases (17.1%) the tumor was incompletely excised. Similar rates of incomplete excision were present for the ulcerative and multicentric patterns of basal cell carcinoma (20.0% and 16.6%, respectively).

The morphea type, however, was much more likely to be incompletely excised. Nine (36%) of the 25 cases with the morphea pattern were incompletely excised—a rate about twice that of the other patterns. The high incidence of incomplete excision of the morphea type of basal cell carcinoma was probably caused by the more extensive tissue invasion and the inability to determine the margins of the tumor clinically and at the time of surgery.

The various morphologic patterns of basal cell carcinoma appear to have different degrees of aggressiveness.

We evaluated incompletely excised basal cell carcinomas with respect to the type of the tumor and possible differ-

ences in biologic behavior. Of the 34 incompletely excised tumors, there was no recurrence in 16 cases in which no additional treatment was given. Of those 16 cases, 12 were nodular, two were ulcerative, one was the morphea type, and one was multicentric. The predominance of the nodular type in this group of incompletely excised tumors and the lack of recurrence indicated the lower degree of biologic activity or aggressiveness of this form.

Five additional cases of the nodular type were incompletely excised. Of these five, three tumors were promptly re-excised, one received radiation therapy, and one recurred four months after the initial excision. The tumor recurred in only one of 13 (7.7%) incompletely excised nodular tumors not treated by re-excision or radiation therapy.

Seven of the 35 (20%) ulcerative basal cell carcinomas were incompletely excised. Five of these seven cases did not receive immediate re-excision or radiation therapy, and three of the five (60%) recurred.

When the morphea type of basal cell carcinoma was incompletely excised, the tumor was aggressive. Of the 25 tumors with the morphea pattern, nine (36%) were incompletely excised. Three of these nine were promptly re-excised, and all three showed evidence of residual basal cell carcinoma. Two other tumors were treated with radiation therapy and did not recur. Three of the four (75%) morphea tumors that were incompletely excised and that did not receive re-excision or radiation therapy recurred within four to 12 months. One did not recur.

Although the numbers used in the statistical analysis were not large, the recurrence rate of an incompletely excised morphea type of tumor was ten times that of the nodular type, and the ulcerative type recurred eight times more often than the nodular type.

Orbital exenteration was the surgical procedure in two cases of basal cell carcinoma that had invaded the orbit; in both cases the tumor was the morphea type. Postoperatively, one exenteration patient received 4,800 rads of radiation therapy, and maintenance therapy with cyclophosphamide (1 mg daily for four years). Both patients were alive without tumor recurrence after four and six years, respectively.

Two enucleations were performed in patients who had basal cell nevus syndrome (Gorlen's syndrome). In both patients, extensive surgical reconstruction and postoperative radiation therapy had produced contracted eyelids. Exposure keratopathy, ulceration, and perforation necessitated enucleation in both cases. There were no other patients with a tumor-diathesis syndrome in this series.

Aurora and Blodi[18] attempted to clarify the uncertainties of the biologic behavior of basal cell carcinomas by providing an elaborate classification. Their types included solid, superficial, adenoid, keratotic, fibrosing, and mixed. We found this classification to be useful but somewhat redundant. Our classification of four morphologic types seems to have more clinical application and, possibly, predicts biologic behavior better.

Beard[19] recognized two basic forms of basal cell carcinoma; he called these the nodular and the sclerosing forms. Although our views reflect those of Beard, we have further subdivided the tumor types. Beard combined the nodular and the ulcerative pattern of basal cell carcinoma as one type, but we have shown them to have different properties. The ulcerative basal cell carcinoma is classified not only according to its clinical appearance, but also by the increased dermal infiltration of the tumor, which

accounts for its deeper and wider extension. Also, in our series the course of the tumors that had been incompletely excised showed the nodular and ulcerative types to have distinct features. The ulcerative pattern, because of its deeper and more extensive involvement of the dermis, is more likely to recur after incomplete surgical excision.

Applying these concepts should be helpful in the management of basal cell carcinoma. The surgical approach can be adapted to the type of tumor. The nodular type of basal cell carcinoma is usually well circumscribed and does not extend deeply into the dermis. The nodular lesion may be bound by a pseudocapsule of surrounding connective tissue. Dermal involvement is usually confined to the area of apparent epithelial involvement. The tumor should be excised with 2 to 3 mm of apparently normal tissue to insure adequate excision. If the surgical margins are doubtful, frozen sections should be used to monitor them.

The ulcerative, the morphea, and the multicentric types often extend beyond the clinically evident area. This is caused by the dermal involvement of the tumor in areas without changes in the overlying epithelium. Excisional biopsy of basal cell carcinoma of those types is likely to result in incomplete removal of the tumor in a considerable percentage of cases and we strongly recommend that surgical margins of the tumor be monitored by examination of frozen sections.

Chalfin and Putterman[5] estimated the extent of tumor involvement preoperatively with a slit lamp and loupes, but they were correct in only 50% of their cases. They obtained at least 3 to 4 mm of normal-appearing skin at all margins. However, lesions that appear to be localized may require extensive surgery to remove the tumor entirely.

The postoperative management of patients with basal cell carcinoma should, of course, extend far beyond the time of suture removal. Patients who have a basal cell carcinoma are likely to develop additional tumors. Thus, these patients should be examined regularly for tumor recurrence and new tumors.

The proper management of incompletely excised basal cell carcinoma depends partly on the tumor type. In the present series, follow-up data showed that when a nodular tumor received no further postoperative therapy, there was a tumor recurrence in only one of 12 cases (8.3%). When an ulcerative or morphea type was incompletely excised, however, the recurrence rate was 60% and 75%, respectively. Thus, even from this limited number of patients, it appears that close observation of an incompletely excised nodular basal cell carcinoma is acceptable, because the recurrence rate is less than 10%. However, close observation of the other three types (the ulcerative and especially the morphea and the multicentric types) without re-excision or radiation therapy will probably result in tumor recurrence. Therefore, when those types of tumors are incompletely excised they are best managed by early surgical re-excision. The need for re-excison is greatly reduced by frozen-section monitoring during surgery.

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CRYPTOPHTHALMOS SYNDROME WITH BILATERAL RENAL AGENESIS

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We conducted a postmortem study of an infant with the abortive form of the cryptophthalmos syndrome. He had syndactyly and anal atresia and also showed features of Potter's (renal nonfunction) syndrome, including bilateral renal agenesis, pulmonary hypoplasia, and the typical facies. Histopathologic examination of both globes disclosed that the superior eyelid folds were partially replaced by skin that fused with the superior third of each cornea; there were also anomalies of the superior canaliculi and scarring of the corneas. We found no specific abnormality of the intraocular contents.

Cryptophthalmos is a rare, usually bilateral congenital malformation in which the eyelid folds are replaced by skin that passes from the forehead over the eye onto the cheek.[1] The incomplete form is characterized by involvement of the nasal orbital area alone; in the abortive form, the superior eyelid is replaced by an epidermal fold that adheres to the upper third of the cornea while the inferior eyelid seems to be normal.[2-4] The orbicularis oculi and levator palpebrae superioris muscles are generally present but the other eyelid structures, including remnants of the tarsal plates, are rarely evident. In most cases, the lacrimal puncta and lacrimal gland are absent and the globe, especially the anterior segment, is disorganized.

This malformation is usually part of a more generalized syndrome that includes many systemic anomalies. Sugar[2] named this syndrome cryptophthalmos-syndactyly in 1968 and François[3] called it malformative syndrome with cryptophthalmia in 1969. The mode of genetic transmission is usually autosomal recessive although cases with probable dominant inheritance have been reported. Different variations of the malformation, including absence of the eyelid anomaly,[5] may occur in the siblings. The most frequent malformations are syndactyly and craniofacial and urogenital anomalies.[2,3] Less common features include laryngeal, vesical, and anal atresia, umbilical hernia, displacement of the nipples and of the umbilicus, separation of the pubic symphysis, mental retardation, cardiac anomalies, and renal malformations, including agenesis.

Bilateral renal agenesis is the most common cause of the renal nonfunction syndrome described by Potter.[6,7] It is characterized by a typical facial appearance, pulmonary hypoplasia, limb positioning defects, and growth retardation.

We performed a postmortem study of an infant with cryptophthalmos, syndac-

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tyly, and bilateral renal agenesis who showed manifestations of both the cryptophthalmos syndrome and Potter's syndrome.

Case report

A male infant weighing 2,520 g was born by elective cesarean section after 35 to 36 weeks of gestation. This was the second pregnancy of the 27-year-old mother who had given birth to a normal girl one year previously. Severe oligohydramnios had been detected by ultrasonography two months before delivery. The Apgar scores were 1 at one minute and 0 at five minutes. The child died of severe asphyxia 18 minutes after birth.

Nonocular postmortem findings—The infant had the characteristic Potter facies, including a broad beak-like nose, distorted low-set ears, and micrognathia with a marked depression between the lower lip and the tip of the chin (Fig. 1).[6,7] The hairline extended over the temples to join the eyebrows. The hands and feet showed fusion of the second to the fifth digits beyond the proximal interphalangeal joints (Fig. 2). The nail-beds were rudimentary and consisted only of linear slits. The skin was dry and loose, especially on the hands, which resembled mittens. Internal anomalies included hypoplastic lungs, absence of the kidneys and ureters, a contracted bladder with no identifiable ureteric orifices, anal stenosis with a dilated sigmoid colon and rectum, and a laryngeal cyst measuring 0.5 cm in diameter.

Ocular postmortem findings—On the nasal side, the right and left superior eyelids were replaced by membranes of skin 7 and 20 mm long respectively. The membranes had no eyelashes and fused with the globe in the superior aspect of each cornea (Figs. 3 and 4). The superior lacrimal puncta were absent. Temporally, the eyelids appeared more normal, but the superior fornices, which were absent nasally, were shallow. There was ectropion of the thickened and shortened inferior eyelids with partial obliteration of the inferior fornices. The inferior lacrimal puncta appeared patent. A fold of conjunctiva covered the inferior corneoscleral limbal area of both globes and was especially prominent inferonasally. The opalescent right and left corneas measured 8 × 7 mm and 9 × 8 mm, respectively; the globes, which appeared normal in other respects, each measured 17 mm in diameter. We sectioned them vertically and found the intraocular contents to be normal.

Microscopic examination—The right upper eyelid, which was present only temporally, had a poorly

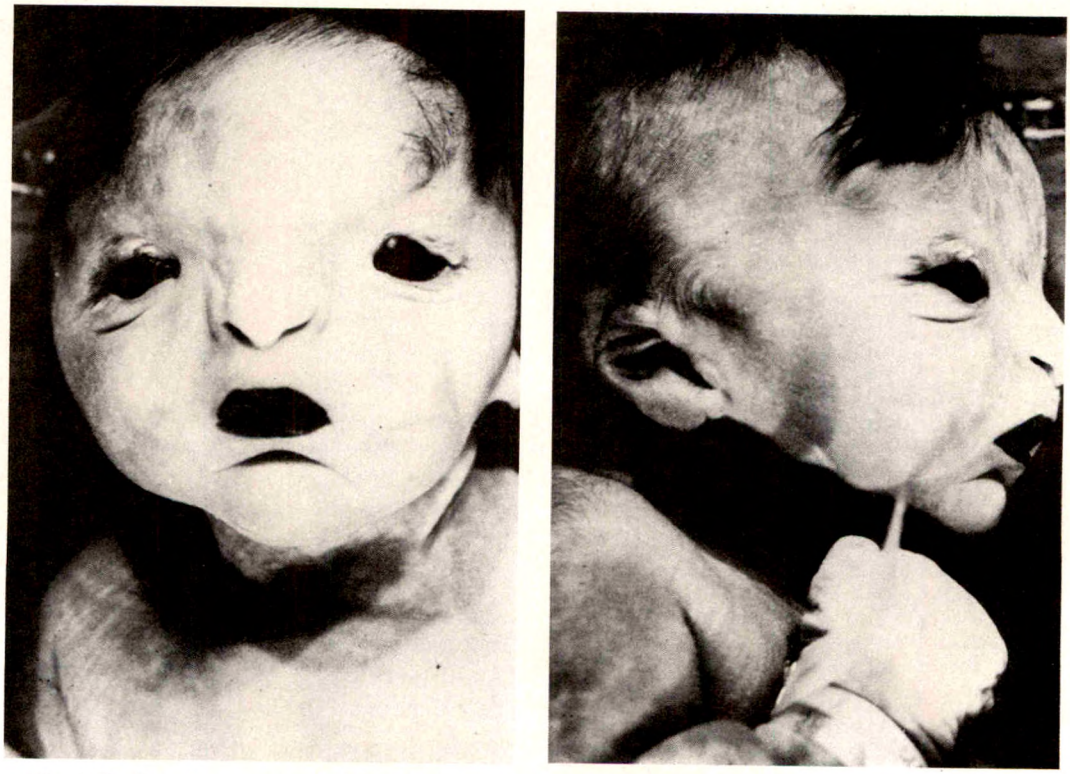


Fig. 1 (Codère, Brownstein, and Chen). Left, Potter's facies with broad beak-like nose and micrognathia. The scalp hair extends over the temples to join the temporal remnants of the eyebrows. Right, Distorted low-set ear and prominent depression below lower lip.

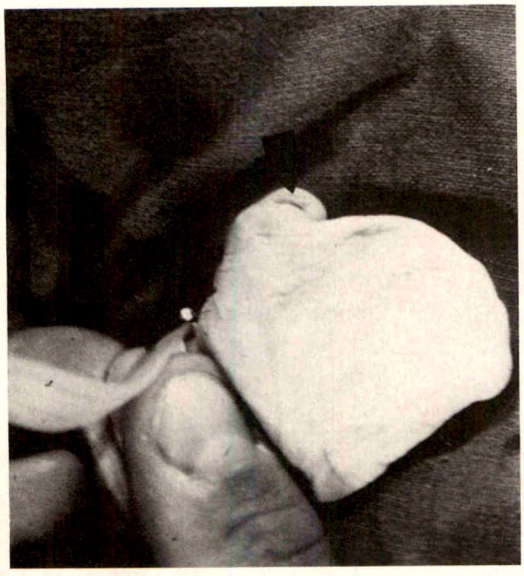


Fig. 2 (Codère, Brownstein, and Chen). Syndactyly of second to fifth digits and rudimentary slit-like nail on deformed thumb (arrow) of right hand.



Fig. 3 (Codère, Brownstein, and Chen). Right orbital contents show replacement of nasal aspect of superior eyelid fold by skin that fuses with the superior cornea. Inferiorly, a thick conjunctival fold is present and there is an entropion of the shortened and thickened inferior eyelid.

developed tarsus and meibomian glands (Fig. 5). Lacrimal gland tissue extended to the superior margin of the right tarsus. No tarsus, meibomian glands, or canaliculus were evident in the superior skin membrane that replaced the nasal portion of the right upper eyelid. The left upper eyelid was present only in the most temporal sections and was devoid of meibomian glands but displayed some eyelashes at its margin (Fig. 4); the tarsus was malformed. The left lacrimal gland was present in its normal position. Serial sections demonstrated the presence of the left superior canaliculus but it terminated at the medial extension of the conjunctiva instead of opening at the eyelid margin (Fig. 6).

The cutaneous membranes that replaced the nasal portion of the eyelids covered the superior third of the cornea on both sides (Fig. 7). Its epithelium displayed epidermidalization with rete peg formation and hyperkeratosis but there were no skin appendages. Adjacent to the fusion site of the skin and cornea, the superficial cornea had the appearance of conjunctiva; it showed its normal corneal architecture more centrally, although there was focal scarring of Bowman's membrane and of the superficial stroma.

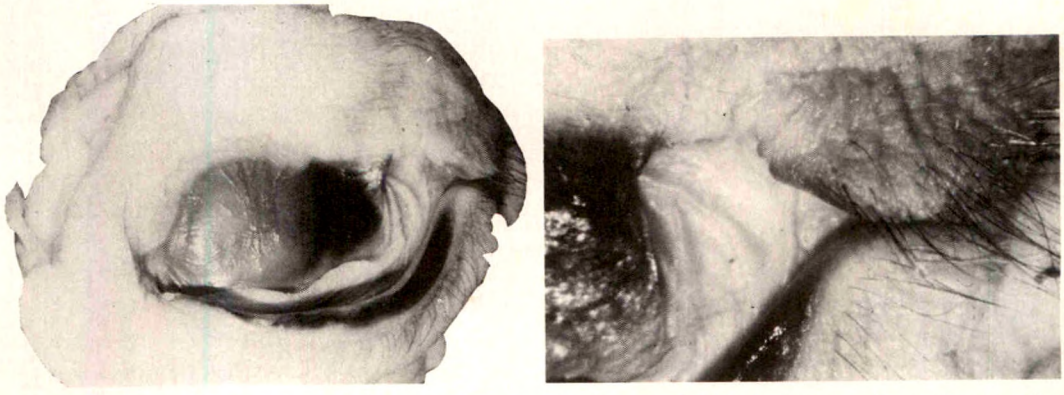


Fig. 4 (Codère, Brownstein, and Chen). Left, Left orbital contents show findings similar to those on the right (shown in Figure 3) except that the replacement of the superior eyelid fold by skin is more extensive. Right, Only the most temporal portion of the superior eyelid appears normal and has eyelashes.

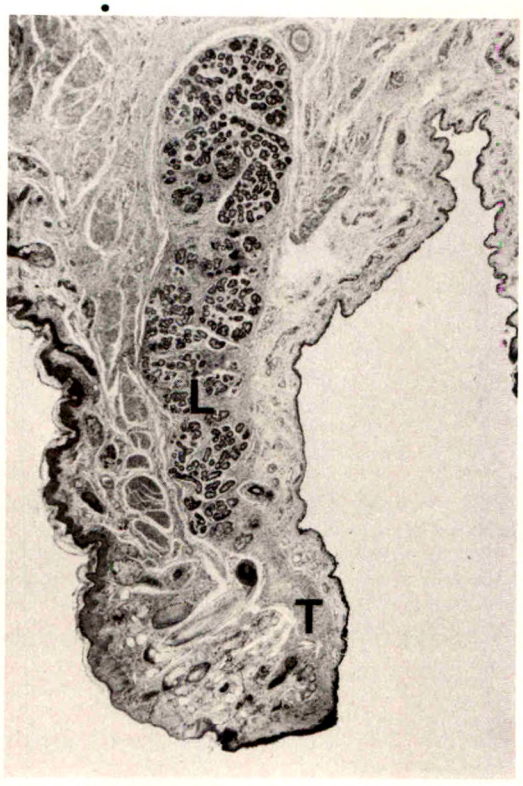


Fig. 5 (Codère, Brownstein, and Chen). Temporal portion of the right upper eyelid contains lacrimal gland tissue (L) and poorly developed meibomian glands in the deformed tarsus (T). The fornix is shallow (hematoxylin and eosin, ×25).

Both lower eyelids contained a malformed tarsus and meibomian glands. Many goblet cells were present in the inferior conjunctival fold that replaced the inferior peripheral corneal epithelium. The inferior canaliculi and puncta appeared normal. The deeper portion of both corneas and the remainder of both globes showed no significant abnormalities.

Discussion

This infant, who had the abortive form of the cryptophthalmos malformative syndrome, had bilateral renal agenesis. François[3] noted renal anomalies in five of the 46 cases of cryptophthalmos that he reviewed. Three of the patients had unilateral renal agenesis; the malformation in the other two was not specified. In a study of 50 cases of bilateral renal agenesis, the only significant ocular anomaly Potter[6] found was microphthalmia in one patient. Brownstein, Kirkham, and Kalousek[8] reported one case of bilateral renal agenesis associated with multiple intraocular anomalies; the eyelids were normal. More recently, Carter, Evans, and Pescia[5] reviewed 103 cases of bilateral renal agenesis and found two siblings with anomalies suggestive of the cryptophthalmos syndrome. The boy had unilateral cryptophthalmia and absent kidneys, and his sister had syndactyly, laryngeal atresia, and bilateral renal agenesis; her eyelids were normal.

In Potter's (renal nonfunction) syndrome, the pulmonary hypoplasia and typical facies have been attributed to the associated oligohydramnios.[6,7] The other malformations noted in our patient, including the syndactyly, anal atresia, and abnormal hair distribution, are frequently encountered in the cryptophthalmos syndrome.[3] The superior lacrimal punctum is frequently absent in cryptophthalmia; this may be the result of the entire canaliculus being absent, as occurred on the right side of our patient, or of an aberrant opening of the canaliculus into the conjunctiva, as occurred in our patient's left globe. Lacrimal gland tissue, even when ectopic, is seldom found in cryptophthalmos; however, it was present bilaterally in our patient. Most cryptophthalmic globes tend to display internal disorganization, especially of the anterior segment; this is sometimes associated with microphthalmia and orbitopalpebral cyst formation.[9] In our case, except for the superficial corneal scarring that was probably caused by intrauterine exposure to the external environment, the intraocular structures were normal, a finding rarely described in globes from other patients with this condition.[3,10]

Genetic counseling—In most cases the counsellor should assume that the mode of inheritance is probably autosomal recessive. A complete family history should be obtained and examinations (including

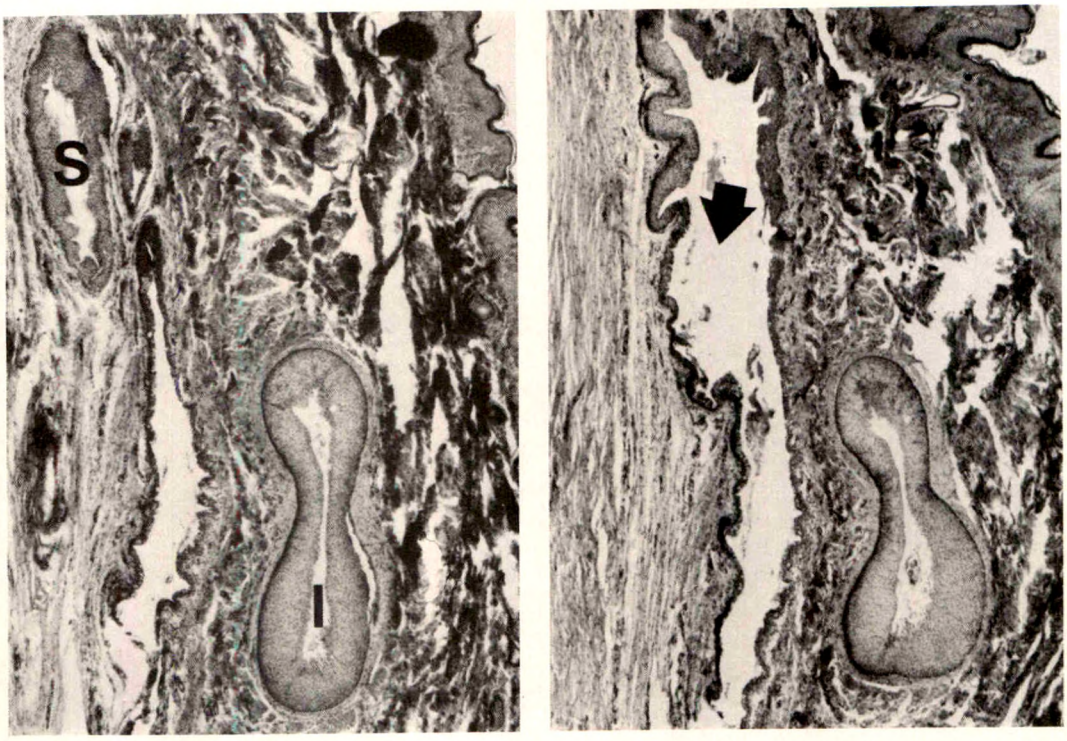


Fig. 6 (Codère, Brownstein, and Chen). Left, Left superior (S) and inferior (I) canaliculi near inner canthal region (hematoxylin and eosin, ×40). Right, Left superior canaliculus opens into conjunctival sac (arrow) (hematoxylin and eosin, ×40).

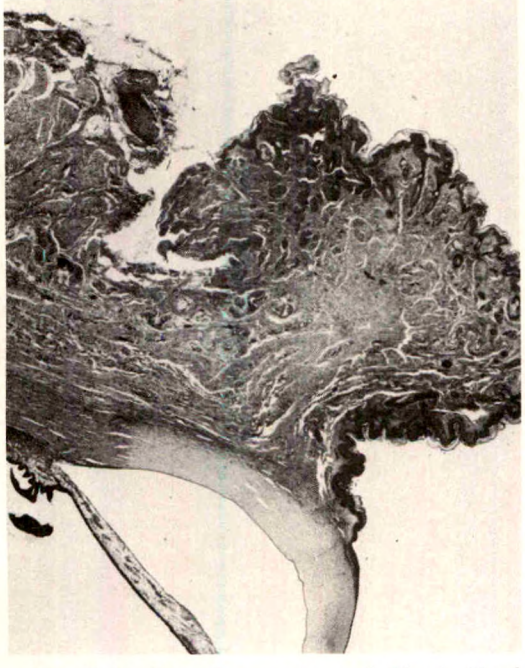


Fig. 7 (Codère, Brownstein, and Chen). Nasal aspect of right upper eyelid is replaced by epidermal tissue that fuses with superficial, superior third of cornea (hematoxylin and eosin, ×25).

renal ultrasonography) performed, especially of the siblings, to uncover formes frustes cases of this syndrome, including those without ocular anomalies but with other substantial but occasionally less apparent malformations such as unilateral renal agenesis.[5] It is important to differentiate those cases of renal agenesis that are part of the cryptophthalmos syndrome from those that occur in classical Potter's syndrome, because the likelihood of recurrence in subsequent pregnancies is different. In the cryptophthalmos syndrome, the risk of another malformed child is approximately 25%, whereas in Potter's syndrome it has been estimated to be about 3.5%.[5]

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OPHTHALMIC MINIATURE

As respects treatment, the patient had, fortunately for his sight, resisted the importunities of flocks of itinerant eye doctors, who, to use the forcible language of an eccentric medical friend, have "gone a whoring with the word oculist." Such impertinent, pestiferous vagabonds, now infest our medical Israel in swarms, such as "neither our fathers, nor our fathers' fathers have seen, since the day that they were upon earth!"

O! for a professional Moses, that might find favor with the Lord, and successfully entreat Him to smother out the vile frogs, and flies, and lice, and locusts, that have been showered down upon us, in retribution for the woful and unpardonable neglect of the scientific profession, in acquainting themselves with the beautiful science of ophthalmology!

E. Williams, *North American Medico-Chirurgical Review*
September, 1857

TWO CASES OF SUSPECTED FAMILIAL ERYTHROPHAGOCYTIC LYMPHOHISTIOCYTOSIS

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Boston, Massachusetts

We studied two children who died of suspected familial erythrophagic lymphohistiocytosis. The first child, a 7-year-old boy, had fever, diffuse pulmonary infiltrates, bilateral choroidal effusions, diabetes insipidus, and marked meningitis with increased cerebrospinal fluid protein. Ocular histologic studies disclosed diffuse choroidal infiltrates consisting of atypical mononuclear cells with foamy cytoplasm. Similar histologic changes were seen in the lungs, liver, brain, and kidneys. The second child, the 5-year-old male cousin of Patient 1, had fever, hepatosplenomegaly, pulmonary infiltrates, and cerebrospinal fluid pleocytosis. Histologic changes were similar to those in the first case, but viral particles of the herpes simplex virus group were noted in brain tissue as well. The findings of virus particles in this histiocytic proliferative disease suggested a defect in the inflammatory response in these patients.

Disseminated histiocytic inflammatory lesions and malignancies can be classified by the degree of cellular maturity and functional capacity found in them. The histiocytic lesions discussed in this report fall into two categories: histiocytosis X and the familial lymphohistiocytoses. Our cases showed features of both Letterer-Siwe disease and the syndrome of familial erythrophagocytic lymphohistiocytosis. We did not find orbital involvement with proptosis, a well-known ocular manifestation of Letterer-Siwe disease and Hand-Schüller-Christian disease, in our patients, nor have other investigators observed it in their patients with familial erythrophagocytic lymphohistiocytosis.[1,2] Also, Letterer-Siwe disease is unlikely to produce cerebral lesions and is not familial.[3-5] Both our cases contained these features.

Familial erythrophagocytic lymphohistiocytosis is a lymphoproliferative disorder characterized by diffuse pulmonary infiltrates, bilateral choroidal effusions with secondary retinal detachments, diabetes insipidus, and mild meningitis with cellular pleocytosis and markedly increased cerebrospinal fluid protein. Our second patient, a cousin of the first, died of a clinically similar disease, but he had clinically proven infectious mononucleosis and may have had Epstein-Barr virus infection.

CASE REPORTS

Case 1—This 7-year-old boy had been in good health until three weeks before he entered the hospi-

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tal. At that time, he began to complain of headaches, photophobia, and blurred vision. He had polyuria and polydipsia. He had spiking fevers, with temperatures as high as 40 C. A chest X-ray film disclosed diffuse pulmonary infiltrates.

He had had pneumonia in 1976 and Stevens-Johnson syndrome, cause undetermined, in 1977. He had had no previous ocular involvement. When admitted to the hospital, the patient was alert, but preferred to remain quiet and still. He was pale; his heart rate was 116 beats/min, respirations, 36 breaths/min, and temperature was 37.2 C. There was no lymphadenopathy. The chest examination disclosed fine bibasilar rales; results of a cardiac examination were normal. His abdomen was soft and nontender, and the liver and spleen were not enlarged.

Visual acuity was R.E.: hand motions, and L.E.: counting fingers at 3 ft. An ophthalmoscopic examination disclosed marked choroidal effusions with secondary retinal detachment bilaterally. There was pigment clumping in the macular area but no papilledema.

Initial laboratory studies gave the following values: hematocrit, normal; white blood cell count, 8,400/mm^3 with 65 polymorphonuclear leukocytes, 3 band cells, 26 lymphocytes, 3 monocytes, 1 eosinophil, 1 basophil, and 1 myelocyte; platelet count, 263,000/mm^3; and erythrocyte sedimentation rate, 40 mm/hr. Electrolyte levels were normal, as were creatinine, blood urea nitrogen, and glucose levels. Serum immunoglobulin G and M showed diffuse mild increases; immunoglobulin A was moderately increased. Liver function tests were normal except for the lactic dehydrogenase level, which was increased to 498 units/ml.

Lumbar puncture disclosed an increased protein level of 650 mg/100 ml; the glucose level was normal. There were 12 red blood cells and 21 white blood cells (16% polymorphonuclear leukocytes and 84% lymphocytes).

In summary, this child had diffuse pulmonary infiltrates, bilateral choroidal effusions, diabetes insipidus, and mild meningitis with markedly increased cerebrospinal fluid protein. None of the cultures grew pathogens.

The patient was treated with intravenously administered erythromycin and synthetic antidiuretic hormone for diabetes insipidus. His condition stabilized, and his visual acuity improved to R.E.: 6/60 (20/200) and L.E.: 6/15 (20/50). The choroidal effusions resolved. Pulmonary infiltrates, however, remained unchanged, and the administration of corticosteroids did not affect the course of the illness.

After two months of treatment, the child appeared well, although he continued to have temperatures between 37.8 C and 38.3 C. A few weeks after he left the hospital, his condition deteriorated. When he was readmitted, the physical examination showed a profound peripheral neuropathy. A second lumbar puncture showed fewer white blood cells, but the protein content had climbed to 1,040 mg/100 ml. He had spiking temperatures as high as 40 C. The white blood cell count was 5,000 to 8,000/mm^3 with a left shift, and there were myelocytes and metamyelocytes on the peripheral smear. All blood cultures were negative. Despite the intravenous administration of erythromycin, the patient suddenly became comatose. Computed axial tomography confirmed the presence of a massive right ventricular hemorrhage. Supportive therapy was administered, but the patient died shortly thereafter.

Pathologic findings—At autopsy, the lungs showed many areas of consolidation and fibrous adhesions to the pleura. The liver and spleen were enlarged and congested.

Histologic study of the lungs showed extensive intra-alveolar and interstitial collections of foamy histiocytes, some PAS-positive, with central areas of coagulative necrosis. Plasma cells and lymphocytes dominated the infiltrate. Brown-Brenn and fungal stains were negative (Fig. 1).

In the liver, sinusoids contained histiocytes and hepatocytes were filled with large fat vacuoles. Splenic follicles were well-formed and showed marked congestion and an increase in the plasmacytoid series. T cell areas appeared depleted, but B cell areas were adequately populated with cells and degenerated plasma cells (Russell bodies).

The thymus showed extensive lymphoid depletion, with predominant fibrovascular stroma, a few remaining aggregates of epithelial cells, psammoma bodies, and occasional Hassal's corpuscles. Thoracic and abdominal lymph nodes were enlarged, with nodular aggregates of foamy histiocytes that showed central coagulation necrosis.

Several histologic sections from the cerebral hemispheres, brain stem, spinal cord, and peripheral nerves showed a diffuse perivascular cellular infiltrate, more prominent in the white matter, composed of histiocytes, plasma cells, and small lymphocytes. A similar infiltrate appeared in the peripheral sacral and popliteal nerves, with patchy demyelination.

We found sheets of foamy histiocytes, associated

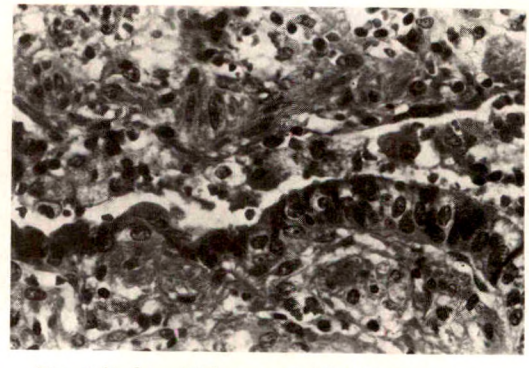


Fig. 1 (Lubin, Walton, and Albert). Case 1. Lung shows interstitial and intra-alveolar collections of foamy histiocytes. Plasma cells and lymphocytes are also present (hematoxylin and eosin, × 160).

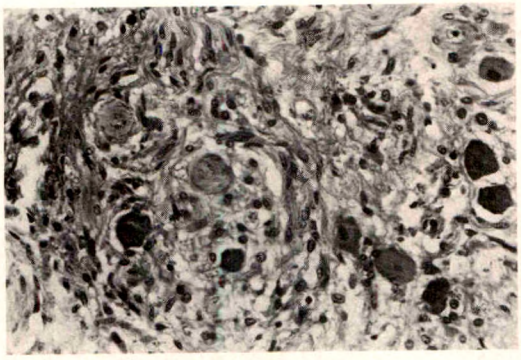


Fig. 2 (Lubin, Walton, and Albert). Case 1. Ganglion with inflammatory cell infiltrate (hematoxylin and eosin, × 100).

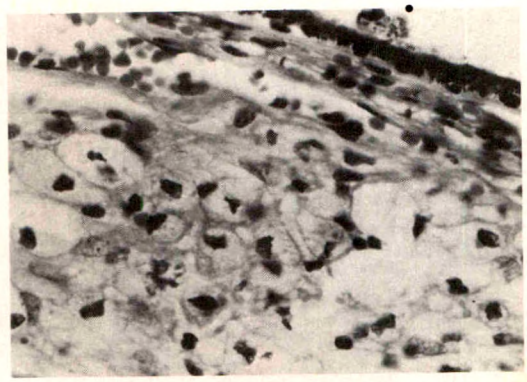


Fig. 3 (Lubin, Walton, and Albert). Case 1. Histiocytic cell infiltrate in the choroid (hematoxylin and eosin, × 200).

with plasma cells and lymphocytes, in the lungs, liver, diaphragm, lymph nodes, testes, kidneys, brain, peripheral nerves, and ganglia (Fig. 2). The dense nuclear chromatin pattern and eosinophilic cytoplasm showed that these histiocytes were fairly well differentiated. Phagocytosis of erythrocytes by these cells was observed in some sections. The cornea showed peripheral inflammatory pannus and chronic inflammation in the trabecular meshwork. We found mild lacy vacuolization of the iris pigment epithelium and chronic nongranulomatous inflammation of the stroma. Ciliary processes were hyalinized, with mild round-cell infiltration in the ciliary body. The lens showed posterior migration of nuclei. The vitreous was normal. The choroid was diffusely infiltrated with large, atypical mononuclear cells, with pleomorphic nuclei and foamy cytoplasm (Fig. 3). There was a serous choroidal detachment and choroidal hemorrhages were observed in some areas. The retina was detached, with serous subretinal exudate. There was loss of ganglion cells and peripheral cystoid changes. The optic nerve showed papilledema, and astrocytes had proliferated.

Electron microscopy showed histiocytes in several areas of the choriocapillaris. Some of these cells, which resembled macrophages, possessed cytoplasmic projections that engulfed collagen fibers (Fig. 4); others contained many cytoplasmic ingesting vacuoles.

Case 2—This 5-year-old boy, the maternal cousin of Patient 1, became ill with vomiting, diarrhea, persistent feelings of weakness and fatigue, and a low-grade fever. His medical history was noncontributory. Physical examination disclosed moderate generalized lymphadenopathy, and the spleen and liver were palpable 2 cm below the costal margins. Laboratory studies showed that the hemoglobin was 11.6 g/100 ml and the white blood cell count 5,600/mm^3 with an abnormal differential. A monospot test was positive, and heterophil antibody was positive at 1:56. Liver function studies gave markedly increased results. The preliminary diagnosis was infectious mononucleosis.

One week later, the boy had a rash and a series of seizures on the right side. These seizures were poorly controlled by medication. The pupils were equal and sluggishly reactive, and there was nystagmus on right lateral gaze. There was a maculopapular rash on the face, trunk, and extremities. Laboratory studies disclosed a white blood cell count of 15,150/mm^3 with 58% lymphocytes (25% of which were atypical) and 42% polymorphonuclear leukocytes. Lactic dehydrogenase and serum glutamic oxaloacetic transaminase levels were too high to be quantified. Lumbar puncture disclosed a cerebrospinal fluid protein level of 230 mg/100 ml, glucose level of 66 mg/100 ml, and 265 cells/mm^3, 99% of which were mononuclear.

The patient continued to have generalized seizures for the next three or four weeks. Papilledema developed and there was progressive central nervous system deterioration. The monotest continued to be positive, and heterophil antibody was positive at 1:112 (not absorbed). The hemoglobin level decreased to 10.2 g/100 ml; the white blood cell count was 12,500/mm^3 with 55% polymorphonuclear leukocytes, 35% lymphocytes, and 10% monocytes. A cerebrospinal fluid study for heterophil, lupus erythematosus preparations, and antinuclear antibody tests were negative. Complement fixation titers for cytomegalovirus, adenovirus, herpes simplex virus, and *Mycoplasma pneumoniae* were less than 1:8. Indirect fluorescent-tagged antibody for Epstein-Barr virus was positive (1:50) on two separate occasions. Mononucleosis B cell tests were positive (1:40) on two occasions (this was significant because it indicated a convalescent state). Blood cultures were negative. A chest X-ray film showed perihilar and peribronchial infiltrates. One day before the boy died, a lumbar puncture showed an opening pressure of 470 mm Hg, a white blood cell count of 179/mm^3 with 90% lymphocytes and 10% monocytes, and a protein level of 188 mg/100 ml. Despite corticosteroid therapy, anticonvulsants, and antibiotics, the patient died.

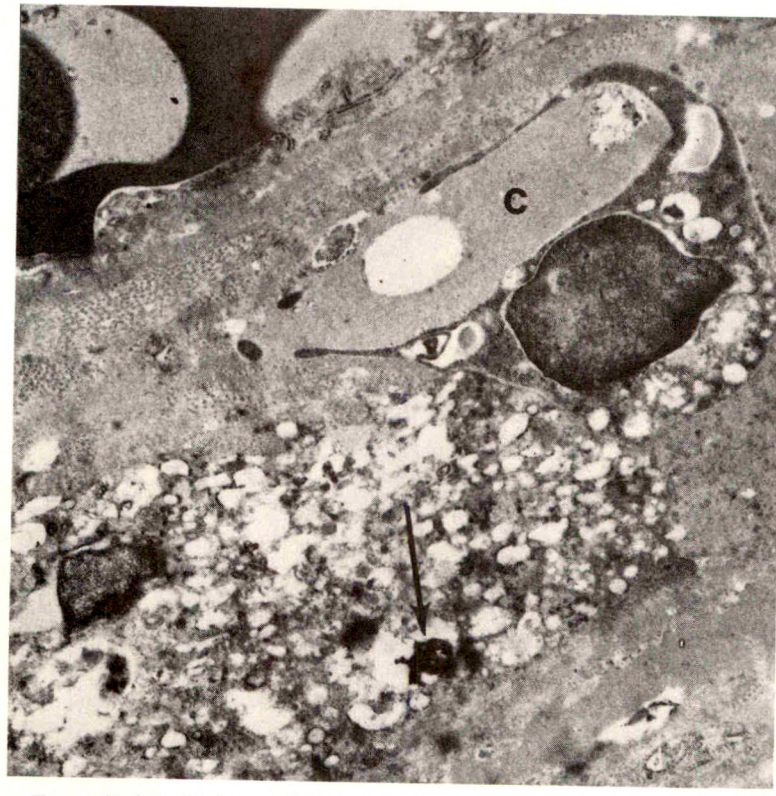


Fig. 4 (Lubin, Walton, Albert). Case 1. Histiocytes in the choriocapillaris have many vacuoles containing electron-dense debris (arrow). The histiocyte at the upper right has cytoplasmic projections engulfing collagen fibers (C) (× 10,920).

Pathologic findings—The autopsy showed bilateral acute bronchopneumonia. There were histiocytes in the alveoli and foreign-body giant cells were found in several areas (Fig. 5). The liver was enlarged, with a portal infiltrate of many histiocytic cells, lymphocytes, and plasma cells. The pituitary gland and spleen were also infiltrated. The brain sections disclosed white, softened areas in the left caudate nucleus and bilaterally in the globus pallidus. There was leptomeningeal, perivascular, and intraparenchymal infiltration by many pleomorphic monocytic cells, histiocytes, giant cells, and plasmacytoid cells. Large vacuolated histiocytes were present as well. The thymus gland was markedly depleted, with extensive fibrosis. Lymph nodes were also depleted, but contained nodules of histiocytes and lymphocytes.

The cytologic characteristics of the histiocytes in this case showed less differentiated cells than those found in the previous case. The cells were enlarged, with vesicular, irregular, or folded nuclei and had a loose chromatin pattern with prominent nucleoli. Cytoplasm was abundant and eosinophilic. Cellular phagocytosis was evident and mitoses were common.

Electron microscopic studies of the necrotic lesions in the central nervous system showed virus-like particles with diameters of 90 to 110 nm and dense cores measuring 60 to 80 nm with a lighter halo and rim in the cytoplasm of some well-preserved cells. The structure and dimensions of these particles were similar to those of the herpes simplex virus.[6]

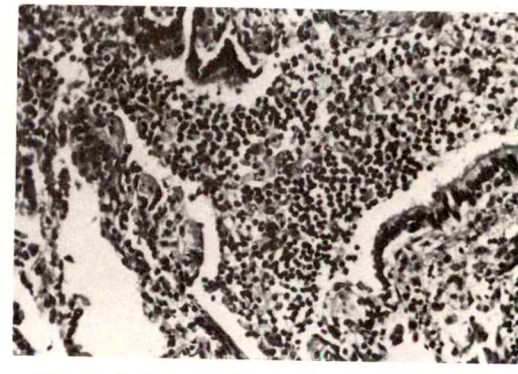


Fig. 5 (Lubin, Walton, Albert). Case 2. Lung shows acute bronchopneumonia with histiocytes (hematoxylin and eosin, × 100).

Discussion

These cases illustrate the difficulty of separating and categorizing the histiocytic diseases. Two related children died of lymphoproliferative disorders. Patient 1 had a syndrome clinically similar to Hand-Schüller-Christian disease, but with a rapidly progressive downhill course like that of Letterer-Siwe disease. Pathologic studies indicated familial erythrophagic lymphohistiocytosis with intraocular manifestations. The second case clinically appeared to be infectious mononucleosis,[7] but histopathologic examination of tissues at autopsy disclosed histiocytic infiltrates consistent with familial erythrophagocytic lymphohistiocytosis and similar to those found in Letterer-Siwe disease. Farquhar and Claireaux[8] first described familial erythrophagocytic lymphohistiocytosis in 1952 as familial hemophagocytic reticulosis. Nelson and associates[9] described the syndrome more fully in 1961. Published reports have described 38 affected children from 21 families. As many as five siblings have died,[4] and Buist, Jones, and Carens[10] reported another instance of the disease occurring in first cousins. Price and associates[5] emphasized the involvement of the central nervous system. By the time of their report (1971), the disease, which includes diffuse intracerebral lymphohistiocytic infiltrations, was considered familial erythrophagic lymphohistiocytosis.[11] The subject of central nervous system involvement in familial erythrophagic lymphohistiocytosis had been reviewed by Schoek and associates in 1963. Peterson and Kuwabara[1] described the ocular findings in 1968, and Appen and associates[2] described the ocular findings again in 1976, but did not find the choroidal involvement observed by Peterson and Kuwabara.

This disease has been reported in children from 2 weeks to 7 years old, but two thirds of the cases appear in the first three months of life. The clinical course usually involves intermittent fever, failure to thrive, pancytopenia, white blood cell counts of 5,000/mm^3 that are normocytic and normochromic, anemia, and hepatosplenomegaly. Pulmonary infiltrates have been found in one third of the cases.[12] There is minimal peripheral lymphadenopathy, and no skin lesions. The disease has a familial occurrence and a rapidly fatal course. Histiocytic infiltration causes reticuloendothelial failure that may cause disseminated intravascular coagulation, leading to a bleeding diathesis, the terminal event in our Case 1. Lymphocytic meningitis is also a common finding, with central nervous system dysfunction occurring in 29% of the cases.[12]

Although no infectious agent has ever been cultured from lesions in these children, an infectious origin has not been ruled out.[5] This condition has many similarities to X-chromosome-linked recessive reticuloendotheliosis,[13] but typically has diffuse infiltration of mature plasma cells and bizarre mononuclear cells which resemble T cells in their response to phytohemagglutinin.

Familial erythrophagic lymphohistiocytosis differs from Letterer-Siwe disease in having more mature histiocytes that lack malignant cellular features. There may be some lipid material in the cytoplasm.[14] Letterer-Siwe disease is not considered a familial disorder, nor does it include cerebral involvement. In familial erythrophagocytic lymphohistiocytosis, cohesive cell masses do not appear, whereas simulated tumor may appear in Letterer-Siwe disease. In Letterer-Siwe disease, there are eosinophils in the infiltrate and plasma cells are lacking. The opposite occurred in our patients. Erythrophagocytosis by stimulated histiocytes was prominent in our patients but is absent in Letterer-Siwe disease. Our Patient 1 did

not have the skin rash typical of Letterer-Siwe disease. Histiocytic medullary reticulosis[15] differs from familial erythrophagocytic lymphohistiocytosis because it usually occurs in adults and has more malignant features such as cohesive cell masses with cellular atypia, frequent mitoses, and multinucleated giant cells. These features were not present in our cases.

To the best of our knowledge, there have been only two published pathologic descriptions of the ocular findings in familial erythrophagocytic lymphohistiocytosis.[1,2] In both studies erythrophagocytosis was observed. The choroid was involved in Peterson and Kuwabara's case[1]; Appen and associates[2] found only retinal involvement. Both specimens disclosed meningeal involvement with lymphocytic infiltration. Our ocular specimen did not show this involvement, but the pathologic examination of the brain did. In both the previous cases, papilledema was present; in our Case 1, papilledema was found only by the histopathologic studies. The anterior uveal tract in our Patient 1 was infiltrated by histiocytes and lymphocytes, whereas this is a distinctly unusual finding in Letterer-Siwe disease.[16]

These unusual manifestations of the lymphoproliferative disorders probably should not be classified as part of the histiocytosis X group but as familial erythrophagic lymphohistiocytosis. The clinical and pathologic pattern in Case 2 differed from those in Case 1, and were perhaps more consistent with X-chromosome-linked reticuloendotheliosis.[13] However, an infection may have been the triggering event in both cases, and perhaps these syndromes are closely related.

The histiocytic proliferative response to a known agent, such as a virus, may be appropriate; however, inherited or poorly understood malfunctions of the immune system may make this proliferation uncontrolled and ultimately lead to death. X-chromosome-linked recessive lymphoproliferative syndromes with subtle immunodeficiencies to infectious agents (rubella, vaccinia, and Epstein-Barr virus) have been reported recently.[17] Bar and associates[7] described a 16-year-old adolescent with a rapidly progressive fatal lymphoproliferative disease in which the Epstein-Barr virus was identified. A brother and two male cousins all died of a similar disease. Immunofluorescent studies disclosed replacement of T lymphocytes by atypical B lymphocytes, and loss of mitogen-stimulated lymphocyte transformation. Purtilo and associates[18] concluded that the Epstein-Barr virus triggered the proliferation of B cells and that B cells continued to proliferate in affected males because of defective immunity. The authors believed that the viral infection caused the depletion of the controlling T cell populations, after which a different viral infection produced B cell transformation and uncontrolled proliferation in the T cell-depleted host.

In our Case 1, the many plasma cells and the normal to slightly increased serum immunoglobulins suggested adequate B cell function, and marked thymic involution and the scarcity of splenic periarteriolar lymphocytes suggested a T cell defect. Because the results of screening studies for T cell function were normal, this defect may be subtle, involving the ability to process certain viral antigens and leading to an uncontrolled proliferation of histiocytes.

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THE LONG-TERM COURSE OF MULTIFOCAL CHOROIDITIS (PRESUMED OCULAR HISTOPLASMOSIS)

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AND

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We studied 40 patients with the presumed ocular histoplasmosis triad for ten to 15 years. Another patient developed the choroidal features of the syndrome in previously normal eyes.

The basic lesion is a nodular choroidal infiltrate that evolves from clinically and angiographically normal retina. It is initially hypofluorescent and has an overlying serous detachment. It evolves through episodes of serous detachment and hyperfluorescence into a choroidal scar with breaks in Bruch's membrane and occasional subretinal neovascularization.

Seven of 42 eyes (16.6%) developed new choroidal infiltrates in choroid and retina that were clinically and angiographically normal more than ten years ago.

After ten years about 50% of eyes with central scars will have visual acuity changes of 3 or more lines because of progressive remodeling and extension of the disciform scar.

Nine of 40 fellow eyes (22.5%) sustained a disciform process during the entire follow-up period.

One patient developed typical choroidal lesions in previously normal fundi. Results of extensive physical and laboratory tests for histoplasmosis and other diseases were normal.

The current concept of the presumed ocular histoplasmosis syndrome is based on comparatively short-term studies of a disease that can last at least three or four decades. Patients with the triad of circumpapillary scarring, peripheral atrophic chorioretinal scars, and a parafoveal subretinal neovascular membrane are usually studied and treated between 20 and 59 years of age. Most clinical studies focus on the dramatic parafoveal disciform process; these studies include average follow-up periods of six to 30 months and focus on the response to photocoagulation.

The evolution of the focal choroidal lesions so characteristic of the disease has been presented in four studies. Krill and associates,[1] in a classic paper, described the basic lesions and documented their evolution in a group of patients mainly observed for one to five years. They did

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not describe the appearance of new lesions in clinically and angiographically normal fundi. This report is the basic and most complete description of this disease. Later studies have substantiated and extended their descriptions but the basic conclusions are still valid.

Schlaegel[2] described the evolution of choroidal lesions and the appearance of new spots in the disk macula area during a nine-month period by color photographs alone. Gass and Wilkinson[3] failed to find new macular choroidal lesions, but their follow-up lasted one to ten years and not all patients had fluorescein angiography.

Lewis, Van Newkirk, and Gass[4] reanalyzed these patients and an additional more recent group by color photography and fluorescein angiography during a one- to 17-year follow-up period. They documented new choroidal scars in 9% of eyes but did not document disappearance or evolutionary changes except for increase in size.

Miller, Stevens, and De Venecia[5] have reported two new macular scars in eyes with presumed ocular histoplasmosis and there are two other reports of new peripheral lesions.[3,6] Two others have reported de novo subretinal neovascularization in such eyes.[7,8]

We studied 40 patients for a minimum follow-up period of ten years, and a maximum of 15 years, all by personal survey of baseline and follow-up stereoscopic color photographs and fluorescein angiography.

We also studied the appearance of the basic choroidal lesion in one patient whose eyes previously were shown to be clinically normal. This survey was designed to answer the following questions:

1. Do the characteristic choroidal scars occur as one event early in the patient's life or as a continuing process during the patient's adult life?
2. What are the mode of onset and clinical characteristics of the early lesions and how do they evolve?
3. Most assessments of visual results in this disease are based on a six- to 12-month follow-up. Is there a significant visual change in these eyes over a ten-year period and does light coagulation affect this?
4. What is the long-term evolution of the eyes with central disciform scars?

Material and Methods

The study included two groups of patients. Group A included 40 patients, 16 women and 24 men between the ages of 20 and 60 years, first seen more than ten years ago, with an average follow-up of 13 years. All patients were seen because of decreased vision caused by parafoveal disciform lesions. Most were seen only occasionally; ten were seen and photographed frequently during this period.

All the patients in Group A had the triad typical of this syndrome; all had had excellent quality color photographs and fluorescein angiograms taken at the initial examination more than ten years ago. These photographs were compared with recent color photographs and angiograms taken at the end of the follow-up period. Extensive posterior pole scarring or central cataract precluded recent examination of the symptomatic eyes of three patients.

Twenty-six eyes were treated with xenon photocoagulation to the paracentral disciform lesion and 24 eyes were untreated.

Five of the patients also had color photographs and fluorescein angiograms of the fellow eye available for comparison.

Thus, a total of 42 eyes in 40 patients were available for an assessment of the long-term evolution of presumed ocular histoplasmosis.

Group B consisted of one patient who developed typical choroidal lesions and later a paracentral disciform process in

eyes that had been clinically normal at a previous examination.

Results

Group A—During the ten- to 15-year study period, seven of 42 eyes (16.6%) developed new choroidal lesions in areas that were normal by clinical examination, color photographs, and fluorescein angiograms. No lesion definitely disappeared from the angiogram although one did disappear on color photographs.

Twelve of the 42 eyes demonstrated a distinctive evolution of the basic choroidal lesion from onset to maturity to atrophic scar or disciform fibrovascular proliferation. In two eyes the choroidal lesion underwent periods of serous retinal detachment and spontaneous remission without progression to a subretinal neovascular membrane.

The earliest choroidal lesions developed in clinically and angiographically normal choroid. They varied from lesions so small as to be visible on angiography only as small hypofluorescent spots approximately 100 μ in diameter to visible yellow spots approximately 300 μ in size (Figs. 1 and 2, top left). These involved the choriocapillaris and pigment epithelium. The margins were indistinct and there was probably overlying retinal edema or serous detachment.

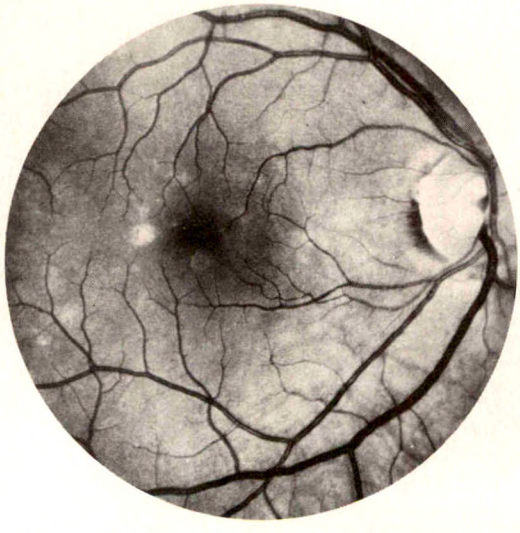


Fig. 1 (Watzke and Claussen). Patient 12, fellow eye. March 1970. The eye is asymptomatic but shows foveal scars.

New lesions were generally not symptomatic but some patients described a relative scotoma corresponding to the lesions, particularly when clustered. Two also noted repetitive light flashes corresponding to the involved retinal areas.

Fluorescein angiography demonstrated that the early lesions were hypofluorescent in the early choroidal and arteriovenous phases. There was faint late staining (Fig. 2, top right and bottom left).

Within four weeks after onset, the choroidal lesion lost its indistinct margin and overlying serous detachment (Fig. 3). It still remained thick and opaque. The best measure of this change was on fluorescein angiography where the lesions became hyperfluorescent in the choroidal and arteriovenous phases without any appearance of a subretinal neovascular membrane or late leakage. Faint staining persisted in late films. Symptoms began to resolve at this time.

In four patients, the choroidal lesions underwent episodes of morphologic change that were self-limited and did not progress to subretinal neovascular membrane formation. The lesions became more opaque, yellow, and prominent. The overlying retina showed a serous detachment (Fig. 4, left). Some lesions were hypofluorescent and others showed early staining and late leakage. Most remarkable was the appearance of new hypofluorescent spots and the tendency in two patients for all of the choroidal lesions in the macula to show this activity simultaneously (Fig. 4, right). Relative scotomas corresponding to these choroidal lesions could be plotted.

These periods of reactivation usually lasted three to four weeks. The choroidal

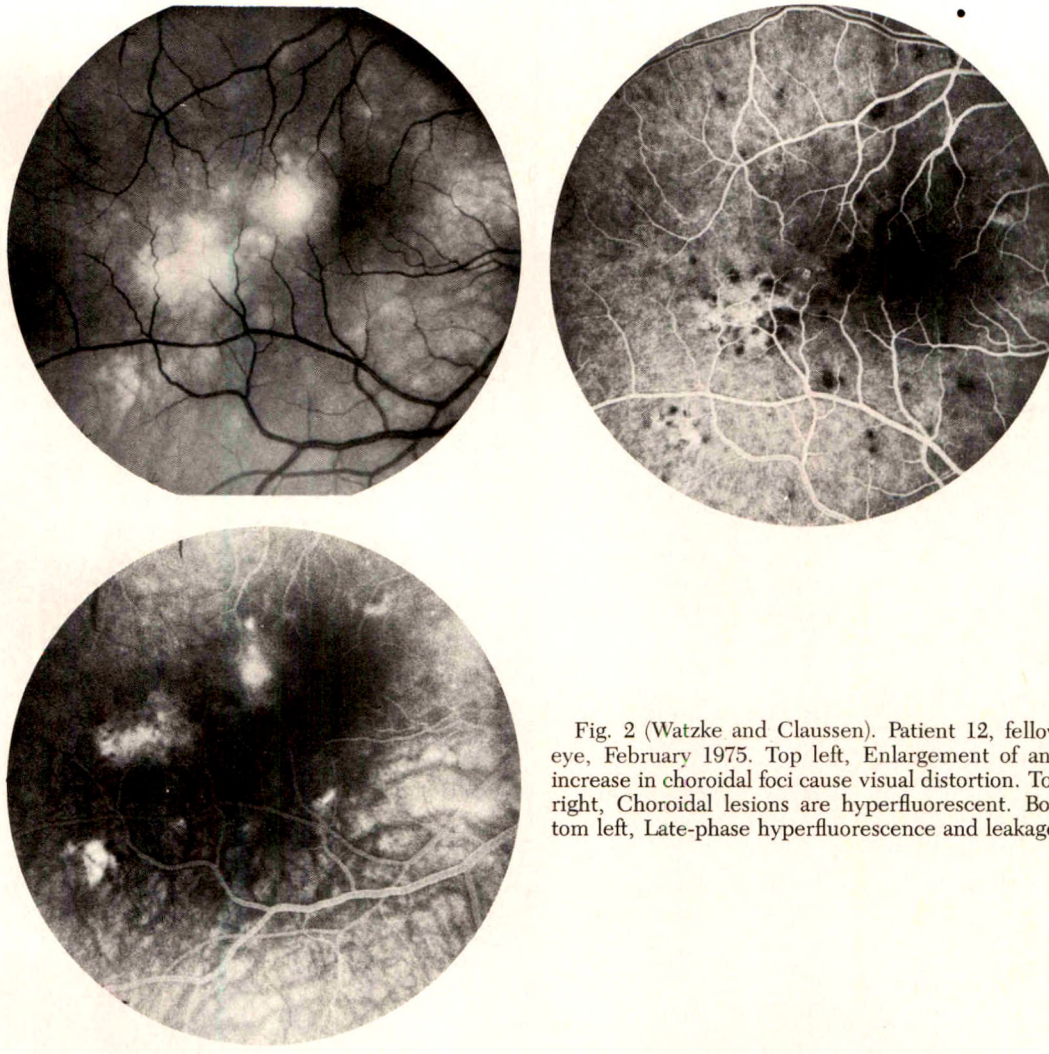


Fig. 2 (Watzke and Claussen). Patient 12, fellow eye, February 1975. Top left, Enlargement of and increase in choroidal foci cause visual distortion. Top right, Choroidal lesions are hyperfluorescent. Bottom left, Late-phase hyperfluorescence and leakage.

lesions then became more distinct, the serous retinal detachment resolved, the angiograms showed only faint staining and no leakage, and symptoms resolved.

Most choroidal lesions eventually evolved into the well-known atrophic scars. There was atrophy of the pigment epithelium and choriocapillaris with migration of pigment toward the center. Parafoveal scars were usually less atrophic and pigmented than in the midperiphery. Scars were usually randomly scattered throughout the fundus but occasionally were arranged in bizarre linear patterns. They became progressively more pigmented.

An occasional choroidal lesion developed a progressively expanding subretinal neovascular membrane. The earliest signs were a faint rim of pigment in the overlying pigment epithelium at the lesion's margins and a persistent overlying serous detachment with early fluorescein staining and late leakage. The late sequelae of progressive hemorrhage and fibrosis are well known.

Most eyes (34 of 49) with central subretinal neovascular membrane formation

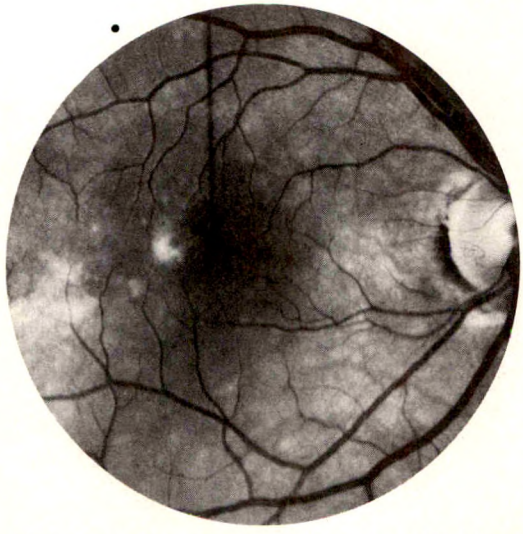


Fig. 3 (Watzke and Claussen). Patient 12, fellow eye, April 1975. Lesions have regressed to asymptomatic scars.

formed a central scar which, during a period of ten to 15 years, evolved into a large hypertrophic elevated scar surrounded by an areola of atrophic pigment epithelium (Fig. 5). The overlying retina usually had a serous detachment with cystoid degeneration.

This process progressed until most of the central chorioretinal scars were included (Fig. 5).

Some eyes developed a crescent or linear gliotic subretinal scar leading from the entrance of the subretinal membrane to the fovea (Fig. 6).

The process was unrelated to photocoagulation treatment.

Three patients developed bilateral central disciform scars; only 43 eyes were available for a ten- to 15-year assessment of visual acuity.

Visual acuity of almost half (21 of 43) the eyes either increased or decreased more than 2 lines on the Sloan chart[9] during a ten- to 15-year period compared with the six- or 12-month status. More patients' visual acuity worsened during this long follow-up than improved. Photocoagulation therapy did not influence this result (Table 1).

We examined 40 patients in order to determine the incidence of disciform involvement of the second fovea. Nine (22.5%) sustained involvement of the fellow eye by such a process. Patients were at risk continuously during the ten- to 15-year follow-up period (Table 2).

Because fellow eyes were not routinely

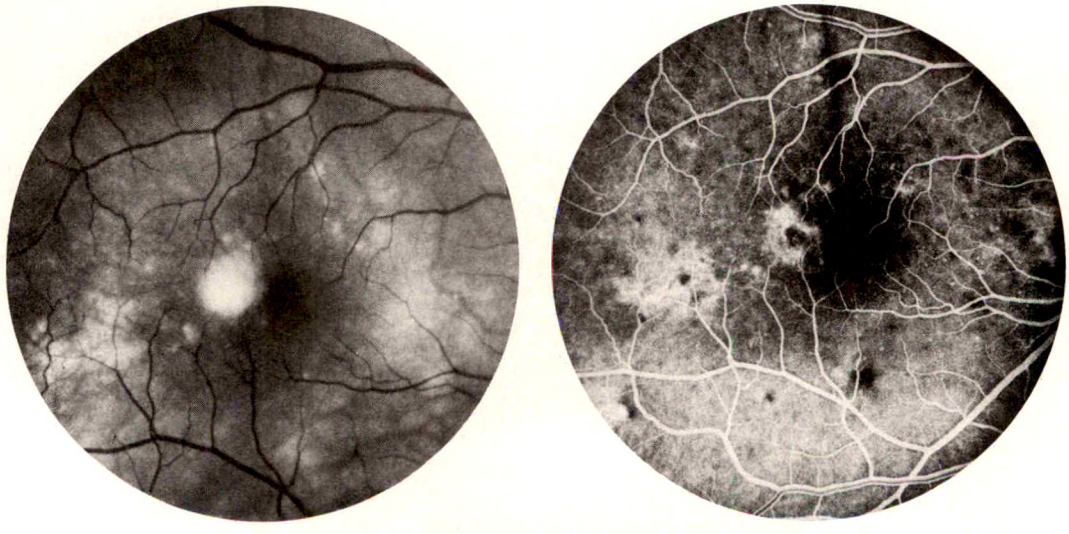


Fig. 4 (Watzke and Claussen). Patient 12, fellow eye, January 1980. Left, Choroidal foci are enlarged and opaque. Right, Hyperfluorescence of old foci and new hypofluorescent spots.

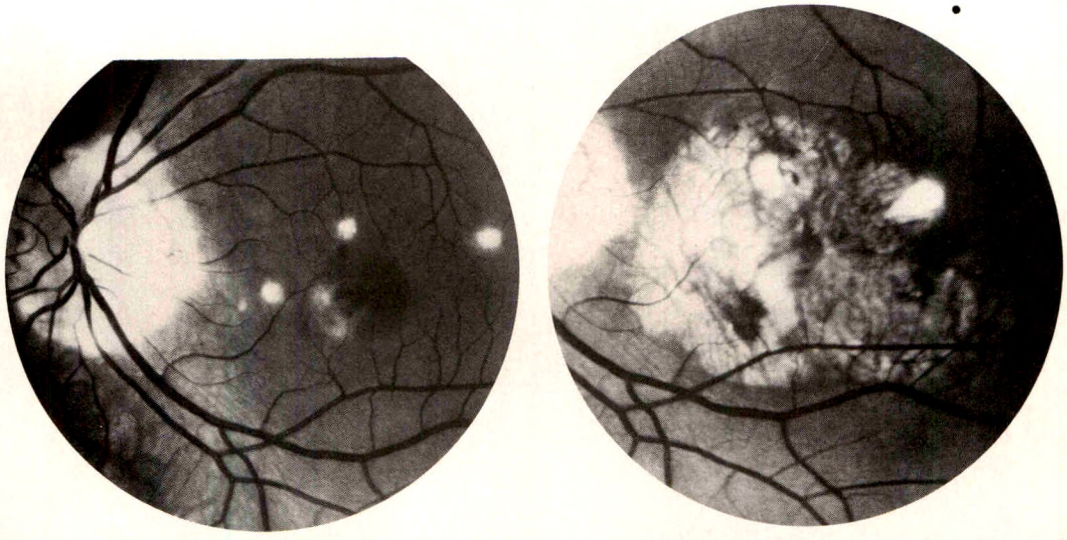


Fig. 5 (Watzke and Claussen). Patient 22, affected eye. Left, March 1968. Central active subretinal neovascular membrane with parafoveal choroidal lesions. Right, December 1978. Disciform scar has enlarged to include all the macular lesions.

photographed, we could not correlate incidence with the presence of parafoveal scars. A 22.5% risk for the fellow eye agrees with other studies.[3,4,10,11]

Group B—This patient developed bilateral choroidal lesions and subretinal neovascular membranes in clinically normal eyes.

Case report

A 31-year-old woman had been observed by one of us (R. W. C.) since she was 12 years old because of axial myopia; results of fundus examinations were normal. In September 1977 she developed an operculated retinal hole inferiorly in the right eye with a cuff of fluid, and received cryotherapy. At that time a complete indirect and direct ophthalmoscopic examination of both eyes again disclosed no other chorioretinal lesions.

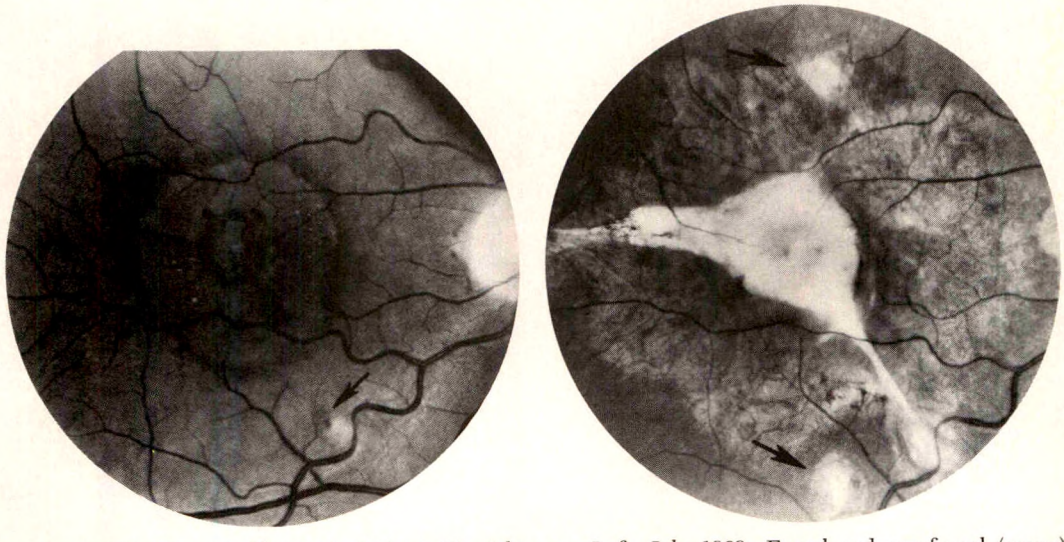


Fig. 6 (Watzke and Claussen). Patient 19, right eye. Left, July 1969. Foveal and parafoveal (arrow) neovascular membranes. Right, July 1978. Central scar with crescent or track joining the parafoveal lesion. Three new choroidal scars have appeared (arrow).

TABLE 1

CHANGE IN VISUAL ACUITY TEN YEARS AFTER CENTRAL SCAR FORMATION (> 2 LINES)

Change	No. of Eyes	Photocoagulation Therapy
None	22	12 Yes
		10 No
Improved	7	4 Yes
		3 No
Worsened	14	6 Yes
		8 No

TABLE 2

INVOLVEMENT OF FELLOW EYE

Patient No.	Time Elapsed Between Involvement of First Eye and Fellow Eye (mos)
28	5
27	17
16	20
10	26
1	36
22	63
19	97
8	108
40	164

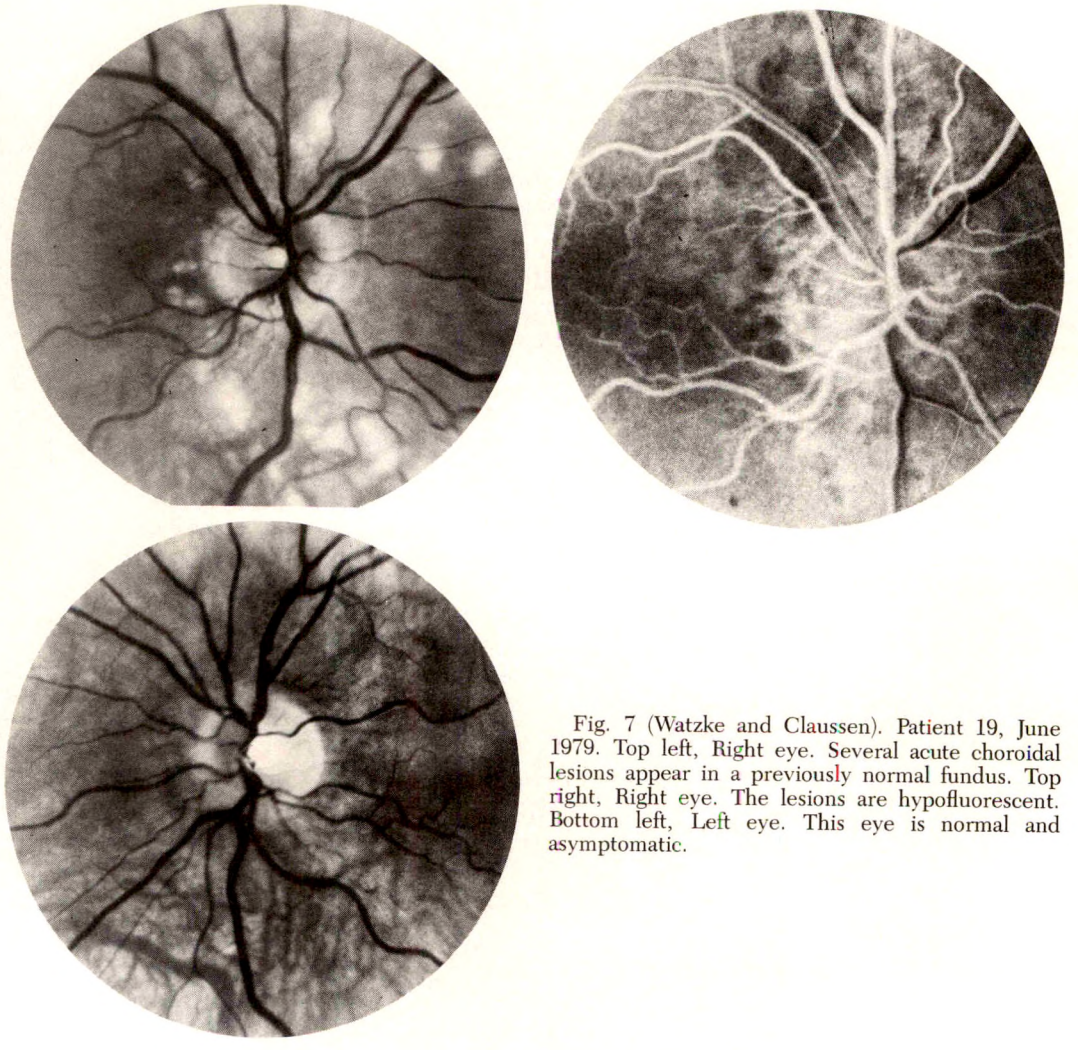


Fig. 7 (Watzke and Claussen). Patient 19, June 1979. Top left, Right eye. Several acute choroidal lesions appear in a previously normal fundus. Top right, Right eye. The lesions are hypofluorescent. Bottom left, Left eye. This eye is normal and asymptomatic.

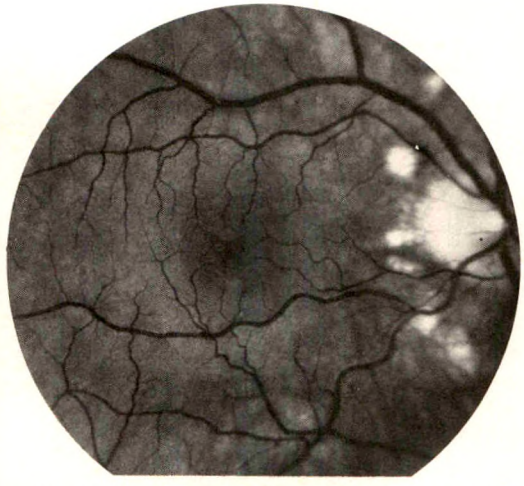


Fig. 8 (Watzke and Claussen). Patient 19, right eye, January 1980. Lesions have evolved into asymptomatic chorioretinal scars.

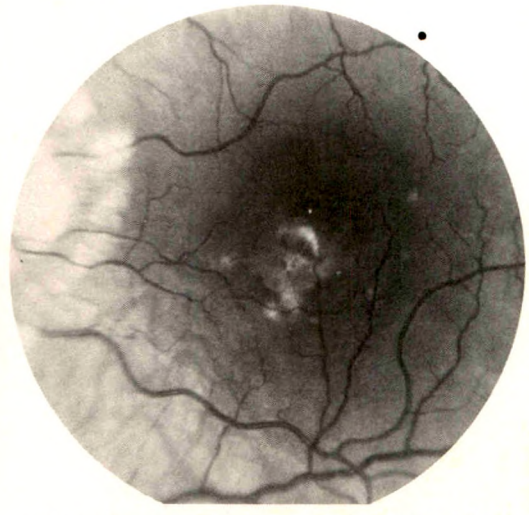


Fig. 9 (Watzke and Claussen). Patient 19, left eye, November 1979. Choroidal lesions and a foveal neovascular membrane have appeared.

On June 7, 1979, she complained of a cloud over the temporal half of the right eye of three days' duration. Visual acuity was 6/7.5 (20/25) in the right eye and 6/6 (20/20) in the left eye. There was no afferent pupillary defect. Visual fields showed a temporal paracentral scotoma. The aqueous and vitreous were clear. The right fundus contained a group of yellow, choroidal infiltrates approximately 300 to 500 μ in size surrounding the disk and in the nasal fundus (Fig. 7, top left). Three of these lesions directly nasal to the disk were pigmented. Some of the lesions were atrophic and depressed; others had a somewhat thickened opaque appearance. They all appeared to be at the level of the pigment epithelium. The optic disk was slightly congested.

On fluorescein angiography of the right eye, the lesions blocked choroidal fluorescence in the early arteriovenous phase with faint late staining (Fig. 7, top right).

There were no choroidal infiltrates, histo spots or disk abnormalities in the left eye (Fig. 7, bottom left).

By July 8, the visual field was unchanged and there were several new lesions superiorly between the vessel arcades. By July 17, the temporal scotoma was fading but there were more new lesions in the right fundus. By Aug. 3, the visual fields and symptoms were much improved, there were no new lesions, and the choroidal lesions were becoming atrophic choroidal scars (Fig. 8, top left).

Visual acuity was stable until Sept. 17, 1979, when she complained of flashing lights in the temporal field of the left eye of one week's duration. Visual acuity was 6/6 (20/20). The fundus now contained similar chorioretinal lesions about and inferior and nasal to the disk. There was one small spot just below the left foveola. The vitreous was clear.

Two weeks later the parafoveal spot had enlarged to about 300 μ in size. By Nov. 20, 1979, a subretinal neovascular membrane had appeared between it and the foveola (Fig. 9). Laser photocoagulation was performed and a paracentral scar ensued; visual acuity was 6/60 (20/200).

On March 13, 1980, the patient noted a relative scotoma in the right eye that corresponded to a new choroidal infiltrate above the right fovea (Fig. 10, left). Examination showed a typical choroidal infiltrate above the right fovea that hyperfluoresced and leaked into a small overlying serous detachment (Fig. 10, right). By March 19, the serous detachment and choroidal infiltrate were larger and appeared more typical of a subretinal neovascular membrane. This also was treated with laser photocoagulation. A stable chorioretinal scar ensued.

Results of physical, neurological, orbital echographic, chest X-ray, electro-oculographic, and electroretinographic examinations were normal. Table 3 summarizes the normal and abnormal laboratory studies.

Discussion

This survey of 40 patients with a minimum follow-up of ten years illustrates that the choroidal infiltrate so characteristic of the presumed ocular histoplasmosis syndrome can appear from clinically and angiographically normal fundi during the patients' adult years. In 16% (7 of 42) of these eyes new choroidal lesions appeared. In only one eye did an infiltrate



Fig. 10 (Watzke and Claussen). Patient 19, right eye, March 1980. Left, A new symptomatic chor has appeared above the fovea. Right, The lesion is hyperfluorescent. Compare appearance here v Figure 7, top right.

"disappear," and the angiographic evidence for this was equivocal.

This study substantiates and amplifies the classic descriptions of Krill and associates[1] concerning the evolution of these choroidal infiltrates. Choroidal foci seen in a very early phase can be described as hypofluorescent when they first appear, before they assume the hyperfluorescent character of more mature lesions and the pigment epithelial defect characteristic of the late atrophic scars. Miller, Stevens, and De Venecia[5] also noted this characteristic of an early choroidal lesion in one patient.

Once a neovascular membrane appeared most eyes formed an extensive hypertrophic macular scar that eventually included all posterior pole foci. This extensive remodeling was progressive and affected the stability of visual acuity for a long period. In almost half of the eyes, visual acuity changed by 3 or more lines during the study.

This study included the largest number of patients followed up for more than ten years. If presumed ocular histo] is a continuing and evolutionary we would expect more new lesions to appear in patients wi follow-ups. Our incidence of 16' er than that of 9% reported by L Newkirk, and Gass[4] and far hig that reported in smaller and sh ries.

We do not know whether lesions that appear from clini angiographically normal choroic foci from some ongoing infectiv or whether they are a cellular to some antigenic foci from a pr posure. Certainly the early cli angiographic appearance of rescence could be explained by infiltrate in the choriocapillaris

Most histopathologic correla ies have concerned older chc scars and have stressed the Bruch's membrane and neova tion seen in such advanced ca seems more useful to study t choroidal lesions before modif

TABLE 3

RESULTS OF LABORATORY STUDIES IN CASE 1

Test	Finding
Complete blood cell count	–
Erythrocyte sedimentation rate	–
Blood constituent levels	
Albumin	–
Calcium	–
Carbon dioxide	–
Chloride	–
Creatinine	–
Glucose	–
Phosphate	–
Potassium	–
Sodium	–
Total protein	–
Urea nitrogen	–
Uric acid	–
Enzymes	
Alkaline phosphatase	–
Lactate dehydrogenase	–
Complement titers	
Total complement	–
C3	–
C4	–
Antinuclear antibody-SC	–
C-reactive protein	–
Immunoglobulin levels	–
Serum protein electrophoresis	–
VDRL test	–
Tuberculin skin test*	+
Antibody tests	
Candida, serum coating	–
Chlamydial group	–
Rheumatoid	–
Toxoplasmosis	+ (1:64)
Trichina	–
Buffy coat examination for fungal and inclusion bodies	–
ELISA test	–
Acute and convalescent histoplasmin, yeast and mycelial titers	– (<1:8)
Blastomycosis and coccidiomycosis titers	– (<1:8)

*First purified protein derivative test.

atrophy, defects in Bruch's membrane, neovascular invasion, and disciform changes. Two studies of such lesions have reported foci of lymphocytes, plasma, and epithelioid cells without organisms. [14,15] Indirect immunohistopathologic staining was positive at these sites when antihistoplasma sera were used. However, the amount of tissue was insufficient for testing with other immunologic reagents. [14]

This disease occurs in patients with variable evidence of previous exposure to histoplasmosis but also in areas where histoplasmosis is virtually unknown. Exposure to other organisms may also be involved. [16,17]

In 1969, Krill and associates[1] described this syndrome and termed it multifocal inner choroiditis. Multifocal inner choroiditis or presumed ocular histoplasmosis is a continuum. It may represent a continuously evolutionary process originating from several antigenic foci deposited in the inner choroid early in life or a continuous reinfection by some unknown organism. Present evidence supports the first theory. We could learn more about this enigmatic disease if histopathologic and immunopathologic studies were directed to these choroidal foci, even in eyes without the disciform process.

ACKNOWLEDGMENT

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RETINAL PERIVASCULITIS IN PHACOLYTIC GLAUCOMA

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A 63-year-old man with a posteriorly dislocated lens developed phacolytic glaucoma. The lens was removed at vitrectomy and extensive retinal perivascular infiltrates were observed at the time of surgery. Two days later, his intraocular pressure was normal and the retinal perivascular changes had completely disappeared.

Perivasculitis of retinal vessels is not part of the usual description of phacolytic glaucoma.[1,2] I observed extensive retinal perivascular cuffing in an eye with phacolytic glaucoma secondary to a posteriorly dislocated, hypermature lens. Within 48 hours of lens removal, the retinal vasculature appeared normal.

CASE REPORT

A 63-year-old man complained of severe pain in his left eye of one week's duration. Six years previously he had suffered a blow to the left eye that subluxated the lens and caused peripheral retinal pigmentary changes. A Marcus Gunn pupil on the left was present at that time. Intraocular pressures remained normal upon subsequent examinations. Visual acuity measurements were unreliable because of a concomitant psychiatric conversion reaction. His medical history included alcoholism, hypertension, and a positive purified protein derivative test that had been treated with isoniazid.

When I examined him, his visual acuity was R.E.: 6/6 (20/20) and L.E.: light perception. The right pupil measured 3 mm and the left 7 mm. An afferent pupillary defect was elicited indirectly in the left eye. Slit-lamp examination showed 2+ nuclear sclerosis in the right lens; there was 3+ epithelial edema in the left cornea. The endothelial surface at the corneoscleral limbus of the left eye was covered with floccular white material; gonioscopy showed that the angle was filled with this debris. There were 3+ cells and 2+ flare, but no keratic precipitates. The lens was dislocated into the inferior vitreous cavity, and the surrounding vitreous also contained floccular material. Applanation tonometric pressures were R.E.: 15 mm Hg and L.E.: 65 mm Hg. An ophthalmoscopic examination disclosed a normal fundus in the right eye. No fundus details could be seen in the left eye.

The patient was treated for phacolytic glaucoma with orally administered glycerin and acetazolamide, topical corticosteroids, and atropine. His pain subsided somewhat but the intraocular pressure in his left eye remained increased at 45 mm Hg.

Four days later he consented to surgery and underwent a pars plana vitrectomy for removal of the dislocated lens. Vitreous aspiration samples for cytologic and cultural studies were obtained during the surgery. As the media cleared, a small preexisting perforation of the lens capsule at its posterior pole was seen. Milky lens content was streaming into the surrounding vitreous cavity through the perforation. The lens was hypermature and aspirated easily without fragmentation. An ophthalmoscopic examination showed fluffy white deposits, which appeared globular in some areas, sheathing both the veins and arteries in the inferior posterior pole.

In an attempt to remove this material from the vessels, the surgeon inserted a flute needle into the eye so that the tip was located over the surface of the retina. The perivascular deposits could not be dislodged by this suction. We took photographs after these intraocular maneuvers (Fig. 1).

On the first postoperative day, the patient's intraocular pressure in his left eye was 17 mm Hg. Visual acuity had improved to finger counting at 3 m with a +11.50 lens. The retinal perivascular abnormalities had completely resolved by the second postoperative day and the anterior chamber cleared. Cultures of intraocular fluid for bacteria and fungi showed no growth; cytologic studies disclosed many large macrophages laden with lens material (Fig. 2).

Visual acuity in the left eye was still finger counting two weeks postoperatively. The media were clear with no cells or flare present. An ophthalmoscopic examination disclosed only mild pallor of the optic nerve in the left eye, with no perivascular abnormalities.

DISCUSSION

Posterior segment abnormalities secondary to phacolytic glaucoma have not received much attention. Schofield[3] dis-

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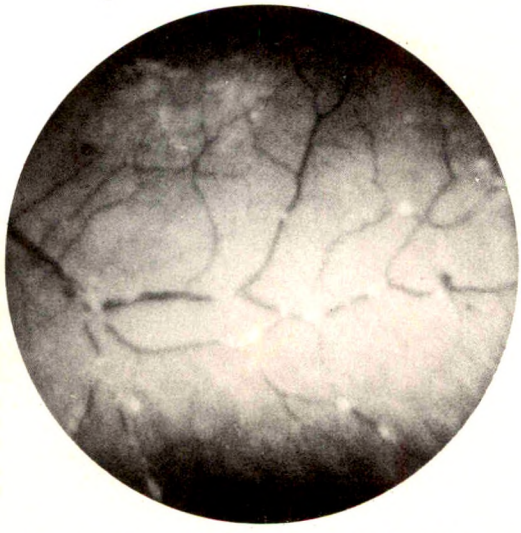


Fig. 1 (Friberg). Left eye. Perivascular inflammation along inferior temporal arcade.

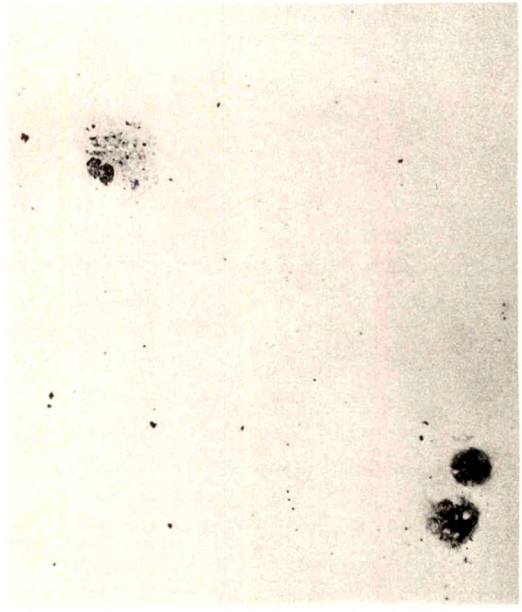


Fig. 2 (Friberg). Vitrectomy aspirate showing macrophages laden with lenticular debris.

cussed the pathologic findings in four eyes diagnosed as having phacolytic glaucoma after enucleation. In three he found macrophages lying on the retinal surface. He suggested that these cells may migrate into the eye from all parts of the globe as a generalized response to free lens material.

Phacolytic glaucoma results from denatured lens protein leaking out of a hypermature cataract with phagocytosis of the debris by macrophages. The lens capsule may be thinned or ruptured, but may be intact.[1] Aqueous drainage is blocked by these cells, thus causing glaucoma. There is no fibrinous exudate in the aqueous and the phagocytes do not adhere to one another. Thus, keratic precipitates and posterior synechiae do not form.[3]

There are two other conditions in which lens material may induce glaucoma. Phacoanaphylactic uveitis is caused by autosensitization to lens protein liberated through a ruptured capsule. Clinically the inflammation may be very severe with mutton-fat keratic precipitates and extensive posterior synechiae. The course is typically long-term. The anterior uvea is infiltrated with eosinophils, polymorphonuclear leukocytes, epithelioid cells, lymphocytes, and giant cells.[4] Phacotoxic uveitis is a less well defined response to the effects of lenticular protein, but it also is characterized by mutton-fat keratic precipitates and a long-term course. Histologically the uvea is infiltrated by plasma cells, lymphocytes, and phagocytes.[5]

The ophthalmic findings in my patient were consistent with phacolytic glaucoma.[1] No keratic precipitates were present despite an abundance of cellular material in the anterior chamber. Cytologic studies of the aspirate at vitrectomy showed typical large macrophages with intracellular lenticular debris (Fig. 2). After lens removal, the intraocular pressure decreased immediately without medication and the anterior chamber quickly cleared.

The unusual feature in this case was the perivascular infiltration found along both arteries and veins, especially inferiorly,

where the lens had dislocated (Fig. 1). It is unlikely that this perivascular material was simply lying on the retinal surface. A flute needle was used during the vitrectomy in an effort to vacuum it from the retinal surface. The perivascular accumulations could not be aspirated. Within 48 hours after lens removal, however, the previously affected retinal vasculature appeared normal.

The posterior segment changes in this patient were probably induced by the proximity of the hypermature lens to the retina, and the relatively long duration of his phacolytic glaucoma. Possibly, these retinal changes occur in other patients with this disease. The media are hazy in the acute stages from the corneal edema and cataract. Additionally, rapid resolution of the perivasculitis after lens extraction may occur, making detection of these retinal abnormalities impossible unless the fundus is examined in the immediate postoperative period.

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DIAGNOSING ACUTE RETROBULBAR NEURITIS BY VITREOUS FLUOROPHOTOMETRY

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We studied six patients with acute retrobulbar neuritis (including two with definite multiple sclerosis) during acute episodes and in convalescence, clinically and with fluorescein angiography and vitreous fluorophotometry. We compared vitreous fluorophotometric readings with visual-evoked responses. In seven of ten eyes with abnormal visual-evoked responses, there was an acute increase in posterior vitreous fluorophotometric readings.

Vitreous fluorophotometry may be useful as objective evidence of (1) acute retrobulbar neuritis, (2) involvement of the asymptomatic eye in acute retrobulbar neuritis, (3) the duration of neuritic inflammation in acute retrobulbar neuritis, (4) a recurrence of acute retrobulbar neuritis, (5) alterations in the blood-ocular barrier in patients with acute retrobulbar neuritis in multiple sclerosis, and (6) the effects of corticosteroids and other therapeutic agents in the treatment of acute retrobulbar neuritis.

McDonald[1] said that, "Perhaps the commonest source of difficulty in the diagnosis of optic neuritis is the presence of a normal disc." Bowden and associates[2] reported that 35% of their patients with optic neuritis had normal-appearing optic disks, and Nikoskelainen[3] indicated that 46% of 185 patients with optic neuritis had normal-appearing optic disks. Because of insufficient clinical evidence, a number of subjective and objective tests are often used to confirm the diagnosis of optic and retrobulbar neuritis. We have used the term retrobulbar neuritis throughout this report to mean optic neuritis without apparent clinical or angiographic evidence of papillary abnormality.

The clinical pattern of acute unilateral impairment of vision, pain on eye movement, central scotoma, color desaturation, and afferent pupillary defect is characteristic of retrobulbar neuritis; since all but one of these components are subjective, objective measurements assume diagnostic importance. The visual-evoked response test, for example, demonstrates impaired latency and amplitude and is helpful diagnostically in optic neu-

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ritis and multiple sclerosis.[4-7] Abnormal visual-evoked responses have disclosed asymptomatic lesions in patients with optic neuritis and multiple sclerosis who have normal fundi and no history of optic neuritis.[5,7-12] Fluorescein angiography has detected minimal permeability defects in optic disk vessels in some instances of retrobulbar neuritis.[13-15] Rosen and Ashworth[16] used fluorescein angiographic densitometry to analyze ten cases of unilateral acute retrobulbar neuritis by comparing the affected with the unaffected fundus. They concluded that the findings on fluorescein angiography were probably abnormal in nine patients, with a suggestion of increased leakage.

We evaluated vitreous fluorophotometric measurements in both eyes of six patients with acute retrobulbar neuritis and correlated the findings with other conventional tests.

SUBJECTS AND METHODS

During the six-month period from August 1979 to January 1980, we examined 12 patients with an optic neuritis syndrome. Of these, we selected six who had acute retrobulbar neuritis without evidence of papillitis or optic disk edema (clinically or angiographically) for this study.

All six patients underwent complete examinations, including best corrected visual acuities for distance and near (at 14 inches with Lebensohn chart). Neuro-ophthalmologic evaluation included assessment of the pupils (particularly to detect an afferent pupillary defect), motility, nystagmus, color vision comparison between the eyes (red and green bottle tops or Hardy-Rand-Rittler plates, or both), black-on-white Amsler grid, and Goldmann visual fields of both eyes. All patients underwent slit-lamp biomicroscopy, applanation tonometry, and ophthalmoscopy with scleral depression. Optic nerve assessment was done by two of us (L. S. B. and M. F.). The patients were examined again during convalescence.

Fundus photography, fluorescein angiography, and vitreous fluorophotometry were done at the first examination and after recovery. We defined recovery as a visual acuity of 6/6 (20/20) (or 4-point vision for near) without Amsler grid defects or a visual acuity worse than 6/6 (20/20) (or less than 4-point vision for near) if it was stable for at least two weeks.

Neurologic studies included a complete physical examination, cerebrospinal fluid studies, and exclusion of other diseases by appropriate investigation, including computerized tomography with orbital views and contrast infusion.

All visual-evoked response tests (except in Case 3) were performed by the same examiner (J. R. H.). The photic lamp (stimulus intensity of 8 relative units) was placed 10 cm from the eyes; flashes appeared every 1.5 seconds, and 128 responses were analyzed by computer. Readings were taken of each eye separately in both occipital regions and analyzed according to the designation of five distinct waves of Kooi and Bagchi.[17] The visual-evoked response test in Case 3 was performed elsewhere with 4-, 8-, 16-, 32-, and 64-mm reversing checkerboard patterns. Reversing patterns were one per second, and 50 results were averaged by computer.

Each patient received an intravenous injection of 25% sodium fluorescein in a dose of 14 mg/kg of body weight. Fluorescein angiography was performed on the affected eye (or the more seriously affected eye if involvement was bilateral) immediately after the injection, and late-phase pictures of both eyes were taken at ten minutes. Vitreous fluorophotometry was performed one hour after the injection. We considered values greater than

TABLE 1

CLINICAL SUMMARIES FOR SIX PATIENTS WITH ACUTE RETROBULBAR NEURITIS

Case No., Sex, Age (yrs)	Days Between Visual Loss and Testing	Best Corrected Visual Acuity			
		Distance		Near	
		R.E.	L.E.	R.E.	L.E.
1, F, 21	50*	6/6 (20/20)	6/6−2 (20/20−2)	J1	J1
	26†	6/60 (20/200)	6/9−1 (20/30−1)	<J14	J2
	68	6/12+1 (20/40+1)	6/12+2 (20/40+2)	J1	J1
2, M, 33	4	6/6−2 (20/20−2)	CF at 60 cm‡	J1	<J14
	48	6/7.5 (20/25)	6/18−1 (20/60−1)	J1	J1
3, F, 32	14-21	6/7.5−2 (20/25−2)	6/6 (20/20)	J1	J1
	89-96	6/6 (20/20)	6/6 (20/20)	J1	J1
4, F, 41	18	6/6−3 (20/20−3)	6/60 (20/200)	J1	<J14
	118	6/6−1 (20/20−1)	C.F. at 30 cm‡	J1	<J14
5, F, 36	27	6/18−2 (20/60−2)	6/6−1 (20/20−1)	J5	J1
6, M, 49	23	6/6 (20/20)	6/6 (20/20)	J1	J1
	197	6/6 (20/20)	6/6 (20/20)	J1	J1

*First episode.
†Second episode.
‡C.F., counting fingers.
§Minimal distortion.

1.0×10^{-8} g/ml in the posterior vitreous (3.5 mm away from the retina) to be abnormal. This figure was based on a background value of $0.7 \pm 0.08 \times 10^{-8}$ g/ml in the posterior vitreous of normal patients described previously.[18-20] The standard deviation of 0.08×10^{-8} g/ml is greater than but similar to the standard deviation of 0.03×10^{-8} g/ml reported by Krupin and associates[21] in 31 control subjects between 21 and 49 years of age. Both fluorescein angiography and vitreous fluorophotometry were repeated after recovery. This procedure enabled us to assess individual variations in those patients with an unaffected eye. We evaluated differences between patients by comparing them with previous standards[18-20] and with four additional patients with various optic nerve diseases. Two of these four had tobacco-alcohol optic neuropathy; one had end-stage glaucoma with marked visual field constriction and a cup-disk ratio of 0:9 bilaterally; and the fourth had hereditary spinocerebellar degeneration with a best corrected visual acuity of R.E.: 6/7.5 (20/25) and L.E.: 6/9 (20/30), 5-point visual acuity for near bilaterally, normal optic disks, and prolonged latency and low amplitude on the visual-evoked response test in the left eye.

RESULTS

We found abnormal vitreous fluorophotometric readings in one or both eyes of all six patients with acute retrobulbar neuritis who had no evidence of optic disk edema by ophthalmoscopy or fluorescein angiography.

All six patients had been examined initially because of decreased or blurred vision in one or both eyes. The pertinent neuro-ophthalmologic findings are shown in Table 1. Bilaterally decreased visual acuity was subjectively noted in Cases 1 (second episode) and 6. In cooperative patients the Amsler grid was

TABLE 1 *(Continued)*

CLINICAL SUMMARIES FOR SIX PATIENTS WITH ACUTE RETROBULBAR NEURITIS

Orbital Pain		Afferent Pupillary Defect		Central Amsler Grid Defect		Color Desaturation		Remarks
R.E.	L.E.	R.E.	L.E.	R.E.	L.E.	R.E.	L.E.	
Absent	Absent	Absent	Absent	Absent	Present	Absent	Present	Definite multiple sclerosis
Present	Absent	Present	Absent	Present	Absent	Absent	Absent	
Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	
Absent	Present	Absent	Present	Absent	Present	Absent	Present	Sickle-cell trait
Absent	Absent	Absent	Absent	Absent	Present	Absent	Absent	
Absent	Absent	Absent	Absent	Present	Absent	Absent	Absent	———
Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	
Absent	Absent	Absent	Present	Absent	Present	Absent	Present	Ocular hypertension
Absent	Absent	Absent	Present	Absent	Present	Absent	Present	
Absent	Absent	Absent	Absent	Present	Absent	Present	Absent	Definite multiple sclerosis
Absent	Absent	Absent	Absent	Present[§]	Present[§]	Absent	Absent	———
Absent	Absent	Absent	Absent	Absent	Absent	Absent	Absent	

the most helpful subjective test in confirming the diagnosis.

All six patients had normal optic disks by ophthalmoscopy and fluorescein angiography, and all six had retrobulbar neuritis. Figure 1 shows a representative late-phase fluorescein angiogram (Case 2) that appeared normal four days after the patient had noted a visual deficit. On that day, the vitreous fluorophotometry reading for his left eye was 2.8×10^{-8} g/ml, the highest in this study (Table 2).

The visual-evoked responses correlated with higher vitreous fluorophotom-

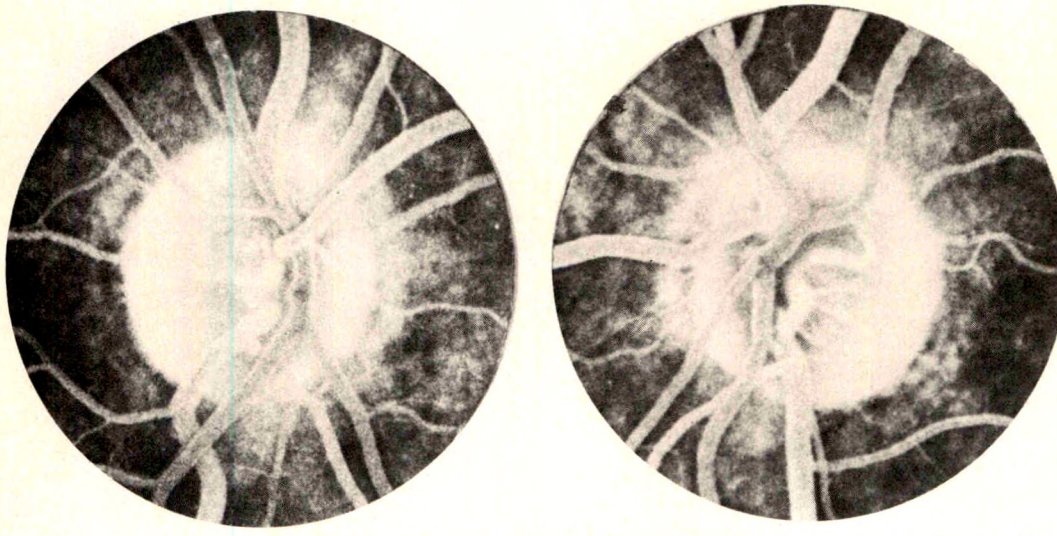


Fig. 1 (Braude and associates). Case 2. Fluorescein angiogram of optic disk ten minutes after injection. There is no leakage or staining. Left, Right eye. Right, Left eye.

TABLE 2

COMPARISON OF VITREOUS FLUOROPHOTOMETRY AND VISUAL-EVOKED RESPONSE IN ACUTE RETROBULBAR NEURITIS

Case No.	Days Between Visual Loss and Testing	Mean Value of Vitreous Fluorophotometry in Posterior Vitreous ($\times 10^{-8}$ g/ml)*		Visual-Evoked Response	
		R.E.	L.E.	R.E.	L.E.
1	50†	0.80	0.85	Long latency of peaks II and III; possible absence of peak V	Normal
	26‡	1.75	0.95	Increased latency of peaks II and III	Increased latency of peak III
	68	0.70	0.65	Not done	Not done
2	4	1.20	2.80	Normal	Lowered amplitude, peaks II and III; peak III delayed
	12	0.90	1.20	Not done	Not done
	48	0.85	0.85	Not done	Not done
3	24†	Unknown	Unknown	Normal	Amplitude was low; latency was prolonged
	14-21‡	1.90	1.30	Not done	Not done
	89-96	0.60	0.60	Not done	Not done
4	18	0.50	1.20	Long latency of peak III (thalamocortical abnormality)	Long latency of peaks I, II, and III
	118	0.50	0.75	Not done	Not done
5	27	1.40	1.00	Long latency of all peaks	Long latency of peaks II and III
6	23	1.30	0.90	Normal	Normal
	197	0.90	0.70	Not done	Not done

*Normal value, $<1.0 \times 10^{-8}$ g/ml $\pm$ 0.08×10^{-8} g/ml.
†First episode.
‡Second episode.

etry measurements in seven of ten eyes (five patients) in whom both tests were done at the time of the acute episode (Table 2). The three eyes with no correlation between abnormal vitreous fluorophotometric readings and abnormal visual-evoked responses occurred in eyes with visual acuities of 6/9 (20/30) for distance or J2 for near or better. Although vitreous fluorophotometric measurements were abnormal in the right eyes of Patients 2 and 6, the visual-evoked responses were normal. Subjectively, however, Patient 6 had bilateral central Amsler grid distortion. The visual-evoked response was abnormal and the vitreous fluorophotometric reading was normal in the left eye of Patient 1.

Patient 1 was unusual in that convalescent baseline readings were available after her first attack. During her second attack, she had bilaterally abnormal visual-evoked responses and a positive vitreous fluorophotometric reading in the eye with the more marked visual deficit (Fig. 2 and Table 2).

One patient (Case 3) did not have visual-evoked response tests repeated at the time of her second attack, but had bilaterally positive vitreous fluorophotometric measurements; the higher reading was in the symptomatic eye.

Patients 4 and 5 had abnormal vitreous fluorophotometric measurements in the eyes that also had abnormal visual-evoked responses. (Patient 5 had bilater-

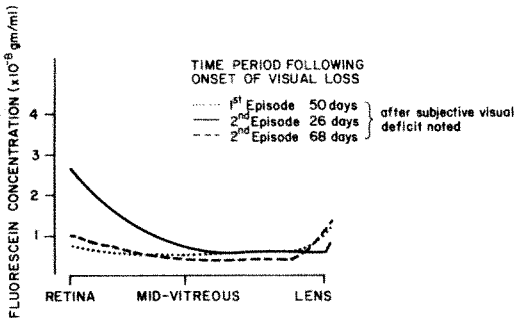


Fig. 2 (Braude and associates). Case 1. Vitreous fluorophotometry of the right eye. Normal value, $<1.0 \times 10^{-8}$ g/ml.

ally abnormal visual-evoked responses and vitreous fluorophotometric findings.)

Computed tomography was performed on all six patients (during the first attack in Cases 1 and 3), but we found no evidence of enlargement or enhancement of the optic nerve image.[22]

Two patients of the six with retrobulbar neuritis also had multiple sclerosis (Table 1).

During the follow-up period, Patient 1 had slight temporal optic disk pallor bilaterally, and Patient 4 had temporal optic disk pallor in the left eye.

All the control patients had vitreous fluorophotometric values within normal limits.

Discussion

The clinician must diagnose acute retrobulbar neuritis without the aid of an observable optic nerve abnormality. The subjective component (complaints of pain, visual acuity testing, Amsler grid, Pulfrich phenomenon,[23-25] brightness comparison, and color desaturation to varying degrees) becomes more important. An afferent pupillary defect, when present, is a helpful objective sign.[26] Laboratory diagnosis assumes greater relevance in documenting the patient's subjective complaints, particularly when hysteria, malingering, or compensation may be present.

Fluorescein angiography is sometimes useful in assessing retrobulbar neuritis.[15,16,27] Younge[27] noted leakage of dye on fluorescein angiography in areas of venous sheathing in patients with acute retrobulbar neuritis, acute papillitis, and multiple sclerosis. Leakage, however, may disappear despite persistence of the sheathing. Our six patients had no evidence of venous sheathing, neovascularization,[28] or fluorescein leakage either on the optic disk or at the retinal vessels.

The presence of sheathing[29-33] is an ophthalmoscopic clue to previous or current breakdown of the blood-ocular barrier. Angiography, however, is subject to difficulties in interpretation; for example, abnormal late-phase staining and increased or prolonged fluorescein leakage are sometimes difficult to differentiate from normal papillary and peripapillary fluorescence.

Rosen and Ashworth[16] used angiographic densitometry to examine ten patients with retrobulbar neuritis. Their study was difficult to appraise because the asymptomatic (allegedly nonaffected) eye was used as the control. Densitometry requires a rigid standardization of photographic technique and no standard time after injection has been shown consistently to be the most effective. Visual-evoked response studies of optic neuritis[7-9] and our study showed that both visual-evoked response (Cases 4 and 5) and vitreous fluorophotometric (Cases 2 and 5) tests can demonstrate abnormalities in the apparently normal eye (Patient 3, second episode, had a positive vitreous fluorophotometric reading in the then asymptomatic eye, but had had a documented attack of retrobulbar neuritis before the study started).

Vitreous fluorophotometry is probably a more accurate and sensitive method than densitometry in determining abnormalities of the blood-ocular barrier. The abnormally high vitreous fluorophotometric findings in all six of our patients showed an increased permeability of the

blood-ocular barrier undetectable by either ophthalmoscopy or fluorescein angiography.

Vitreous fluorophotometry may be useful as objective evidence of (1) acute retrobulbar neuritis, (2) involvement of the asymptomatic eye in acute retrobulbar neuritis, (3) the duration of neuritic inflammation in acute retrobulbar neuritis, (4) a recurrence of acute retrobulbar neuritis, (5) alterations in the blood-ocular barrier in patients with acute retrobulbar neuritis and multiple sclerosis, and (6) the possible effects of corticosteroids and other therapeutic agents in the treatment of acute retrobulbar neuritis.

Objective evidence of acute retrobulbar neuritis—The neuritic or vasculitic component of retrobulbar neuritis that alters the blood-ocular barrier is located in the posterior pole, because vitreous fluorophotometric readings do not increase in the anterior vitreous. We found this acute increase in posterior vitreous fluorophotometric readings in seven of ten eyes with abnormal visual-evoked responses (Table 2).

Patient 6 was particularly interesting because his bilateral central Amsler grid distortion was only confirmed objectively by an increased vitreous fluorophotometric reading in the right eye and not by the visual-evoked response test. The diagnostic value of vitreous fluorophotometry has been demonstrated by the finding that the results of a visual-evoked response test are false-negatives in approximately 5% to 6% of patients with optic neuritis, even when the recording is obtained during the acute attack.[34,35] In two of the ten eyes in which visual-evoked response testing was done during the acute phase of the disease, at the same time as vitreous fluorophotometry, the visual-evoked response was normal but the results of vitreous fluorophotometry were abnormal. Thus, vitreous fluorophotometry may help confirm the diagnosis of acute retrobulbar neuritis, particularly when other objective signs are lacking.

Involvement of the asymptomatic eye in acute retrobulbar neuritis—In Case 5, with symptomatic unilateral visual deficit and bilaterally positive vitreous fluorophotometric readings and visual-evoked responses, the more marked alteration in the visual-evoked response correlated positively with the higher vitreous fluorophotometric readings. In Patient 3, whose visual-evoked response test was not repeated during the second attack, the left eye was also asymptomatic and had a positive vitreous fluorophotometric reading. These findings suggested that vitreous fluorophotometry may prove to be a sensitive indicator of active subclinical disease in asymptomatic eyes of patients with retrobulbar neuritis. The confirmation of subclinical involvement of the asymptomatic eye in retrobulbar neuritis requires comparing the results of vitreous fluorophotometry, visual-evoked response tests, and psychophysical tests, such as perceptual delay.[36] We did not make these comparisons in our study.

Duration of neuritic inflammation in acute retrobulbar neuritis—We obtained positive vitreous fluorophotometric readings from four to 27 days after the subjective onset of visual deficit (Table 2). The highest vitreous fluorophotometric reading recorded (more than 2.5 times the mean normal posterior vitreous value) occurred in Patient 2, who was tested only four days after he noticed the visual deficit (Fig. 3). The asymptomatic eye also had an abnormally increased value on vitreous fluorophotometry at that time. This suggested that vitreous fluorophotometry is probably most helpful when it is performed soon after the visual deficit is noticed.

Vitreous fluorophotometric values returned to normal in five cases. (Patient 5 was lost to follow-up.) A normal value was

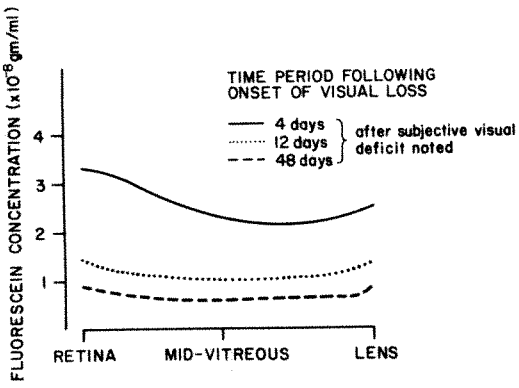


Fig. 3 (Braude and associates). Case 2. Vitreous fluorophotometry of the left eye. Normal value, $<1.0 \times 10^{-8}$ g/ml.

first detected seven weeks after the onset of visual deficit (Patient 2, right eye). However, weekly vitreous fluorophotometric measurements might have shown an earlier return to normal after an acute attack. The return of the vitreous fluorophotometric measurements to normal demonstrated the regenerative capacity of the blood-ocular barrier, even after a second attack (Case 1).

A recurrence of acute retrobulbar neuritis—Because vitreous fluorophotometric findings returned to normal after resolution of the acute attacks (Figs. 2 and 3 and Table 2), this technique may be useful in documenting recurrent attacks of retrobulbar neuritis (as occurred in Case 1), whereas abnormal visual-evoked responses can persist as long as five years, even in the absence of clinical symptoms.[6] Patient 1 had an abnormal visual-evoked response and a normal vitreous fluorophotometric reading in the asymptomatic period after her first documented episode of acute retrobulbar neuritis in the right eye. Halliday, McDonald, and Mushin[10] said, "It appears that once present, the increase in latency is unaffected by the recovery process which leads to restoration of normal amplitude."[10] A recurrent attack, however, may increase the latency in the visual-evoked response (Case 1). Because vitreous fluorophotometric readings are at a normal level once the second attack has subsided), demonstrating recurrences becomes easier. Possibly, however, after many recurrences of retrobulbar neuritis, the vitreous fluorophotometric measurements might be normal even if a recurrence has been demonstrated by other means (false-negative test), or it may remain abnormal (false-positive test). The regenerative capacity of the blood-ocular barrier after many attacks of inflammation needs further research.

Possible alterations in the blood-ocular barrier in patients with acute retrobulbar neuritis and multiple sclerosis—Although the inflammation in retrobulbar neuritis is presumed to be in the retrolaminar portion of the optic nerve, two areas alone or in combination may contribute to the raised permeability of the blood-ocular barrier in the posterior pole. The first is the optic nerve. The second site of alteration in the blood-ocular barrier may be the posterior pole vasculature, even in the absence of venous sheathing. Venous sheathing in multiple sclerosis apparently does not extend nearer to the optic papilla than 2 disk diameters,[32] but the posterior pole vasculature may be involved in the alteration of the blood-ocular barrier and may be less likely to be sheathed than more peripheral vessels. A vascular or perivascular reaction has been shown histologically in multiple sclerosis plaques in other areas of the central nervous system.[37-40] Computed tomography[41-44] and radionuclide brain scans[44,45] have demonstrated increased permeability of the blood-brain barrier. These tests substantiate the relationships between multiple sclerosis plaques and leaky, incompetent veins in the central nervous system.

Possible effects of corticosteroids and other therapeutic agents in the treatment

of acute retrobulbar neuritis—Because corticosteroids appear to stabilize the blood-ocular barrier, patients with acute attacks of retrobulbar neuritis may recover earlier. Bird and associates[46] concluded that intraconally administered triamcinolone produced "a trend toward more rapid recovery of vision in patients with optic neuritis resulting from demyelination," but did not influence the final visual acuity. We agree with Hepler[47] that, "the natural tendency toward spontaneous improvement of optic neuritis makes the effect of treatment difficult to assess." Our Patients 1 (both attacks), 2, 4, and 5 received adrenocorticotropic hormone intravenously or intramuscularly, or corticosteroids orally, or both. We did not detect any significant difference with vitreous fluorophotometry between our treated and untreated patients. Nonetheless, the objectivity of vitreous fluorophotometric measurements allows more valid assessment of therapeutic agents for retrobulbar neuritis than has been possible in the past.

Finally, a word of caution is appropriate with any diagnostic test. An abnormal vitreous fluorophotometric measurement is not specific for retrobulbar neuritis and only indicates an alteration in the blood-ocular barrier. (Similarly, most investigators have been careful to emphasize that an abnormal visual-evoked response is not specific for acute retrobulbar neuritis in patients with multiple sclerosis or acute isolated retrobulbar neuritis, but may be caused by degenerative and compressive lesions of the optic nerve.[48]) This change may be secondary to a variety of situations, and the results, therefore, must be added to the patient's history, clinical findings, and other investigations for proper interpretation. We feel confident, however, that vitreous fluorophotometry has a significant role in the management of the patient with retrobulbar neuritis.

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NARROW-BAND (540-NM) GREEN-LIGHT STEREOSCOPIC PHOTOGRAPHY OF THE SURFACE DETAILS OF THE PERIPAPILLARY RETINA

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We used narrow-band (540-nm) green-light stereoscopic fundus photography to study the surface details of the peripapillar retina in 14 patients. Each photograph was divided into four areas: the papillomacular bundle, the upper arcuate bundle, the lower arcuate bundle, and the nasal sectorial fibers. We scored each area on a scale of 0 (normal) to 3 (total loss of nerve fiber layer), and compared these scores to those obtained by testing the corresponding visual fields. The scores were the same in 88 of 111 comparisons; the retinal scores were higher in 17 comparisons and the visual field scores were higher in six comparisons.

This photographic technique makes the nerve fiber layer easier to see and evaluate and the detection of defects becomes less difficult.

Visualization of defects in the nerve fiber layer has been the concern of a number of papers in the past few years.[1-13] Ducrey, Delori, and Gragoudas[14] pointed out that,

> . . . restriction of the spectral content of the illumination to an appropriate narrow spectral band or the use of quasi-monochromatic light improves both contrast and optical quality over that obtained with white light and results in enhanced visualization of structures.

They found that 540 nm was the best wavelength for enhancing the details of the nerve fiber layer.

We believed that combining the restricted spectrum technique with stereoscopic photography would improve the resolution of details further. Our purpose was to determine the value of this technique by correlating the appearance of the nerve fiber layer, viewed in 14 cases by an unbiased observer, with the corresponding perimetric findings.

SUBJECTS AND METHODS

Instruments—We modified a Zeiss fundus camera with an Allen stereoseparator by adding a new recoss disk equipped with six narrow-band interference filters. We used only the green filter (peak filter transmission, 540 nm; one-half bandwidth, 50 nm). One of us (R. S. M.) performed the visual field examinations with the Goldmann perimeter.

Subjects—We selected 14 patients who had been referred here to serve as subjects. They ranged in age from 15 to 69 years. Seven were men and seven were women (Table 1).

Method—One of us (N. S.) analyzed the positive transparencies of the monochromatic slides without any knowledge of the patient's condition. Each fundus photograph was divided into four peripapillar areas: the papillomacular bundle, the upper arcuate bundle, the lower arcuate bundle, and the nasal sectorial fi-

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TABLE 1

DIAGNOSES AND VISUAL FIELD FINDINGS IN 14 PATIENTS

Case, Sex, Age (yrs)	Diagnoses	Visual Field Findings
1, M, 57	Multiple sclerosis	R.E.: nerve fiber bundle defect
2, M, 60	Leber's optic atrophy	Caecocentral scotomas
3, F, 41	Craniopharyngioma	Left upper quadrantanopia
4, M, 27	Acromegaly; deep excavations	Normal
5, F, 43	Pseudotumor cerebri; long-standing papilledema	R.E.: normal. L.E.: increased blind spot; upper temporal defects
6, M, 69	Previous operation for pituitary adenoma; optic atrophy	Both eyes: lower nasal loss. R.E.: relative upper nasal loss
7, F, 26	Normal control	Normal
8, F, 32	Deep-seated tumor on right side; optic tract syndrome	Left-sided hemianopia
9, F, 27	Tumor of 3rd ventricle; optic tract syndrome	Right-sided hemianopia. L.E.: relative central scotoma and relative lower temporal loss
10, M, 33	Diabetes; hypertension; long-standing papilledema	R.E.: nasal defect, increased blind spot. L.E.: inferior nasal loss; relative temporal defects
11, M, 50	Previous operation for pituitary adenoma; optic atrophy	R.E.: temporal defect, greater in upper field; nasal contraction. L.E.: blind
12, F, 15	Normal control; congenital blurring of optic disk margins	Normal
13, M, 45	Optic atrophy, cause unknown	Both eyes: altitudinal lower defects; absolute nasal defects; relative temporal defects
14, F, 22	Multiple sclerosis	Both eyes: relative central scotomas; temporal defects. L.E.: upper nerve fiber bundle defect

bers. The scoring system we used for evaluating the retinal areas and the corresponding visual field areas are shown in Table 2.

RESULTS

We correlated the scores assigned to the four peripapillar areas of the nerve fiber layer in each eye (retinal score) and the scores of the corresponding visual field (visual field score) (Fig. 1). In the 28 eyes studied, there were 111 correlations between retinal scores and the corresponding visual field scores. (In one case the upper arcuate bundle was not visible in the picture and, therefore, the correlation could not be made for this area.) In 88 of the 111 correlations, the retinal scores were the same as the visual field scores. Among these 88, the most frequent correlation was between the normal nerve fiber layer area and the normal visual field (60 evaluations). Next in frequency was the total loss of the nerve

TABLE 2

THE SCORING SYSTEM USED TO COMPARE PHOTOGRAPHS OF RETINAL AREAS AND THE CORRESPONDING VISUAL FIELDS

Score	Retinal Area	Corresponding Visual Field
0	Normal nerve fiber layer	Normal visual field
1	Gaps in nerve fiber layer	Arcuate scotoma
2	Moderate diffuse loss of nerve fiber layer	Relative defect
3	Total loss of nerve fiber layer	Total loss of visual field

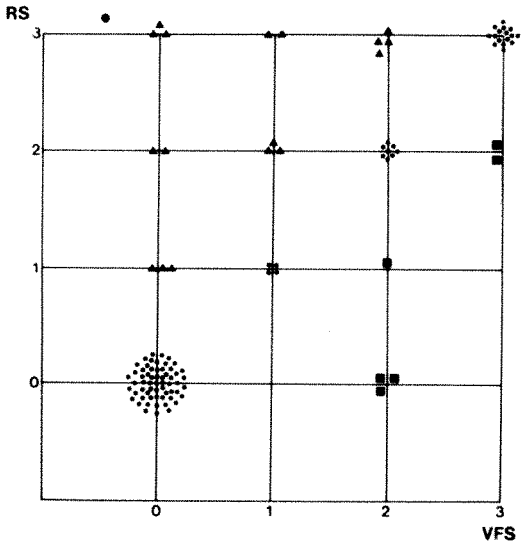


Fig. 1 (Manor and associates). The correlation between the score assigned to each retinal area (RS) by an unbiased observer and the score of the corresponding visual field (VFS). Circle, RS equal to VFS; triangle, RS greater than VFS; square, RS less than VFS.

fiber layer and the total loss of the corresponding visual field (16 evaluations). The instances of exact agreement between the retinal and the visual field scores were almost equally distributed among the four peripapillar areas; there were 22 instances in the papillomacular bundle, 21 in the upper arcuate bundle, 22 in the lower arcuate bundle, and 23 in the nasal sectorial fibers.

In 17 readings (six of them in the papillomacular bundle), the retinal score was greater than the visual field score. In eight of these, the retinal score was between 1 and 3 but there were no detectable defects in the visual field. The retinal score was lower than the visual field score in only six of 110 readings.

Discussion

Data collected by fundus photography with red-free light have indicated that it is sometimes difficult to demonstrate the early diffuse thinning of the nerve fiber layer and the slit defects located outside the upper and lower arcuate bundles. It must be noted, however, that the ability to discriminate between a normal and an abnormal retinal nerve fiber layer depends on the quality of the photographic technique.

Using a green narrow-band filter rather than the red-free filter, which transmits a wide range of wavelengths, increases the contrast and the visibility of the nerve fiber layer. Adding the third dimension, that is, stereopsis, further enhances the nerve fiber layer. Thus, we found that one unbiased observer could evaluate accurately the normal nerve fiber layer and most cases of defective nerve fiber layer.

Hoyt, Frisen, and Newman[6] described the fundoscopic appearance of the normal nerve fiber layer in red-free light in detail.

When examining a normal peripapillar nerve fiber layer with a pair of narrow-band (540-nm) stereoscopic slides, we received an impression of retinal thickness as well as a clearer picture of the normal striations. We even observed a bulging forward of the crowded axons in the upper and lower arcuate bundles. This technique also makes visible the continuity of the nerve fiber layer over the border of the optic disk up to the central excavation.

Recognizing a normal nerve fiber layer is important in treating patients at risk for optic nerve atrophy because of slow-growing intracranial tumors or large optic disk excavations and ocular hypertension. For example, a woman with acromegaly (Case 4) who coincidentally also had deep excavations of the optic disks was found to have normal retinal and visual field scores (Fig. 2).

With our technique, the attenuated nerve fiber layer gives an impression of reduced retinal thickness. This impression is accentuated when there is an area

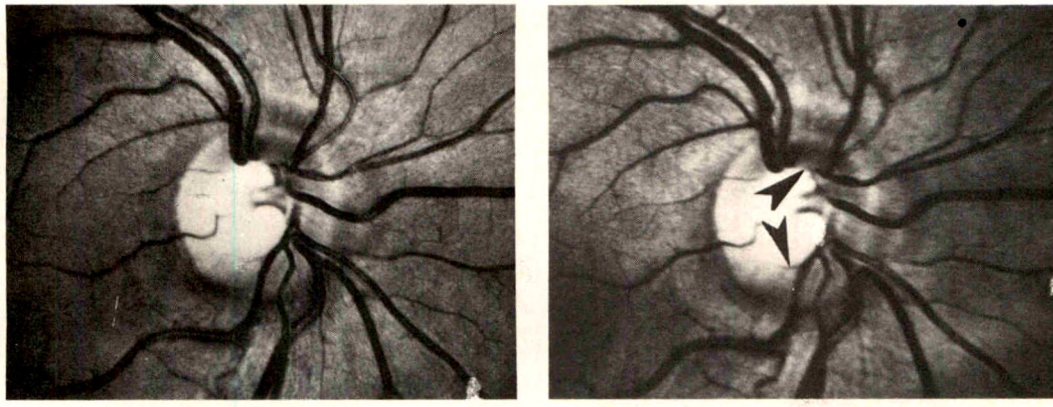


Fig. 2 (Manor and associates). Case 4. Right fundus of a 27-year-old woman with acromegaly and deep optic nerve head excavations. Retinal thickness and nerve fiber layer striations are normal. Note the continuity of the striations on the optic disk up to the excavation area (arrowheads).

of atrophy near normal areas or when the retina is compared with the normal retina of the fellow eye.

In one patient with multiple sclerosis (Case 1), we found a lack of striations and a decrease in retinal thickness, particularly in the upper arcuate bundle. There was a scotoma in the visual field of the lower arcuate bundle. In the atrophic area, the peripapillar retina appeared to be on the same level as the optic disk border, whereas in the lower nasal area the nerve fiber layer appeared to overlay the border of the optic disk (Fig. 3).

Diagnosing pituitary adenoma can be difficult. One of the factors contributing to this difficulty is that the optic disks are frequently almost normal in color, even when there are considerable defects in the visual field and the nerve fiber layer.

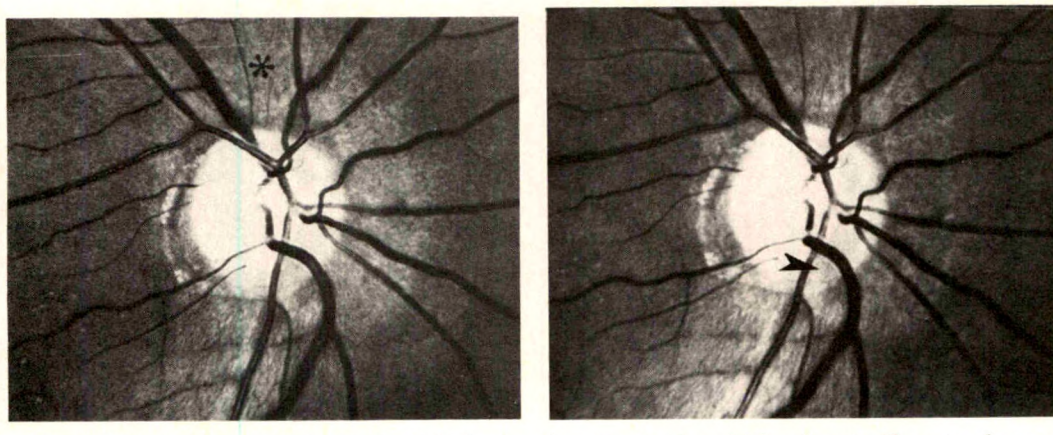


Fig. 3 (Manor and associates). Case 1. Right fundus of a patient with multiple sclerosis. There is a decrease in retinal thickness and striations, particularly in the papillomacular bundle and upper arcuate bundle (asterisk), contrasting with the greater preservation in the lower arcuate bundle area, where there is an evident bulging of the retina and the nerve fiber layer to overlay the border of the optic disk (arrowhead). Note the superior temporal vein ascending from an area devoid of nerve fibers to the area of the optic disk in contrast to the position of the inferior vein, which is held forward by the nerve fiber layer.

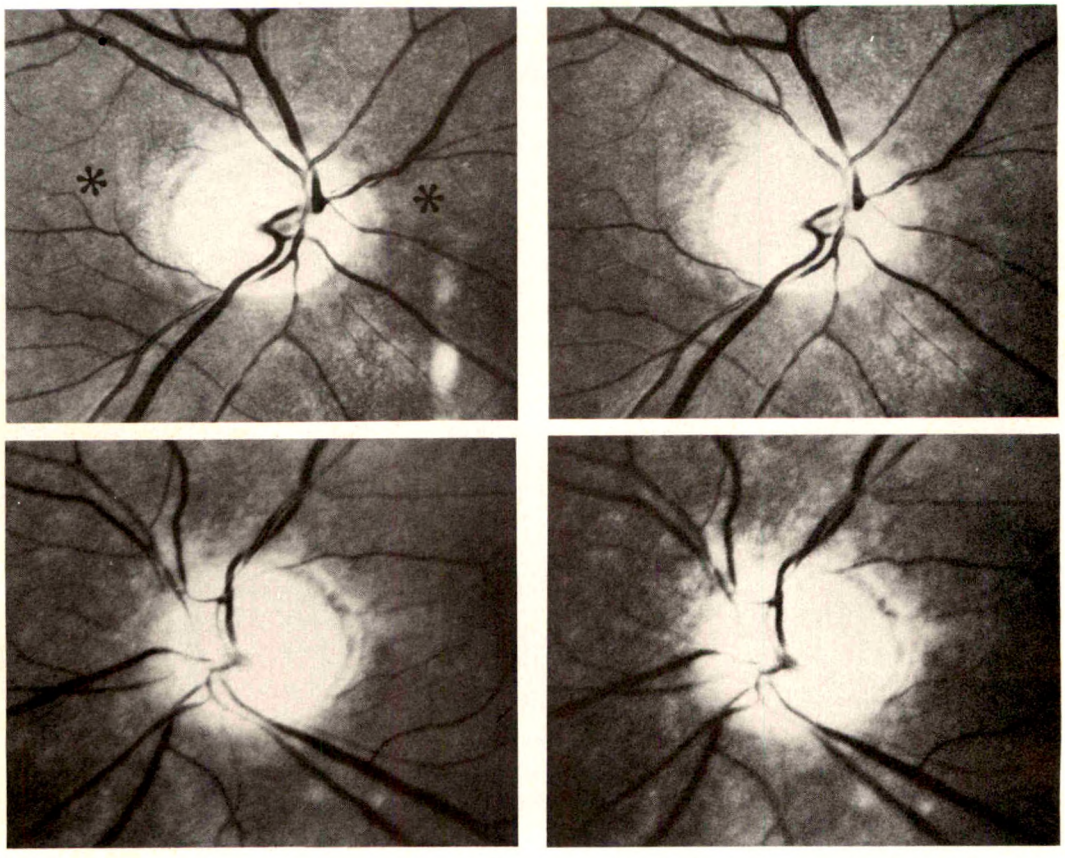


Fig. 4 (Manor and associates). Case 6. Fundi of a patient with pituitary adenoma. Top, The right fundus lacks striations and has lost retinal thickness in a horizontal band nasal and temporal to the optic disk (asterisk), as well as in the lower arcuate bundle. Bottom, The left fundus has a thin retina and lacks striations. Note the absence of the striations that normally encroach upon the optic disks.

The loss of nerve fiber layer, therefore, is an important aid in diagnosis. In one of our patients (Case 6), who had a pituitary adenoma with temporal field loss in the right eye, there were no striations and retinal thickness was lost in a horizontal band nasal and temporal to the optic disk as well as in the lower arcuate bundle. The left eye of this patient was blind and there were no striations or retinal thickness in the peripapillar retina (Fig. 4).

The pregeniculate origin of a right-sided hemianopia was shown by the stereoscopic slides in a patient with left optic tract syndrome (Case 9). The normal striations, retinal thickness, and bulging of the upper and lower arcuate bundles in the right eye were in striking contrast to the thinness and lack of striations of the retina temporal and nasal to the optic disk border in this eye and in the upper and lower arcuate bundles of the left eye. This demonstrated the atrophy of nerve fiber layer in the blind halves of the retina, a finding characteristic of optic tract syndrome.[3,4,15] Additionally, the left eye also had defects in the upper nasal fibers and in the papillomacular bundle because of the simultaneous involvement of the left optic nerve by the tumoral process (Fig. 5).

Monochromatic stereoscopic photographs can also be used to demonstrate more easily the thinning of the inner

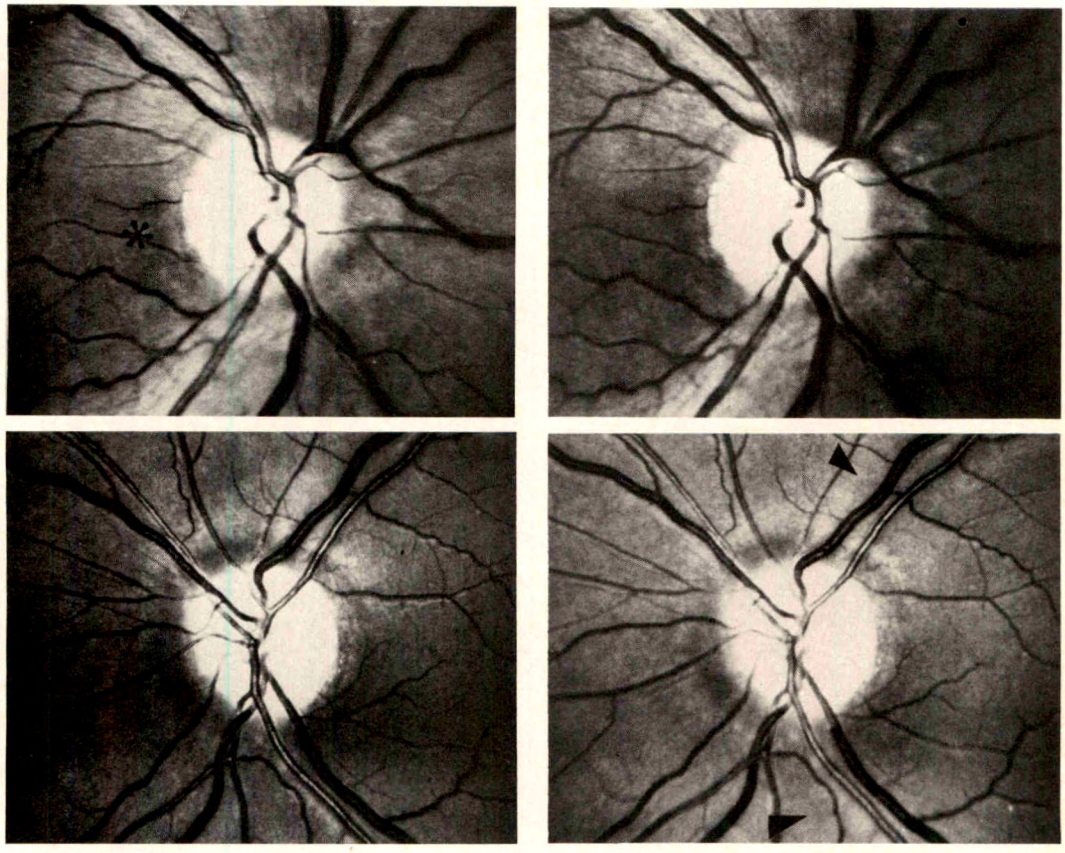


Fig. 5 (Manor and associates). Case 9. Fundi of a patient with left optic tract syndrome and involvement of the left optic nerve. Top, The right fundus has a thin retina and lacks striations temporal and nasal to the optic disk border, corresponding to the lack of temporal field in this eye. This lack of striations is best seen in the papillomacular bundle area (asterisk). Note the bulging of the retina in the normal upper and lower arcuate bundles. Bottom, The left fundus has a thin retina and lacks striations in the upper and lower arcuate bundles (arrowheads), corresponding to the lack of nasal field in this eye. There are also defects in the nasal fibers, particularly in the papillomacular bundle, corresponding to the simultaneous involvement of the left optic nerve.

retina described by Foos[16] as being present after arteriolar occlusion and the small areas of retinal depression considered to be a sign of microinfarcts by Goldbaum.[17]

Obviously, further study of the clinical uses of narrow-band green-light stereoscopic photography is needed. Despite the small number of cases we studied, the use of only one scorer, and the difficulty in scoring retinal and visual field defects, this photographic technique appears to be a promising way of improving the assessment of the nerve fiber layer.

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THE EFFECT OF pH AND OSMOLARITY ON THE ABILITY TO TOLERATE ARTIFICIAL TEARS

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Many patients with keratitis sicca complain that the commercially available artificial tears burn when they are instilled and fail to relieve their distressing symptoms. We examined the effects of altering both the pH and osmolarity of a hydroxypropyl methylcellulose tear substitute on the tolerance of 15 patients with varying degrees of keratitis sicca. Approximately equal numbers of patients selected the neutral hypotonic tear preparations and the isotonic or near-isotonic alkaline tear substitutes. The isotonic alkaline tear preparation was preferred by the majority of our patients with moderate or severe dry eyes. A commercially available alkaline tear substitute is needed.

Keratoconjunctivitis sicca is one of the more common distressing clinical problems facing the ophthalmologist. The symptoms range from mild ocular irritation to severe, disabling ocular pain and visual loss from corneal damage. Although artificial tears are the main therapy for the dry eye, many patients complain that the commercially available tear substitutes burn when instilled and fail to provide relief for their discomfort.

In an effort to provide our patients with a more comfortable artificial tear, we set out to determine empirically what features of an artificial tear were important for improved tolerance by the patient. Having confirmed that an alkaline tear substitute was preferred by the majority of our patients with keratitis sicca,[1] we then examined the effects of varying both the pH and osmolarity of a hydroxypropyl methylcellulose tear substitute.

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SUBJECTS AND METHODS

During an 18-month period, we studied 15 patients (three men and 12 women, ranging in age from 27 to 76 years) with keratitis sicca. All had moderate to severe symptoms of burning, foreign-body sensation, photophobia, or dryness associated with rose bengal staining of the cornea and conjunctiva, diminished height of the precorneal tear film meniscus (less than 0.5 mm), and less than 10 mm of wetting by Schirmer strip testing.

We asked each subject to grade the symptoms of burning, foreign-body sensation, dryness, itching, photophobia, tearing, blurred vision and tired eyes as absent, mild, moderate, or severe. Conjunctival injection, conjunctival chemosis, and rose bengal staining of the cornea, conjunctiva, or both were graded in a similar fashion. The height of the precorneal tear film meniscus was measured with the variable slit of a slit lamp.

All ocular therapy was discontinued 24 hours before the patient entered the study. The trial was divided into three parts and conducted in a randomized, double-masked, cross-over fashion.

The test solutions were a hydroxypropyl methylcellulose tear substitute with a borax-boric acid buffer system.

In part 1 of this study, we examined the patients' ability to tolerate tear substitutes with a pH of 7.4 and osmolarities of 250, 200, and 150 mOsm/liter (Table 1). Each preparation was used for a two-week period. The patients were instructed to instill the drops as often as necessary but no less than four times per day. At the conclusion of each two-week period, we evaluated the patients' symptoms and the slit-lamp findings. The patients were asked which solution they preferred.

Part 2 was conducted in a similar manner and involved comparing the patients' stated preferences from part 1 with an isotonic (300 mOsm) alkaline tear substitute with a pH of 8.4 (Table 1). Alkaline artificial tears with approximately this pH have been well tolerated by patients with dry eyes.[1-3]

In the final phase of this project, we compared the patients' ability to tolerate tear substitutes with a pH of 8.4 and osmolarities of 250, 200, and 150 mOsm/liter (Table 1). The patients were then asked to state their overall preference.

Results

Of the 15 patients, 14 expressed a preference after completing part 1 of this study. Six selected the 250-mOsm solution, four chose the 200-mOsm solution, and four preferred the 150-mOsm solution.

We then correlated these preferences with the clinical severity of the symptoms as determined by the patients' subjective responses. The results are shown in Table 2.

Each patient's preferred osmolarity solution at pH 7.4 was then compared with an isotonic (300 mOsm) alkaline tear substitute with a pH of 8.4. Seven of 14 patients selected the alkaline artificial tear as the most comfortable. Of these, five had originally selected the 250-mOsm solution and two the 200-mOsm solution. Of the seven other patients who chose the neutral (pH 7.4) tear substitute, one selected the 250-mOsm solution, two chose the 200-mOsm solution, and four preferred the 150-mOsm solution. These results and the relationship between clinical severity and osmolarity preference are shown in Table 2. Interestingly, the four patients who had preferred the 150-mOsm solution at pH 7.4 in part 1 made the same choice in part 2.

The final phase of this project compared the patients' ability to tolerate artificial tears with a pH of 8.4 and varying osmolarities. Twelve of 14 patients expressed a preference in this part of the study. Seven of 12 patients selected the 250-mOsm solution, three chose the 200-

TABLE 1

Composition of tear substitutes

Constituents	Osmolarity (mOsm/liter)						
	At pH 7.4			At pH 8.4			
	250	200	150	300	250	200	150
Sodium chloride	4.18 g	3.2 g	2.49 g	4.5 g	3.0 g	2.31 g	1.57 g
Potassium chloride	2.23 g	1.48 g	0.53 g	3.7 g	2.5 g	1.46 g	0.53 g
Borax	0.2 g	0.2 g	0.2 g	2.0 g	2.5 g	2.5 g	2.5 g
Boric acid	3.0 g	3.0 g	3.0 g	2.5 g	2.5 g	2.5 g	2.5 g
Hydroxypropyl methylcellulose	3.0 g	3.0 g	3.0 g	3.0 g	3.0 g	3.0 f	3.0 g
Benzalkonium chloride	0.2 ml	0.2 ml	0.2 ml	0.2 ml	0.2 ml	0.2 ml	0.2
Purified water	Enough to reach a volume of 1,000 ml						

TABLE 2

CLINICAL SEVERITY OF SYMPTOMS AND OSMOLARITY PREFERENCES

Preferred Osmolarity (mOsm/liter)	pH	No. of Patients With Keratitis Sicca		
		Mild	Moderate	Severe
		Part 1 of Study		
150	7.4	2	1	1
200	7.4	1	2	1
250	7.4	1	3	2
		Part 2 of Study		
150	7.4	2	1	1
200	7.4	0	2	0
250	7.4	0	1	0
300	8.4	2	2	3
		Part 3 of Study		
150	8.4	1	1	0
200	8.4	1	0	2
250	8.4	1	4	2

mOsm solution, and two preferred the 150-mOsm solution (Table 2).

DISCUSSION

The search for the ideal artificial tear has led to an emphasis on the development of tear substitutes that mimic the composition of normal tears. Normal tears are iso-osmotic with a mean pH value of 7.0.[4,5] Commercial tear substitutes vary in their polymer content and viscosity, but tend to be isotonic with a pH ranging from neutral to acid (Table 3). Unfortunately, many patients with keratitis sicca complain that these tear preparations burn when they are instilled and tend to aggravate rather than relieve their ocular discomfort.

TABLE 3

OSMOLARITY AND pH OF COMMERCIAL TEAR SUBSTITUTES

Product	pH	Osmolarity
Adsorbotear	7.23	Isotonic
Isopto-Tears 0.5%	7.45	Isotonic
Isopto-Tears 1.0%	7.50	Isotonic
Tears Naturale	6.91	Isotonic
Liquifilm Tears	4.66	Isotonic
Liquifilm Forte	5.41	Isotonic
Lyteers	7.34	Isotonic
Hypotears	7.00	Hypotonic

Jones and Coop[2] were the first to demonstrate the value of an alkaline artificial tear in the treatment of patients with dry eyes. Their preparation was a carboxymethylcellulose tear substitute with a bicarbonate buffer system. A major problem with this product was that the bicarbonate buffer was heat-labile, creating problems with heat sterilization. This led to the development of a hydroxypropyl methylcellulose tear substitute with a borax-boric acid buffer system.[3] This preparation has a pH of 8.5 and is marketed in the United Kingdom under the trade name of Hypromellose.

Unfortunately, a comparable tear substitute is not yet commercially available in North America. This study, based solely on the patients' subjective responses, confirmed the value of an alkaline isotonic tear substitute in treating keratitis sicca, especially in patients with moderately severe complaints.

Hypotonic saline has been used in conjunction with hydrophilic bandage lenses to treat severe dry eyes with excellent

results.[6] We have also successfully used half-normal saline solutions to control the symptoms of dry eye in our patients who wear cosmetic hydrogel contact lenses.

One possible explanation of the success of hypotonic saline in these situations is that patients with keratitis sicca have hypertonic tears.[7] Furthermore, Gilbard and Farris[8] found that four of five patients with severe keratitis sicca preferred a half-normal saline tear substitute to an isotonic neutral tear substitute.[8]

Our results supported the view that a hypotonic tear substitute is a valuable therapeutic alternative in treating dry eyes. The majority of our patients with moderately severe dry eyes, however, preferred the isotonic or near-isotonic alkaline tear substitute to the half-normal saline, pH-neutral artificial tear. Part of this difference may be explained by the different methods used to determine clinical severity. In our series, clinical severity was determined by the patients' subjective responses rather than by rose bengal staining of the cornea and conjunctiva. We have found that objective slit-lamp findings correlate poorly with the severity of the patients' complaints.

It is clear that no one tear preparation is consistently better tolerated. The preferences of the patients may change, probably reflecting the fluctuating nature of keratoconjunctivitis sicca. We have, however, demonstrated the value of a hypotonic neutral artificial tear and an isotonic alkaline tear substitute in treating patients with dry eyes. The isotonic alkaline tear substitute is especially valuable in those patients with moderately severe disease. A hypotonic tear substitute is now commercially available, and there is a need for an isotonic alkaline tear substitute as well.

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THE LACK OF TOXICITY OF INTRAVITREALLY ADMINISTERED TRIAMCINOLONE ACETONIDE

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We gave one eye of each of 21 rabbits an intravitreal injection of 1 mg of triamcinolone acetonide; the other eye received an injection of an equal volume of saline solution as a control. Results of slit-lamp examinations, ophthalmoscopy, intraocular pressure, electroretinography, and light and electron microscopy all remained normal throughout the three-month course of the experiment, demonstrating the lack of ocular toxicity of triamcinolone acetonide in the rabbit.

With recent improvements in vitrectomy instrumentation and techniques, it is now possible to reattach initially a high percentage of retinal detachments with extensive massive periretinal proliferation. Unfortunately, these favorable results in the early postoperative period are not maintained and the ultimate prognosis for reattachment is low.[1] This high redetachment rate postoperatively is probably caused by continued proliferation in residual membranes on the surface of the retina. This has led to the search for a pharmacologic method of inhibiting these processes. Intravitreally injected corticosteroids, including triamcinolone acetonide, are effective in inhibiting proliferation in rabbits induced both by the injection of autologous fibroblasts[2] and by perforating trauma.[3]

While many short- and long-acting corticosteroid combinations have been used periocularly, there are few published reports regarding the potential toxic effects of these medications used intraocularly.[4-7] We wanted to determine the possible ocular toxicity of triamcinolone acetonide after intravitreal injection into the rabbit eye.

MATERIAL AND METHODS

We studied 42 eyes of 21 pigmented rabbits weighing between 2.5 and 3.5 kg each. All eyes underwent baseline examinations including slit-lamp examination, ophthalmoscopy, and fundus photography. We obtained intraocular pressure measurements and electroretinographic recordings on three separate occasions.

Each pupil was then dilated with phenylephrine 10% and the rabbits anesthetized with 2 ml of ketamine (100 mg/ml) injected intramuscularly. We applied one drop of proparacaine HCl 0.5% to each eye as a topical anesthetic. We then chose randomly one eye of each rabbit to receive 1 mg of triamcinolone acetonide suspended in 0.1 ml of balanced saline solution. The fellow eye received only an equal volume of balanced saline solution. The eye was gently proptosed by applying orbital pressure. Under

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observation with indirect ophthalmoscopy, the 27-gauge needle of a tuberculin syringe containing 0.1 ml of the material to be injected was inserted approximately 5 to 6 mm posterior to the corneoscleral limbus in the superotemporal quadrant. The bevel-up needle tip was directed to the posterior vitreous cavity and the material was slowly injected over the area of the visual streak. The needle was then withdrawn and the eye repositioned in the orbit.

We performed slit-lamp examinations, ophthalmoscopy, fundus photography, intraocular pressure measurements, and electroretinography at the same time of day on the third, seventh, and 28th days after the injection in all of the rabbits. Additionally, we repeated each test after three months in ten rabbits. A series of five intraocular pressure measurements, done with an applanation tonometer, of each eye were made and the results averaged.

Bilateral, simultaneous electroretinograms were obtained for all rabbits. We designed a dome apparatus containing a fluorescent pre-adapting light encircling a Grass PS22 Photostimulator fixed to the top of the dome. The unanesthetized rabbit was gently restrained and the pupils dilated with one drop of phenylephrine 10%. Topical anesthesia (proparacaine HCl 0.5%) was administered and a bipolar Burian-Allen contact lens was placed in each eye. The rabbit was pre-adapted for three minutes and then dark-adapted for 11 minutes. A series of three flashes spaced one minute apart produced tracings that were photographed from the oscilloscope screen and then measured. We recorded the average values for the A and B wave of each eye as the scotopic measurements, after which the rabbit was light-adapted for three minutes and photopic recordings made.

After the final examination, the rabbits were killed with an overdose of sodium pentobarbital. One rabbit was killed on day 3, ten rabbits on day 28, and ten rabbits three months after the injection.

The globes were enucleated immediately and placed in cold cacodylate-buffered 2% glutaraldehyde. We removed a corneal button and made an incision through the corneoscleral limbus to allow faster penetration of the fixative into the eye. After a minimum fixation time of 24 hours, the eyes were rinsed in cacodylate-buffered solution and bisected circumferentially 4.5 mm posterior to the corneoscleral limbus. The calottes were examined under a dissecting microscope and photographs were taken. A 3 × 3-mm area of the visual streak was then excised from the calotte and post-fixed in 2% osmium tetraoxide in 0.1 M cacodylate buffer dehydrated in ethanol, and embedded in Epon-Araldite. We stained 1-μ sections with paragon for light microscopy, and 60-nm sections with uranyl acetate and lead citrate for electron microscopy. We used growth curve analysis for statistical significance in all comparisons.

Results

Slit-lamp examinations failed to show any changes in the anterior segment in either the corticosteroid-treated or the control eyes. All lenses remained clear throughout the course of the study.

Triamcinolone acetonide was ophthalmoscopically visible as a white mass that became smaller as time passed. We identified it in all rabbits on days 3 and 7. It was still ophthalmoscopically visible in 12 of 20 eyes after one month. After three months, no corticosteroid was visible in any of the ten treated eyes. There were no ophthalmoscopic changes in the retinas of the treated eyes or the control eyes.

There were no significant differences

in intraocular pressure between the corticosteroid-treated eyes and the control eyes at any time during the course of the study ($P > .5$). Additionally, there were no significant differences in intraocular pressure between the pre-injection measurements and the post-injection measurements of the control eyes at any time during the study ($P = .19$), indicating that the corticosteroid had no systemic effect on intraocular pressure.

There were no significant differences between the control and corticosteroid-treated eyes with respect to the scotopic and photopic B waves ($P > .5$) and the scotopic A waves ($P = .12$) throughout the study. Interestingly, there was a significant elevation of the scotopic and photopic B waves and the scotopic A waves for both groups of eyes in the early post-injection period compared to their pre-injection levels ($P < .05$).

Results of both light and electron microscopy of the retina were normal at all times, and we detected no differences between the the two groups.

Discussion

A single, pure, intravitreal triamcinolone acetonide injection appears to be well tolerated in rabbit eyes. The corticosteroid disappeared in eight of 20 eyes by one month after the injection and in all ten treated eyes after three months.

Cataract formation and increases in intraocular pressure have been described with long-term topical, subconjunctival, and systemic corticosteroids, but these changes did not occur during the three-month period of this study. Possibly, some triamcinolone remained in these eyes for longer periods, although it was not visible clinically, but we think that unlikely.

The electroretingraphic data showing no significant differences between the corticosteroid-treated and control eyes were supported by the absence of ophthalmoscopic changes and the normal findings from light and electron microscopy throughout the course of the study. Interestingly, there was an apparent electroretinographic response to the injection itself, manifested by a transient elevation of the A and B waves in the early post-injection period in both groups.

Few corticosteroid preparations have been evaluated for toxicity when injected into the vitreous cavity. Graham and Peyman[4] found intravitreally administered dexamethasone to be nontoxic to rabbits. Oppelt, White, and Halpert[5] failed to produce changes in aqueous humor formation rate or outflow facility with intravitreally administered hydrocortisone in cats. Phillipots,[6] however, described cataract formation after the injection of methylprednisolone acetate, a depot-corticosteroid, into the anterior chamber of humans. It is important to remember that we used a preparation of pure triamcinolone acetonide in this study and that our data can be applied to to commercial corticosteroid preparations only with caution, because our pilot studies have demonstrated a marked ocular toxicity with some commercial betamethasone preparations. The cause of this toxicity may prove to be the vehicle rather than the corticosteroid itself.[7]

Tano and associates[2,3] have shown that intravitreally injected corticosteroids, including triamcinolone acetonide, inhibit experimental intraocular fibroblast proliferation and subsequent traction retinal detachments in rabbits. This study demonstrated triamcinolene acetonide's apparent lack of any significant toxicity when injected into the vitreous cavity of the rabbit. This experimental reduction in proliferation, coupled with the absence of ocular toxicity, suggested that clinical trials of intravitreally administered triamcinolone acetonide in humans

with massive periretinal proliferation should be considered.

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FIXATION TECHNIQUES FOR SECRETORY COMPONENT IN HUMAN LACRIMAL GLAND AND CONJUNCTIVA

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We compared biopsy specimens of human lacrimal gland and conjunctiva preserved by alcohol-acetic acid with the same tissues preserved by freezing to find secretory component. Secretory component was found in lacrimal gland preserved both by alcohol-acetic acid and by freezing. The structure of the tissue was better preserved with the alcohol-acetic acid fixation. Three of eight conjunctival specimens preserved by alcohol-acetic acid showed weak staining for secretory component in a few superficial epithelial cells. We found no nonspecific staining of the conjunctiva with the control antiserum, rhodamine-labeled goat antibovine serum. Two specimens preserved by freezing were unreadable; four of the remaining six stained nonspecifically with both control antiserum and antisecretory component antiserum. One of the remaining two specimens stained with neither the control nor the experimental antiserum; the second may have had more staining with the experimental antiserum than with the control. We found no evidence for including the conjunctiva in the secretory IgA immune system.

The secretory immune system consists of tissues in which IgA-producing plasma cells are adjacent both to epithelial cells that produce secretory component and to a mucosal surface bathed in fluid rich in secretory IgA. Secretory component and IgA are present in human tears[1]; secretory component is present in the human lacrimal gland.[2] The lacrimal gland has IgA-producing plasma cells in its interstitium, secretory-component-producing epithelial cells in its acini, ducts, and tubules, and secretory IgA in the tears bathing its mucosal surface. (Secretory component apparently helps to transport IgA from the interstitium through the epithelial structures to the tears.[3]) Thus, this gland is generally considered a part of the secretory immune system.

We do not know whether the conjunctiva is part of the same system of mucosal immunity. The substantia propria of the conjunctiva has IgA-producing plasma cells, and the conjunctival surface is bathed in tears containing secretory IgA. If secretory component is produced by the conjunctival epithelium, then the conjunctiva can be considered part of the secretory immune system. If not, it probably should not be considered part of the system. This question is of some interest, because the conjunctiva is more exposed to foreign antigens than is the lacrimal

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gland, and may be capable of local clonal expansion of IgA-producing cells.

We reported that, in human tissue preserved by an alcohol-acetic acid mixture, secretory component was present in the lacrimal gland but not in the conjunctiva.[4] By contrast, Franklin, Prendergast, and Silverstein[5] reported that secretory component was present in both the lacrimal gland and the conjunctival epithelium of rabbit tissue preserved by freezing. The difference in the two sets of results may be related to species differences or to the methods of tissue processing. To determine if alcohol-acetic acid preservation could have prevented us from finding secretory component in conjunctival tissues, we used immunofluorescent techniques to search for secretory component in human lacrimal gland and conjunctiva preserved by alcohol-acetic acid and by freezing.

MATERIAL AND METHODS

Tissues—We preserved lacrimal gland biopsy specimens from five patients, two by alcohol-acetic acid and three by freezing. We obtained conjunctival specimens from eight subjects. Each specimen was divided in two; one half was preserved by alcohol-acetic acid and one half by freezing (Table).

Tissue preparation—We placed the tissue for alcohol-acetic acid processing in a fresh mixture of absolute alcohol and acetic acid in a 19:1 dilution by the method of Wolman and Behar[6] and then processed it as previously reported.[7] We placed the tissue for freezing in OCT embedding media and froze it rapidly in a cryostat. Both sets of tissues were cut in sections 4 μ thick.

Staining techniques—We used the direct immunofluorescent technique.

TABLE

CLINICAL DATA AND STAINING RESULTS

Specimen	Diagnosis; Reason for Biopsy	Staining Results			
		Alcohol-Acetic Acid		Freezing	
		Control*	Experimental†	Control*	Experimental†
		Lacrimal Gland			
1	Pseudotumor of orbit	–	++++	Not done	Not done
2	Dry eye; to rule out Sjögren's syndrome	–	++++	Not done	Not done
3	Dry eye; to rule out Sjögren's syndrome	Not done	Not done	–	++++
4	Dry eye; to rule out Sjögren's syndrome	Not done	Not done	–	++
5	Thyroid exophthalmos	Not done	Not done	–	++++
		Conjunctiva			
1	Cataract, glaucoma	–	–	+++	+++
2	Cataract, uveitis	–	+	Unreadable	Unreadable
3	Cataract	–	–	+++	+++
4	To rule out pemphigoid	–	–	Unreadable	Unreadable
5	Contact lens conjunctivitis	–	+	–	–
6	Chalazion	–	–	–	±
7	Chalazion	–	+	+++	+++
8	Chalazion	–	–	+++	+++

*Control, goat antibovine serum.
†Experimental, goat antihuman secretory component.

Tetramethylrhodamine isothiocyanate-labeled goat antihuman secretory component at a dilution of 1:5 was used to stain the tissues as described previously.[7] For a control, we used rhodamine-conjugated goat antibovine serum to stain the tissue in the same fashion. We examined the tissues under a Leitz microscope fitted with an osmium HBO 100 burner and appropriate exciter filters for rhodamine.

Tissues were photographed with Kodak Ektachrome film (ASA 400) with an exposure time of 30 to 60 seconds. The film was developed at ASA 800.

On Ouchterlony analysis, the goat antihuman secretory component formed two lines against human tears; one formed a pattern of identity with antihuman IgA. This antiserum has demonstrated strong antisecretory activity and weak anti-IgA activity on lacrimal gland tissue preserved by alcohol-acetic acid.[4]

On Ouchterlony analysis, the rhodamine-conjugated goat antibovine serum formed one line with normal human serum. There was no identity between this line and a line formed between normal human serum and goat antihuman IgG.

Results

Lacrimal gland—The two processing techniques gave comparable results in the lacrimal gland (Table). Rhodamine-conjugated goat antibovine serum did not stain tissue preserved by alcohol-acetic acid or by freezing (Figure, a and c). Tetramethylrhodamine isothiocyanate-labeled goat antihuman secretory component stained epithelial cells in the lacrimal gland preserved with both alcohol-acetic acid and freezing (Figure, b and d). As expected, secretory component stained some acini but not all acini. Even within acini, secretory component stained some cells but did not stain some adjacent cells. As Allansmith and Gillette[4] previously reported, secretory component stained the acini less brightly than the tubules. The tissue preserved by alcohol-acetic acid was better preserved than the frozen tissue.

Conjunctiva—In the conjunctiva, the results obtained with alcohol-acetic acid and freezing were not the same (Table). All eight specimens preserved with alcohol-acetic acid could be judged accurately for staining with secretory component, whereas two of the eight specimens preserved by freezing were of inadequate quality to be judged accurately.

We found no staining with rhodamine-conjugated goat antibovine serum in any specimen processed with alcohol-acetic acid (Figure, e and g). In five of these specimens, no staining occurred with tetramethylrhodamine isothiocyanate-labeled goat antihuman secretory component (Figure, f). In three of the specimens, areas of the epithelium stained for human secretory component (Figure, h), but not as strongly as did the lacrimal gland. In these specimens, secretory component stained a small proportion of the conjunctival epithelium, compared with the widespread staining of the lacrimal gland epithelium.

The results with frozen conjunctival tissue were quite different. In four of the six frozen specimens, both the control goat antibovine serum and the goat antihuman secretory component stained the conjunctival epithelium diffusely (Figure, i and j). In these specimens, secretory component staining was no more positive than was staining with the control. The staining in the frozen conjunctival epithelium was diffuse, unlike the localized staining in the three tissues preserved in alcohol-acetic acid.

In one frozen specimen, neither goat antibovine serum nor goat antihuman secretory component stained the conjunctival epithelium. In another, we thought that secretory component possibly stained more than the control, but this could not be definitely determined. This specimen was judged to be equivocal.

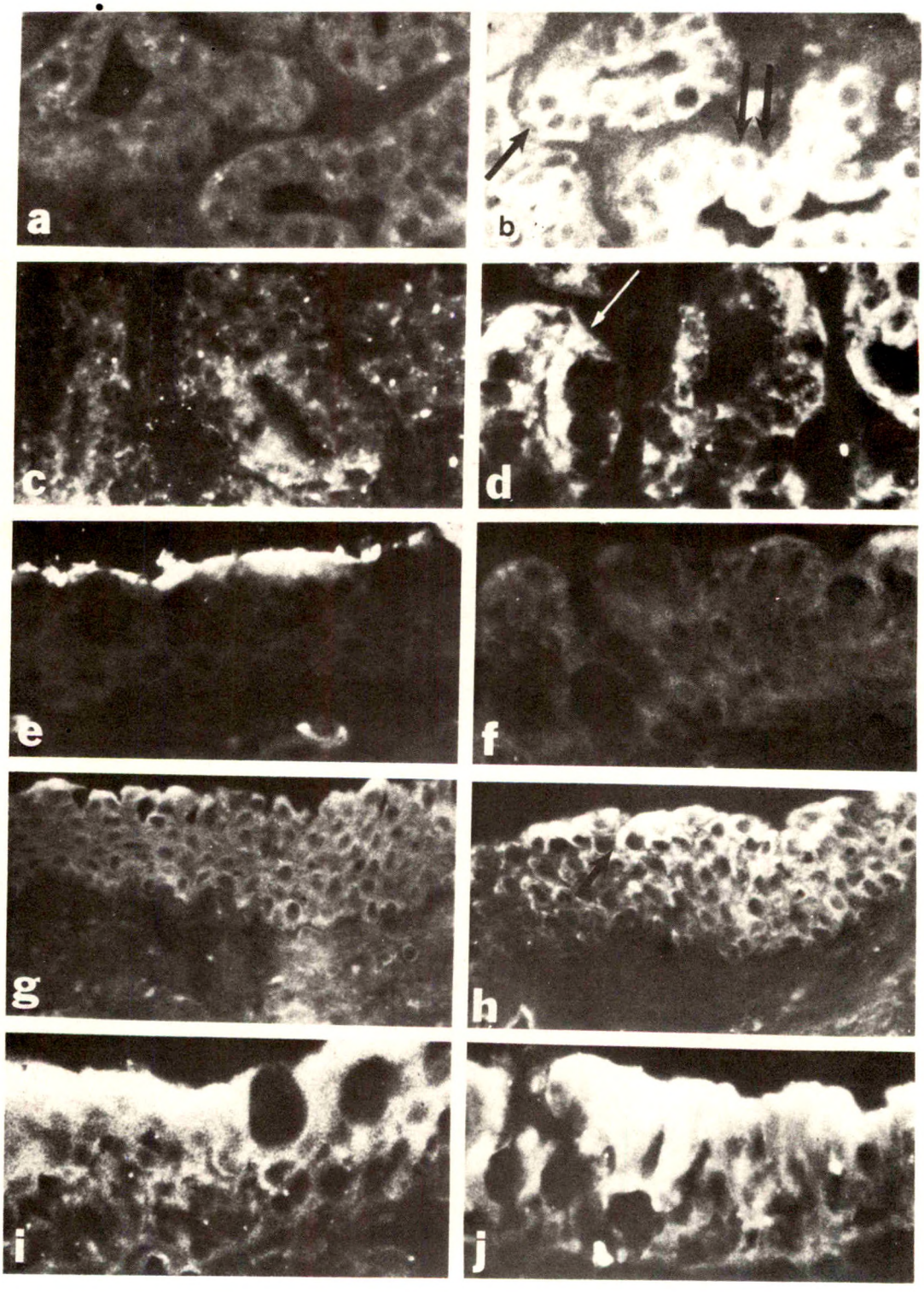

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Figure (Cohen and Allansmith). Sections of human lacrimal gland and conjunctiva. a, Lacrimal gland preserved with alcohol-acetic acid and treated with control antiserum (rhodamine-labeled goat antibovine serum). Note absence of staining. b, Lacrimal gland preserved with alcohol-acetic acid and treated with rhodamine-labeled goat antihuman secretory component. Note staining in acini (single arrow) and small ductule (double arrow). c, Lacrimal gland preserved by freezing and treated with control antiserum. No staining is present. (Lipofuscin granules cause autofluorescence seen in this section.) d, Lacrimal gland preserved by freezing and treated with secretory component. Note staining of acini (arrow). e, Conjunctiva preserved with alcohol-acetic acid and treated with control antiserum. The surface of the epithelium is autofluorescent, but no specific staining is present. f, Conjunctiva preserved with alcohol-acetic acid and treated with secretory component. No specific staining is present. g, Conjunctiva preserved with alcohol-acetic acid and treated with control. No staining is present. h, Conjunctiva preserved with alcohol-acetic acid and treated with secretory component. Specific staining is present in superficial epithelial cells (arrow). i, Conjunctiva preserved by freezing and treated with control. Note diffuse staining. j, Conjunctiva preserved by freezing and treated with secretory component. Note diffuse staining.

Discussion

The findings in lacrimal gland preserved by alcohol-acetic acid and by freezing were comparable. Both stained with goat antihuman secretory component; neither stained with rhodamine-conjugated goat antibovine serum. Thus, in the lacrimal gland, the alcohol-acetic acid fixation method did not destroy secretory component.

In the conjunctiva, the results obtained with the two techniques differed. In three of the eight conjunctival specimens preserved by alcohol-acetic acid, we found areas of staining for secretory component. The staining was weak and limited in extent, compared with that observed in the human lacrimal gland similarly processed. We do not think that the occasional epithelial cell with secretory component is compelling evidence that the conjunctiva produces secretory component. Immunoglobulin has also been seen in epithelial cells,[6] even though it is not produced there. Thus, we have no strong evidence that the conjunctiva is part of the secretory immune system.

In conjunctival tissue preserved by freezing, we observed apparent nonspecific sticking of goat antibovine serum and goat antihuman secretory component to conjunctival epithelium. Therefore, we do not believe we obtained specific information from the conjunctival tissue preserved by freezing.

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NOTES, CASES AND INSTRUMENTS

A SIMPLE METHOD OF REPAIRING INADVERTENT FILTERING BLEBS AFTER CATARACT SURGERY

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We have developed a simple, safe, and effective procedure for repairing inadvertent filtering blebs after cataract surgery with several interrupted 7-0 or 8-0 silk sutures placed externally over the bleb and through the wound cleft, and without opening the conjunctiva or the anterior chamber. We have treated more than 20 eyes successfully with this method.

An inadvertent filtering bleb after cataract surgery is a potentially serious complication. Although many blebs clear up spontaneously, a bleb that does not resolve may lead to several major problems: it increases the risk of delayed intraocular infection[1-4]; it may cause problems with fitting a contact lens, especially a soft lens; and it can result in hypotony, a flat anterior chamber, or, rarely, maculopathy.[5]

Persistent blebs may be the result of a wound dehiscence under the conjunctiva, caused by poor wound closure at the time of surgery, postoperative complications, or blunt trauma to the globe after surgery.[6] Most blebs, however, occur after uneventful and well-performed cataract surgery. Maumenee[7] stated that the use of fine, synthetic, nonreactive sutures has contributed to the increased incidence of inadvertent blebs seen after cataract surgery. Corticosteroids have also been implicated in the formation of such blebs.[8] Whatever the initial cause, there is almost always a wound cleft or tract beneath the bleb that connects with the anterior chamber.

Various treatments for inadvertent filtering blebs have been described. These include direct surgical closure,[9] chemical cauterization,[10] electrical coagulation,[11] and cryotherapy.[5,12] We have developed a simple and safe surgical method for closing such blebs that, to the best of our knowledge, has not been described previously. To date there have been no complications from this technique.

MATERIAL AND METHODS

Before surgery, the patient should undergo gonioscopy to determine the exact location of the cleft in the corneoscleral wound. At surgery topical anesthesia is combined with a subconjunctival anesthetic or a retrobulbar injection if necessary. The bleb is then collapsed and dispersed by pressure from a cotton-tipped applicator (Fig. 1, a, b, and c). This allows the surgeon to see the area of the wound dehiscence through the conjunctiva. Next, one or more interrupted 7-0 or 8-0 silk sutures are placed in a radial fashion through the cornea 2 mm anterior to the corneoscleral cleft. They are carried through the wound at about two thirds of the corneal depth, and out through the conjunctival surface about 2 to 3 mm posterior to the cleft (Fig. 1, d and e). The number of sutures needed depends on the size of the wound cleft. The sutures are tied securely and left in place (Fig. 2) for about two weeks unless there is excessive inflammatory reaction.

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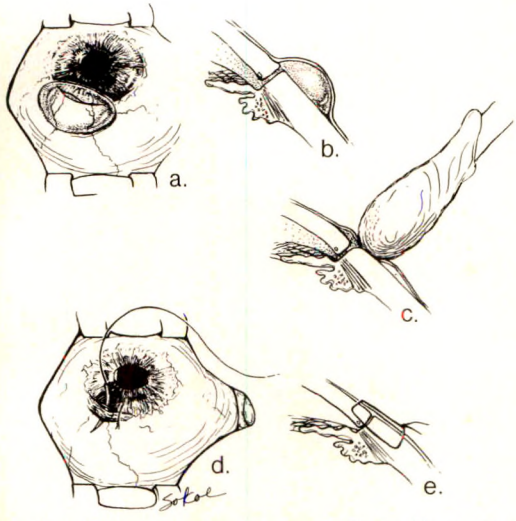


Fig. 1 (Bruner, Maumenee, and Stark). a, Surgeon's view of inadvertent bleb with cleft in corneoscleral wound. b, Cross-section showing bleb and communication with anterior chamber. c, Cotton-tipped applicator used to flatten bleb for better visualization of cleft. d, Placement of silk sutures. e, Cross-section after completion of the procedure.

Results

One of us (A.E.M.) has used this technique to treat more than 20 patients. All the inadvertent blebs were repaired. There have been no clinically significant complications from the procedure and no late recurrences of the bleb.

Discussion

The reported incidence of inadvertent postoperative filtering blebs has ranged from 1% to 7.7%.[6,11] About 70% to 80% of these blebs disappear spontaneously within six months,[6] but serious complications can occur in blebs that persist and are not repaired. There have been several reports of endophthalmitis and visual loss in patients with these blebs.[1-4] Certainly any patient with a clinically significant bleb that persists for more than two or three months and any patient who has foreign-body sensation, ocular irritation, or excessive tearing needs a bleb repair. Also, if persistent leakage of aqueous causes a shallow anterior chamber, hypotony, or macular edema, the bleb should be repaired.

Previous methods of bleb repair have included cryotherapy,[12] electrocautery,[11] chemical cautery,[10] and open suturing of the wound dehiscence.[9] Each of these methods carries a risk of significant complications. Cryotherapy may require several treatments. Cases of sympathetic ophthalmia after chemical cautery of a bleb have been reported.[10] Electrocautery often requires several applications (in some instances, as many as six); and the failure rate is 4% to 5%.[11] Open suturing of the wound dehiscence requires raising a conjunctival flap and exposing the eye to the risks of major intraocular surgery. These risks include the possibility of flat anterior chamber, endophthalmitis, and vitreous loss or lens touch when the patient who has a bleb also has an intraocular lens implant.

Our technique is a closed method, and it does not involve dissecting a conjunctival flap or exposing a wound dehiscence. It is a simple, fast, effective, and safe procedure, and it does not require special instruments.

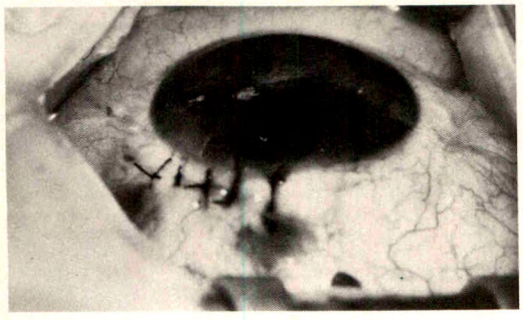


Fig. 2 (Bruner, Maumenee, and Stark). Surgeon's view after completion of the procedure, showing several interrupted silk sutures in place.

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BILATERAL HERPETIC KERATITIS DURING MEASLES

U. SACHS, M.D.,
AND M. MARCUS, M.D.
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A 10-month-old boy with a severe case of measles (including pneumonia, high fever, respiratory distress, and signs of measles encephalopathy) developed bilateral corneal ulcers. Laboratory tests showed that the ulcers were caused by herpes simplex virus. The ulcers healed after mechanical debridement and treatment with topical antibiotics and patching.

Bilateral corneal herpetic ulcers during measles in children may be related to the lowered immune response during infection with measles.

CASE REPORT

A 10-month-old boy was hospitalized with fever, rash, and an upper respiratory infection of three days' duration. He was the youngest of eight children, had no record of previous illnesses, and had received polio vaccine and triple vaccine injections. Measles was diagnosed.

Two weeks after admission the child could not open his eyes because of severe photophobia. An examination disclosed a corneal ulcer in his right eye. Two days later a corneal ulcer was also found in his left eye. Topical treatment with chloramphenicol drops and ointment and systemic vitamin A was begun, but there was no improvement. On the third day, a slit-lamp examination disclosed widespread dendritic corneal ulcers with underlying infiltration of the superficial layers of the stroma in both eyes. Treatment with idoxuridine drops every hour, homatropine 1% twice daily, and tetracycline and idoxuridine ointments at bedtime was instituted. Ten days later there was no change in the shape of the corneal ulcers or the corneal infiltration.

Mechanical debridement of both corneas was performed while the child was under general anesthesia. After debridement both eyes were patched and treated topically with homatropine 1% drops and chloramphenicol ointment. Within three days the corneal ulcer of the right eye had healed, leaving a slight corneal haze. The corneal epithelium in the left eye had to be scraped again a week later, and then healed with topical antibiotics and patching. By that time, the measles, which had taken a severe form, including pneumonia with high fever and respiratory distress and clinical signs of measles encephalopathy, had resolved.

After six months with no further treatment, the keratitis had not recurred. A fine nebula was present in the right cornea.

Laboratory techniques—Measles was diagnosed serologically by a drop in specific antibody titers, tested by the hemagglutination inhibition tech-

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nique.[1] An antibody titer of 1:256 in a blood sample taken four weeks after the illness started was followed three weeks later by a drop in antibody titer to 1:64. Serologic tests for rubella by the hemagglutination inhibition technique[2] were negative (1:8). IgG titers to herpes simplex virus in blood samples taken 19 and 25 days after the onset of clinical herpetic infection were 1:40 and 1:80 respectively, by the indirect immunoperoxidase antibody to membrane antigen technique.[3] However, it should be noted that a 1:40 to 1:80 rise in antibody titer is not considered a significant rise diagnostically, and was probably the result of the serum samples being taken so close together. The diagnosis of herpes simplex viral infection was based on viral isolation.

The removed corneal epithelium was cultured on human embryonic fibroblasts and herpes simplex was isolated and identified as type 1.

Discussion

Primary infection with herpes simplex is most common in the age groups between 6 months to 5 years and 16 to 25 years. The cornea is involved in 40% to 66% of cases of ocular infection, whereas bilateral corneal involvement is relatively uncommon (0.5% to 9.5% of cases).[4-7]

In our case, there was no history of herpes simplex infection in our patient or his family. Our patient was at the lower age limit of the high-risk group, and the main feature of his infection was bilateral corneal involvement accompanied by mild conjunctivitis.

We know that there is a punctate keratitis with catarrhal conjunctivitis in many cases of measles.[4] Bacterial conjunctivitis with bilateral corneal ulcers is possible in debilitated children.[4] The possibility of bacterial secondary infection caused by the lowered immune response in measles has long been suspected.[8,9] Recent studies of viral secondary infection caused by the altered immune response found in measles have implicated adenovirus and herpes simplex.[10]

In our case, the child developed an unusually severe bilateral corneal herpetic infection that seemed to be related to his immunologic state. Possibly some of the reported cases of keratitis during measles or in malnourished children were the result of herpes simplex virus infection.

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NEW SCLERAL PLUGS FOR USE DURING VITRECTOMY

Edward K. Isbey III, M.D.,
Dyson Hickingbotham,
and Maurice B. Landers III, M.D.
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We have devised two new scleral plugs for use in vitreous

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surgery when intraocular pressure is not maintained by the original 20-gauge plug as the incision stretches. Modification of the configuration of the heads of the plugs facilitates their identification during surgery, without hindering the surgeon's ability to handle them with scleral plug forceps.

We have devised two new self-retaining scleral incision plugs that expand the capacities of the scleral plugs designed by O'Malley and Tripp[1] for closed-eye intraocular surgery including vitrectomy. These new plugs, with shafts of 19-gauge and 18-gauge diameter, also have different head sizes to facilitate their identification during the surgical procedure.

The plugs vary in dimension (Fig. 1). The original 20-gauge plug has a head 2 mm in diameter. The new 19-gauge plug has a head 2.4 mm in diameter and the new 18-gauge plug has a head 2.8 mm in diameter. The shaft of all three plugs is 3 mm in length (Fig. 2). The diameter of the 20-gauge plug shaft is 0.9 mm; the 19-gauge shaft is 1.1 mm and the 18-gauge shaft 1.3 mm in diameter. They are made of a high-grade (No. 316) stainless steel.

All the plugs fit in the autoclavable silicone plug holder. All three can be grasped and inserted with standard capsule forceps or with scleral plug forceps.

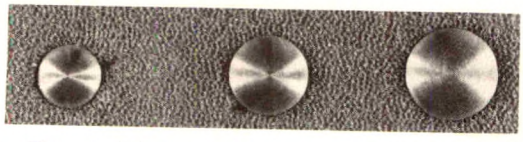


Fig. 1 (Isbey, Hickingbotham, and Landers). Heads of 20-gauge (left), 19-gauge (middle), and 18-gauge (right) scleral plugs. The head of the 20-gauge plug is 2 mm in diameter, that of the 19-gauge plug is 2.4 mm, and that of the 18-gauge plug is 2.8 mm.

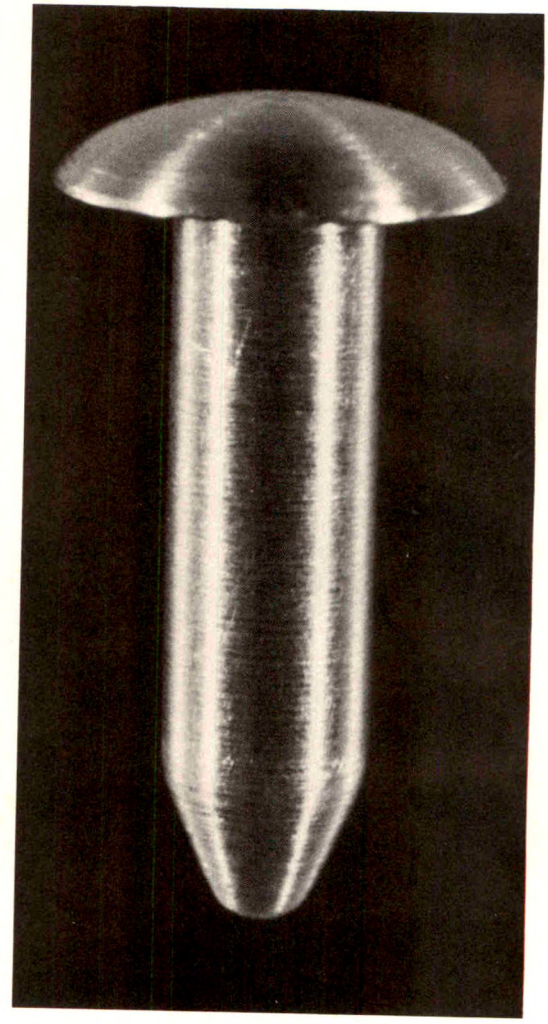


Fig. 2 (Isbey, Hickingbotham, and Landers). Head and shaft of new 19-gauge plug. The shaft has a length of 3 mm and a diameter of 1.1 mm.

The scleral plugs are used during vitrectomy and other types of closed-eye intraocular surgery whenever the incision sites need to be closed temporarily, such as when the eye is to be examined by indirect ophthalmoscopy during a vitrectomy.

In long operations the scleral incisions tend to stretch and widen. The 20-gauge scleral plugs fit loosely into 20-gauge openings, but cannot withstand increased intraocular pressures. One solution to

this problem is a suture at one end of the incision with insertion of the 20-gauge plug. However, this procedure slows the operation and often does not completely solve the problem, because fluid can leak around the suture. The 19-gauge scleral plug can be inserted instead of the 20-gauge plug and will maintain the intraocular pressure. If the original 20-gauge incision has become extremely stretched, it can be firmly plugged with an 18-gauge plug. The 18-gauge plugs can also be used to plug stretched incisions originally made for 19-gauge instruments.

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INCISION SPREADER FOR RADIAL KERATOTOMY

James P. Gills, M.D.,
Dennis L. Williams, M.D.,
Bruce M. Kiskaddon, M.D.,
and Randy Spencer
New Port Richey, Florida

We devised Colibri forceps with reversed points that allow the surgeon to determine the depth of an incision by direct visualization. Although designed for radial keratotomies, the forceps can be used in other procedures as well.

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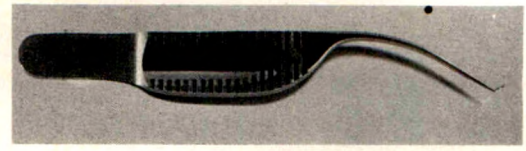


Fig. 1 (Gills and associates). Gills incision spreader (×9).

In radial keratotomy it is essential that the incision extend to the proper depth if the correction obtained is to remain permanent. To ensure that the correct depth is reached, we devised forceps* that allow the surgeon to look into the keratotomy wound and determine the depth of the incision by direct visualization (Fig. 1). These Colibri forceps, which can also be used in other surgical procedures, have reversed points that spread the tissues around the wound apart (Fig. 2).

When the bottom of the incision is in the middle of the stroma, it looks cloudy. As the incision extends deeper, the bottom of the wound becomes glistening and clear, indicating that further cutting is unnecessary.

*Available from Storz Instrument Co., 3365 Tree Court Ind. Blvd., St. Louis, MO 63122 (No. E9015).

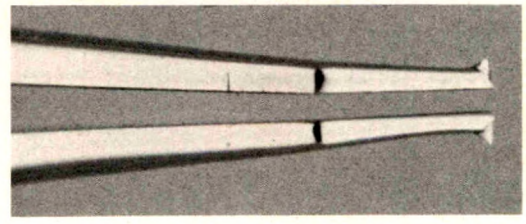


Fig. 2 (Gills and associates). Magnified view of the teeth of the Gills incision spreader (×11.5).

MEETINGS, CONFERENCES, SYMPOSIA

ADVISORY GROUP OF THE WORLD HEALTH ORGANIZATION

The Advisory Group of the World Health Organization's Blindness Prevention Program met in Delhi, India, in February 1981. The group reaffirmed the priorities assigned to eye-care delivery and control of the major preventable and curable causes of blindness in the developing world (blinding infections, malnutrition, filariasis, and cataracts). After three years of attempting to define appropriate strategies, however, the participants concluded that the Blindness Prevention Program was an operational anomaly.

Smallpox eradication has been the prototypical WHO program. It was highly focused and brought enormous resources to bear on a single disease. Its successor, the Expanded Program for Immunization, applies a similar strategy to the prevention of other diseases (diphtheria, tetanus, pertussis, polio, tuberculosis, and measles). Similarly, the Diarrheal Control Program stresses improved water supplies and oral rehydration therapy for the prevention and treatment of all major forms of gastroenteritis.

Prevention of blindness, that is, preventing or reversing a condition (blindness) that has diverse causes, each of which requires its own particular approach, is considerably more complex. Dealing with masses of neglected cataracts requires specialized equipment and surgical skills; onchocerciasis requires coordinated, multinational vector suppression; nutritional blindness requires changes in dietary patterns, and blinding infection requires environmental sanitation and mass treatment.

Prevention of blindness is, therefore, beyond the capabilities of a single program or collection of experts. It can only be achieved through integration into other, ongoing activities. The same improvement in water supplies and sanitation sought for diarrheal control should have a favorable impact on both trachoma and nutritional blindness; expansion of village-level primary health care should provide a vehicle for nutrition education, administration of vitamin A capsules, supervision of antibiotic use, and identification of cataract cases in need of referral; measles immunization will remove one of the commonest precipitating events of xerophthalmia.

From this perspective, the program's most productive venture to date has been the establishment of a worldwide network of collaborating centers. Ten exist (London, Baltimore, San Francisco, Bethesda, Bamaco, Guatemala, Lima, Tokyo, Delhi, and Moscow), with more to follow. Each is a repository of expertise relevant to one or more of the target diseases. They are expected to share this expertise through collaborative training and research. To develop effective intervention strategies, each of these Centers must extend its expertise into relevant public health disciplines, while nonophthalmic institutions competent in public health need to be added to the collaborating network. As a beginning, the Advisory Group endorsed a proposal for a training program leading to a master's degree in public health ophthalmology to be held, alternately, at Johns Hopkins and the University of London.

The challenges and opportunities these new directions raise are likely to dominate the Advisory Group's deliberations in the years to come and to effect a radical shift in its orientation.

ALFRED SOMMER

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THE DILEMMA OF THE MONOCULAR DRIVER

Recent legislative acts at federal and state levels were designed to help the handicapped individual enter the mainstream of everyday life. The Federal Rehabilitation Act of 1977 prohibits discrimination in employment, housing, and education on the basis of handicap, and mandates whatever reasonable assistance individuals need to perform at their chosen occupation. As a result, transportation, social, educational, and business interests have increased their efforts to provide handicapped individuals with a full range of mobility and experience. Conflict occurs when increasing the activities of the handicapped might endanger other individuals. An example of this dilemma is the monocular driver. In May 1980, in the case of Louis ex rel. Welles vs Metropolitan Transit Commission,[1] the Minnesota courts were asked to determine if monocular drivers could function at the most responsible level of driving licensure, driving school buses, emergency vehicles, and commercial passenger vehicles. We, as concerned professionals, are being asked to consider the monocular driver and investigate the scope of the problem.

Investigators have reported that:

1. Gross reduction of the visual field on one side, or monocularity, is associated with a seven-fold increase in intersectional crashes and pedestrian injuries.[2] This is important because 40% of urban crashes occur at intersections.[3]

2. Monocular drivers tend to have injuries on the side of blindness and blindness on the right side is more serious than blindness on the left side.[4] Freytag and Sachs,[5] of the Medical Examiner's office in Baltimore, reported ocular and visual pathway defects in five pedestrians and two drivers killed in Maryland. They demonstrated at autopsy long-standing ocular lesions on the side of the injury. Freytag and Sachs believed that the visual defect contributed to these deaths, and

that visual deficiencies must contribute to many crashes but are obscured by other factors.

3. Liesmaa[6] showed that visually limited drivers have more hazardous driving patterns than others. In his on-the-road study of drivers committing specific driving errors, he matched normally sighted drivers with a control group on the same road. Among 25 drivers entering major roads dangerously, 12% to 48%, depending on the criteria applied, were visually limited and two were blind in one eye.

4. In a Kentucky study,[7] monocularity was four times more frequent among those cited for multiple driving violations than in an ophthalmic office patient population. The incidence of monocularity in general ophthalmic private practice has been calculated at 2% (424 of 21,000 patients).[8] In the Kentucky study of 991 drivers identified for whom medical examinations were recommended on the basis of (1) three or more reportable crashes in 24 months, (b) the driver's statement that he blacked out or could not remember the accident, (c) an obvious medical impairment at the time of license examination, (d) a sworn affidavit by two citizens about the driver's limitation, or (e) a voluntary report by an attending physician, it was found that the incidence of monocularity was 8% (80 of 991 drivers).

Conversely, other investigators[9-11] have stated that there are few research data to support restrictions on drivers based solely on visual limitations, except for the totally blind. Brody[10] said that "numerous statistical studies have found little significant relationship, and often none at all between drivers' visual abilities and their records of safety on the road." Lewis and associates[11] went as far claiming that their study showed that aircraft pilots were significantly more accurate when landing if they used only one eye.

Manus and Less[12] estimated that in some states only one handicapped individual with the potential ability to drive out of four is licensed. Current legislation seeks to reach these three other individuals and encourage them to obtain driving licenses. Driving does extend their experience and provide greater opportunities for freedom and independence. These opportunities must be counterbalanced by personal responsibility: the handicapped driver must know the limitations imposed by his handicap (the monocular driver, for example, should use rapid head turns to lessen the risk of intersectional crashes or of striking a pedestrian on the blind side). We must weigh circumscribed and even conflicting research against sound clinical judgment. "There are statistics that indicate a deaf driver has few accidents and violations, or persons with poor vision have less severe accidents (therefore the blind driver would be the safest driver)."[13]

We must delineate and interpret the visual abnormality limiting the driver, and counsel our patients about their handicap and its effect on driving and other activities. We must report those individuals with handicaps that may affect their driving ability or create added risks for drivers. Physicians do not, and should not, grant, rescind, or limit licenses to drivers. Their critical and professional advice, however, is necessary to sound licensing decisions. Only when the handicapped driver is licensed to function at the highest level that his handicap allows, without added risk of injury to himself or others, will the dilemma be resolved.

After careful consideration it is our opinion that the monocular driver should not be licensed for Class I professional driving such as school buses, public transportation, and emergency vehicles.

ARTHUR H. KEENEY
JAMES GARVEY

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OBITUARY

JEAN NORDMANN

The passing of another great leader has deeply saddened the international community of ophthalmologists and eye researchers. Dr. Jean Nordmann, professor and former chairman of the Department of Ophthalmology at the University of Strasbourg School of Medicine, died on Sept. 23, 1980. He was in his 84th year.

Dr. Nordmann was world-renowned for his contributions to the practice of ophthalmology, his skills as an ocular surgeon, and his imaginative research. His accomplishments were recognized with many awards, including the Grand Gold Medal Cirincione Award of the Italian Society of Ophthalmology, the Cavara Medal of the Ophthalmological Society of Rome, the Colle Award of the French Foundation for Medical Research, and an honory doctorate from Aristotle University in Salonika, Greece. His stature as a leader of ophthalmology is also reflected in the fact that he served as president of many international organizations and chaired numerous meetings and symposia.

The range of Dr. Nordmann's scientific interests was extraordinary. His hundreds of publications include many highly original studies on neuro-ophthalmology, glaucoma, strabismus, and ocular pathology. Throughout his career, however, the research problems that intrigued him the most were the biochemistry of the lens and the nature of cataract. The depth of his interest is evident in his 1926 M.D. thesis on "Contributions to the Study of Cataracts" and his 1946 doctoral dissertation on "Physiology of the Lens." His research on lens biochemistry, published in 1954 under the title "Biologie du Crystallin," was a rich source of information that inspired other investigators to explore the areas opened by his pioneering studies.

Although Dr. Nordmann's scientific and medical accomplishments are well known, I believe that he will be most vividly remembered by those who knew him as a kind, compassionate, and inspiring leader who befriended young physicians and investigators of many nationalities, and one who helped to build the spirit of international scientific cooperation with his personal warmth and charm.

JIN H. KINOSHITA

CORRESPONDENCE

Letters to the Editor must be typed double-spaced on 8½ × 11-inch bond paper, with 1½-inch margins on all four sides, and limited in length to two manuscript pages.

Dangers of Similar Packaging

Editor:

I would like to point out a dangerous similarity in the packaging of a medication frequently used in ophthalmic surgery—hyaluronidase (Wydase)—and that of a potent vasopressor (Wyamine).

This similarity could cause confusion or, more importantly, a serious life-threatening situation if the wrong drug were mixed with a local anesthetic.

The medications are both manufactured by Wyeth and have the logo of the company prominently displayed. The lettering is small and a casual glance might miss the differences (Figure).

One of my patients was almost given a mixture containing Wyamine rather than Wydase before undergoing surgery. Fortunately, the mistake was noticed. Although an injection of Wyamine into the retrobulbar fatty tissue probably would not have been dangerous, an intravenous injection might have caused a life-threatening hypertensive reaction.

Wydase is highly purified bovine testicular hyaluronidase. Wyamine sulfate is a sympathomimetic agent similar to *p*-phenylethylamine and is an indirectly acting sympathomimetic amine that releases norepinephrine. The increase in blood pressure produced by mephentermine is probably the result of an increase in cardiac output.

Unfortunately, look-alike and sound-alike medications are not uncommon. A near-tragic occurrence, caused by similarly confusing packaging, was reported by Duane[1] who pointed out that fluorescite (fluorescein sodium) and fluorouracil are packaged in almost identical fashion and that the ampules are very similar in appearance. Fortunately, a patient who was to undergo a fluorescein angiogram did not receive the 10 ml of fluorouracil intravenously that would have been fatal.



Figure (Rumelt). The two packages resemble each other closely.

The ophthalmic surgeon must be alert to the potentially dangerous situations that may occur in the operating room.

MICHAEL B. RUMELT, M.D.
Creve Couer, Missouri

REFERENCE

1. Duane, T. D.: Fluorescite-fluorouracil. A nearly tragic confusion in identity. Am. J. Ophthalmol. 77:277, 1974.

Acquired Cyclic Esotropia in an Adult Eye

Editor:

There were some points in the article, "Acquired cyclic esotropia in an adult

eye," by B. T. Troost, L. Abel, J. Noreika, and F. M. Genovese (Am. J. Ophthalmol. 91:8, 1981), that I did not understand and whose validity I question. In Figure 2, the middle tracing depicts viewing with the right eye. The right eye starts fixation at zero while the left eye starts in a 10-degree esophoric position. As the right eye moves to the right, there is no significant movement of the left eye. As the right eye moves back to the center, again there is no significant movement of the left eye. I wonder if the authors actually looked behind the cover of their eye-movement recording device to observe the left eye. It is difficult to believe that the left eye did not move while the right eye was making 10-degree saccades to the right and back to primary again. This tracing was performed on the orthotropic day, when there was no manifest deviation.

This same type of eye movement was recorded on the esotropic day (depicted in Figure 3). On the top portion of this tracing, again with the right eye viewing, the left eye makes a conjugate movement to the right as the right eye changes fixation from primary to 10 degrees right. The left eye appears to make a larger movement than the right eye and also a larger movement to refixate back towards the primary position.

This discrepancy is hard to understand and I wonder whether the tracing in the middle of Figure 2 is not an artifact. I performed saccadic velocity tracings on a patient with cyclic esotropia[1] and found that conjugate movements were made with each eye with voluntary, horizontal saccades, no matter which eye was doing the fixing. It is difficult to understand why the left eye would not move to the right (Herring's Law) as the right eye made voluntary horizontal saccades.

Another question concerns Figure 3. On the bottom of this tracing, one done on the esotropic day, the left eye is said to do the viewing and the right eye to be 10 degrees esotropic. However, the tracing shows both eyes starting in the zero position; the right eye is not esotropic. Was this tracing incorrectly labeled? Is this the correct tracing? The tracing as it appears in THE JOURNAL and as it is described in the legend cannot be correct.

HENRY S. METZ, M.D.
Rochester, New York

REFERENCE

1. Metz, H. S., and Jampolsky, A.: Alternate day esotropia. Am. J. Pediatr. Ophthalmol. Strabismus 16:40, 1979.

Reply

Editor:

We agree with Dr. Metz that the eye movements of our patient with acquired cyclic esotropia were unusual. This is why we felt that they were worth reporting. We will discuss his points in order.

The patient's eyes did do what Figure 2 illustrates. The absence of movement in the left eye during right gaze is not an artifact; for larger rightward movements some left adduction was observed. We do find this remarkable behavior in an orthotropic patient. Again, on the patient's esotropic day, the left eye did make hypermetric movements while covered. It also tended to drift while covered.

We agree that the reason for this discrepancy is not immediately apparent. It does not follow, however, that it is an artifact. Unfortunately, the paper by Drs. Metz and Jampolsky was not available to us during the preparation of our manuscript. The eye movements of his patient and ours evidently differed; so, however, do their clinical findings. Ours was a

unique case of alternating esotropia acquired in adulthood and, as such, may well represent a distinct clinical entity. The violation of Hering's Law does not surprise us, as this is a not infrequent finding in nystagmus and strabismus patients.

There is, indeed a drafting error in Figure 3. The zero line for the right eye should be 10 degrees further right. The legend is correct; the eye is esotropic.

LARRY A. ABEL, PH.D.
B. T. TROOST, M.D.
Cleveland, Ohio

Incision Size Needed for the Kelman Intraocular Lens

Editor:

My colleague J. J. Kanski recently recorded an instructional tape on intraocular lenses and sent it to me for my comments. When I listened to it I heard the statement that "the Kelman lens can be introduced into the eye through an incision as small as 3 mm." I questioned this on the grounds that it must be impossible to introduce a rigid object with a diameter of 5 mm into the anterior chamber through an incision 3 mm wide. In reply Mr. Kanski drew my attention to the article, "The changing scene of intraocular implant lens surgery" (Am. J. Ophthalmol. 88:819, 1979), in which N. S. Jaffe says just that. Dr. Jaffe goes on to say that the anterior chamber angle-supported lens "can be used with either an intra- or extra-capsular cataract extraction—This is a significant advantage because it requires the surgeon to change his method of cataract extraction."

Surely what he meant to say was, "because it does not require the surgeon to change his method of cataract extraction."

The reputation of THE JOURNAL is such that statements made in it, particularly by authors as respected as Dr. Jaffe, tend to be accepted as gospel and copied from publication to publication and tape to tape.

Just for the record, there are two versions of the Choyce Mk. IX implant—the 5-mm version that goes in easily enough through a 6-mm incision and the 4-mm version that goes in through a 5-mm incision.

D. P. CHOYCE, M.S., F.R.C.S.
Essex, England

Reply

Editor:

I agree with Dr. Choyce that it is impossible to introduce a currently used Kelman intraocular lens through an incision as small as 3 mm, but I remind him that the original Kelman intraocular lens had a much smaller diameter. I can introduce a posterior chamber intraocular lens 6 mm in diameter through a 6-mm incision. This is possible because there is a certain amount of give to the margins of the incision, permitting a rigid object to pass through.

There is also the problem of the nomenclature. The width of an incision often means the chord length of the incision. This may be considerably different from the arc length of the incision. If we speak of the size of the incision according to the number of clock hours, the arc and chord lengths of an incision can vary greatly depending on the location of the incision. Obviously, in posterior incisions these lengths are greater than in anterior incisions.

My personal view is that there is little to be gained by speaking of a 4-mm incision as having a significant advantage over one of 6 mm.

Finally, Dr. Choyce is correct about

the error in the sentence he quotes. It should read "does not require."

NORMAN S. JAFFE, M.D.
Miami, Florida

BOOK REVIEWS

Orbital Tumors, 2nd ed. By John W. Henderson. New York, Brian C. Decker Division of Thieme-Stratton Inc., 1980. Hardcover, 607 pages, index, 347 black and white figures, 13 color figures. $70

It was a pleasure to read the second edition of Dr. Henderson's monumental work on orbital tumors. The book is organized into three sections. The first covers applied anatomy and diagnostic modalities, the second individual tumors, and the third surgical approaches to orbital tumors. In the first chapter, Dr. Henderson provides the reader with an adequate review of the orbital anatomy and frequently interjects pertinent clinical points related to the anatomy being discussed. The second chapter, which considers diagnostic approaches to orbital tumors, contains a new section on computed tomography. Here, Dr. Henderson briefly presents the basic aspects of this technique and adequately describes the computed tomographic features of the more common orbital tumors.

In Chapter 3, Dr. Henderson classifies the various orbital tumors and discusses their relative incidence, based upon his personal experience with 764 consecutive cases. This gives the reader a framework for categorizing the specific tumors to be discussed later. In Chapters 4 to 23, Dr. Henderson discusses the clinical and pathological features of the many tumors and pseudotumors that involve the orbit. In these comprehensive chapters, he provides a detailed account of the more common orbital tumors but manages to include adequate information on rare orbital conditions as well.

The final three chapters on surgical approaches to orbital tumors are well written, well illustrated, and valuable to the ophthalmologist, neurosurgeon, maxillofacial surgeon, and other specialists. I was personally disappointed that the chapter on orbital exenteration, present in the first edition, was not included in this second edition. Dr. Henderson informed me that it was deleted because of cost and space factors and because the technique of exenteration has been adequately discussed in other publications.

In the preface, Dr. Henderson outlines his reasons for writing this second edition. These included the increasing use of computed tomography in orbital diagnosis, the use of immunochemistry in the diagnosis of lymphoid tumors, changes in the recommended treatment of rhabdomyosarcoma, the increased recognition of "newer" orbital tumors, and the increasing role of electron microscopy in the diagnosis of some tumors of equivocal origin.

For the most part, he has succeeded in blending these new concepts into the body of the text. The computed tomographic features of many tumors are included and the changing concepts of lymphoid tumors are mentioned, although further amplification of these concepts would have been desirable. The treatment of orbital rhabdomyosarcoma has been updated. Some appropriate comments on electron microscopy have been included, although a few electron photomicrographs would have been helpful.

Dr. Henderson's method of presenting

the bibliography at the end of each chapter is superb. He has grouped the references under each specific tumor and thus provided a pertinent minibibliography for each given subject. This gives the reader who wishes to review a specific condition a readily available source for more detailed study.

We should all be grateful to Dr. Henderson for providing this second edition. His vast experience in the diagnosis and treatment of orbital tumors is reflected throughout this text. Every practitioner who encounters diseases of the orbit should have it as a reference source. This updated second edition will most certainly insure that this book will continue to be an authorative treatise on orbital tumors for many years.

JERRY A. SHIELDS

Textbook of Veterinary Ophthalmology. Edited by Kirk N. Gelatt. Philadelphia, Lea & Febiger, 1981. Hardcover, 788 pages, index, approximately 1,000 black and white figures. $85

This is an encyclopedic treatise prepared by 22 contributors. Over 200 pages are devoted to embryology, anatomy, physiology, pharmacology, and genetics as those disciplines relate to animal ophthalmology. Much of the book (more than 250 pages) is devoted to the eye of the dog. This compares with about 35 pages for the horse and little more than 20 pages for all of the commonly used laboratory animals. This probably reflects the scarcity of published studies in those categories and not a bias on the part of the editor.

The chapter on genetics is weak, as is the one on laboratory animals. The coverage on the dog is exhaustive and the chapters on anatomy and physiology are excellent. The comparisons of the anatomy and physiology of the eyes and associated structures of many animals and birds are informative.

The treatise will probably be more used as a reference than as a textbook. Those laboratories using dogs for long-term ophthalmologic studies will find it valuable. The veterinary practitioner interested in dogs will also find it helpful.

The illustrations, with a few exceptions, are of excellent quality. Some of the color reproductions of the eyes of a variety of animals and birds and of several diseases are particularly good. The editing is good and the lack of uniformity seen in books with many contributors is not noticeable. It is a well-written book that fills a special need in veterinary and comparative medicine.

JOHN H. RUST

Journal of Clinical Neuro-ophthalmology, Vol. 1, No. 1. Editor-in-Chief, J. Lawton Smith. New York, Masson Publishing USA, Inc., 1981. Published quarterly. Annual subscription, $49; introductory subscription for residents, interns, and medical students, $24.50

The aim of this new publication, according to its editor, is to cover in one journal our expanding knowledge in all areas of the neurologic and ophthalmologic sciences. The editor hopes to include not only ophthalmology and neurology, but also neurosurgery, neuroradiology, neuropathology, and neuroanatomy. The emphasis will be on diagnosis and office management of medical and surgical therapies.

The editor promises some refreshing innovations. This journal uses almost no abbreviations and prefers drugs to be called by their trade names rather than

their nonproprietary names. Visual acuities are measured in feet. There is a Neuroradiologic Clinical Pathologic Correlation section, and the first issue contains a good editorial on myasthenia gravis by Ronald M. Burde. The abstracts section, called Information Summaries, by John A. McCrary opens with a one-sentence summary of each abstract. (The summaries, but not the corresponding abstracts, are numbered). Each abstract includes the address of the corresponding author (with the zip code). The Editor provides a short overview section called the New Pearls Checklist.

This journal appears to be promising, and we look forward with interest to future issues.

FRANK W. NEWELL

ABSTRACT DEPARTMENT

EDITED BY DAVID SHOCH, M.D.

Acta Dermato-Venereologica

GLAUCOMA AND AMAUROSIS ASSOCIATED WITH LONG-TERM APPLICATION OF TOPICAL CORTICOSTEROIDS TO THE EYELIDS. Vie, R. (Dept. Dermatol., School of Med., Univ. Bergen, Bergen, Norway). Acta Derm. Venereol. 60:541, 1980.

The production of glaucoma by the long-term use of corticosteroid drops has been well documented, but there are only rare reports of this developing as the result of ointments applied to the eyelids. The patient described here was a 29-year-old woman who had applied a 0.5% fluocortolone cream to her face, including the eyelids, daily for 15 years. She had never received any systemic corticosteroid therapy. When first seen for an eye examination, her intraocular pressures were R.E.: 43 mm Hg and L.E.: 63 mm Hg. The left eye was blind and the right eye showed a nasal step. Normal intraocular pressures were found in all of her relatives. The question arises as to whether the glaucoma was caused by absorption from the skin or whether enough ointment got into the conjunctival sacs and thus became absorbed directly into the eye. The authors favor the latter theory. (2 figures, 6 references)—David Shoch

FREQUENCY OF CATARACT IN ATOPIC DERMATITIS. Christensen, J. D. (Dept. Dermatol., Copenhagen County Hosp., DK-1353, Copenhagen, Denmark). Acta Derm. Venereol. 61:76, 1981.

Atopic cataract has been reported to occur in any where from 0.5% to 20% of patients with atopic dermatitis. The author examined 51 patients with atopic dermatitis and did not find a single case of atopic cataract. However, his series consisted of young patients, the youngest being 16 years old and the oldest 39 years old, with a mean age of 24.6 years. One may reasonably conclude that if there is an association of cataracts with atopic dermatitis it occurs in an older group. (1 table, 11 references)—David Shoch

Acta Ophthalmologica

DISC HEMORRHAGE AND GLAUCOMA. Bengtsson, B., Holmin, C. and Krakau, C. E. T. (Vårdcentralen, S-240 10 Dalby, Sweden). Acta Ophthalmol. 59:1, 1981.

A connection between optic disk hemorrhage and glaucoma was recognized nearly 100 years ago by Bjerrum. His finding has been reemphasized in recent years by Drance and now it is reasonable to consider that hemorrhages of the disk margin and glaucoma are the rule rather than the exception. The authors describe 51 patients with at least one disk hemorrhage. Hemorrhage was connected with the progression of field defects, and they are detectible very early in the glaucomatous process. The phrase "forerunner of glaucoma" is most likely justified. Initially these hemorrhages tend to appear at the poles of the disk where later notches tend to appear. Later on, the hemorrhages tend to be located near the equator and therefore the authors conclude that hemorrhages indicate the location of subsequently visible tissue destruction. (8 figures, 14 references)—David Shoch

Annals of Neurology

TREATMENT OF PERIODIC ALTERNATING NYSTAGMUS. Halmagyi, G. M., Rudge, P., Gresty, M. A., Leigh, R. J., and Zee, D. S. (Clin. Neurophysiol. Dept., Royal Prince Alfred Hosp., Camperdown 2050, NSW, Australia). Ann. Neurol. 8:609, 1980.

Periodic alternating nystagmus is a movement disorder that causes oscillopsis and degrades vision. It has been associated with lesions of the vestibular nuclei and cerebellum and has been reported in patients with multiple sclerosis, posterior fossa malformations, and spinocerebellar degeneration. It is thought to be the result of a failure of the oculomotor inhibitory system, perhaps origi-

nating in the cerebellum. Many drugs have been tried to treat this entity without success. It has been reported that intravenous chlorpromazine or barbiturate abolishes the nystagmus transiently. The authors tried baclofen (beta-parachlorophenyl gamma-aminobutyric) in three patients. Two responded well, with the drug inhibiting the nystagmus; the third patient did not respond well. The mechanism of action of this drug is probably the inhibition of the release of the excitatory neurotransmitter glutamate. (2 figures, 18 references)—David Shoch

PAINFUL OPHTHALMOPLEGIA (TOLOSA-HUNT) ACCOMPANIED BY PERIPHERAL FACIAL PARALYSIS. Vallat, J. M., Vallat, M., Julien, J., Duman, M., and Dany, A. (Depts. Neurol., Ophthalmol., and Neurosurg., C.H.U. Dupuytren, 87 031 Limoges, France). Ann. Neurol. 8:645, 1980.

The Toloso-Hunt syndrome is a painful unilateral ophthalmoplegia involving any one of the third, fourth, or sixth nerves. It appears to be caused by an inflammatory process since most cases respond rather dramatically to the administration of corticosteroids. The authors describe three patients who had facial palsies in addition to their ophthalmoplegia. They believe, therefore, that the Toloso-Hunt syndrome may actually be part of a more generalized cranial polyneuropathy rather than a localized phenomenon. (4 references)—David Shoch

"WRONG-WAY EYES" IN SUPRATENTORIAL HEMORRHAGE. Pessin, M. S., Adelman, L. S., Prager, R. J., Lathi, E. S., and Lange, D. J. (Dept. Neurol., Tufts-New England Med. Ctr. Hosp., 171 Harrison Ave., Boston, MA 02111). Ann. Neurol. 9:79, 1981.

It is generally accepted that supratentorial hemorrhages may produce contralateral hemiplegia with conjugate horizontal eye deviation away from the paralysis and toward the side of the lesion. By contrast, in pontine lesions the eyes may conjugately deviate toward the side of the hemiplegia. The patient described here developed conjugate horizontal eye deviation to the side opposite a large frontal perisylvian hemorrhage from a ruptured aneurysm. Autopsy examination confirmed the hemispheric locus of the hemorrhage and failed to disclose any lesion of the brain stem or thalamus that might explain the "wrong-way" eye deviation. (2 figures, 6 references)—David Shoch

British Journal of Ophthalmology

A COMPARISON OF ACYCLOVIR AND IDOXURIDINE AS TREATMENT FOR ULCERATIVE HERPETIC KERATITIS. Coster, D. J., Wilhelmus, K. R., Michaud, R., and Jones, B. R. (Dept. Clin. Ophthalmol., Moorfields Eye Hosp., City Road, London EC1V 2PD). Br. J. Ophthalmol. 64:763, 1980.

Sixty patients with herpetic keratitis were classified by the type of ulcer. They were then treated in a double-masked fashion with either 3% acyclovir ointment or 1% idoxuridine ointment. All of the patients treated with idoxuridine healed, as did all the patients treated with acyclovir. There was no difference in the healing time between the two agents. (3 figures, 1 table, 5 references)—E. Lee Stock

RANDOMISED DOUBLE-BLIND TRIAL OF ACYCLOVIR AND IDOXURIDINE IN DENDRITIC CORNEAL ULCERATION. Collum, L. M. T., Benedict-Smith, A., and Hillary, I. B. (9 Fitzwilliam Pl., Dublin 2 Ireland). Br. J. Ophthalmol. 64:766, 1980.

The results of a randomised double-blind clinical trial of 3% acyclovir and 0.5% idoxuridine ophthalmic ointments in 60 patients with corneal dendritic ulceration are presented. Ulcers in all 30 patients treated with acyclovir healed compared with 22 (76%) of 29 patients treated with idoxuridine ($P < .01$). Patients treated with acyclovir healed more rapidly (average, 4.4 days) than those who received idoxuridine (average, 9.2 days) ($P < .01$). No serious side effects were observed, though transient stinging was recorded in eight patients receiving acyclovir and in two patients receiving idoxuridine. Other side effects in the idoxuridine-treated group were watering in two patients and superficial punctate erosions in six patients. (1 figure, 4 tables, 19 references)—Authors' abstract

Chinese Medical Journal

PATHOLOGIC CLASSIFICATION OF MEIBOMIAN GLAND CARCINOMAS OF EYELIDS: CLINICAL AND PATHOLOGIC STUDY OF 156 CASES. Ni, C., and Guo, B. K. (Ophthalmol. Dept., Eye, Ear, Nose and Throat Hosp., Shanghai First Med. Coll., Shanghai, China). Chinese Med. J. 92:671, 1979.

The very large series of eyelid carcinomas reported here has the additional interest of a long-term follow-up on 100 of the patients. The most salient finding was that of the 100 patients followed up, 41 died of the tumor. Death resulted from lymphatic and blood-borne metastases. This high mortality of 41% certainly makes meibomian gland carcinoma one of the most malignant tumors of the eyelids. The histologic structure of the tumor is extremely variable. It may simulate squamous cell carcinoma, basal cell carcinoma, adenocarcinoma, or spindle-cell carcinomas. In dealing with this large number of cases, the authors evolved a tentative pathologic classification of this tumor into five different types: the differentiated, the squamous cell, the basal cell, the adenoid, and the spindle cell. The differentiated was the least malignant while the basal cell and squamous cell types were the most malignant. The adenoid and spindle cell types were too few to warrant any conclusions. (9 figures, 3 tables, 3 references)—Luke H. Liu

Journal of the American Medical Association

TIMOLOL-INDUCED BRONCHOSPASM IN ASTHMATIC BRONCHITIS. Schoene, R. B., Martin, T. R., Charan, N. B., and French, C. L. (Div. Respiratory Dis., Univ. Washington, RM-12, Seattle, WA 98195). J.A.M.A. 245:1460, 1981.

Aggravation of pre-existing asthma by topical timolol has been reported on numerous occasions. The authors document this problem in a controlled fashion. Two groups of patients, five with no history of obstructive lung disease and five with a documented history of chronic obstructive pulmonary disease, were given eyedrops consisting either of saline or of timolol maleate in a double-masked study. The control subjects showed no decrease in forced expiratory volume with either eyedrop. Conversely, the five patients with chronic pulmonary obstruction showed a 20% to 30% decrease in forced expiratory volume after the administration of timolol but not after saline eyedrops. If timolol is necessary in a particular patient who has some history of respiratory difficulty, it might be wise to obtain a spirometric evaluation before and after topical timolol administration. (1 figure, 1 table, 5 references)—David Shoch

Lancet

HYPERORNITHINAEMIA AND GYRATE ATROPHY OF THE RETINA: IMPROVEMENT OF VISION DURING TREATMENT WITH A LOW-ARGININE DIET. McInnes, R. R., Bell, L., Arshinoff, S. A., Marliss, E. B., and McCulloch, J. C. (Depts. Pediatr., Ophthalmol., and Med., Univ. Toronto and the Hosp. for Sick Children, Toronto, Ontario, Canada). Lancet 1:513, 1981.

A 15-year-old patient with hyperornithinemia and gyrate atrophy of the retina was given a low-arginine diet to reduce plasma ornithine to a concentration near the normal range. After five weeks of treatment, there was improvement in the visual function of one eye which had been severely impaired without improvement for three years. This improved visual function was maintained until compliance with the diet deteriorated at eight months, after which visual function regressed towards pretreatment status. Over-restriction of dietary arginine produced hyperammonemia, indicating that arginine is an essential aminoacid in ornithine-transaminase deficiency. These results suggest that reduction of plasma ornithine may reverse a metabolic neuroretinopathy in this disease, and offer hope that progression of the retinal atrophy can be arrested as well. (3 figures, 1 table, 15 references)—Authors' abstract

Laryngoscope

SPHENOETHMOID APPROACH TO THE OPTIC NERVE. Sofferman, R. A. (One

South Prospect St., Burlington, VT 05401). Laryngoscope 91:184, 1981.

The classic surgical approach to the canalicular segment of the optic nerve is via transfrontal craniotomy. More recently, an external ethmoid technique has been used to provide limited extracranial access to the optic canal. The author suggests using a modification of the transsphenoidal approach now commonly used for the treatment of pituitary tumors causing visual field defects. This technique allows exposure of the optic nerve from the orbital apex to the optic chiasm. The approach is by a Caldwell-Luc incision and the technique is obviously contraindicated in cases where a vascular lesion is suspected. (9 figures, 29 references)—David Shoch

Medical Journal of Australia

RETROLENTAL FIBROPLASIA TODAY. Stark, D. J., Manning, L. M., and Lenton, L. (40 Annerley Rd., Wooloongabba 4061, Australia). Med. J. Aust. 1:275, 1981.

The incidence of retrolental fibroplasia is increasing as more premature infants of lower birthweight are surviving. Sophisticated monitoring techniques have not eliminated the problems of hyperoxia. Infants at risk need heightened vigilance by pediatricians monitoring oxygen levels, together with ophthalmologic review. Placing arbitrary ceilings on oxygen concentrations would increase morbidity and mortality. (1 figure, 25 references)—Ronald Lowe

New England Journal of Medicine

SUPPLEMENTARY CREATINE AS A TREATMENT FOR GYRATE ATROPHY OF THE CHOROID AND RETINA. Sipilä, I., Rapola, J., Simell, O., and Vannas, A. (Children's Hosp., Univ. Helsinki, SF-00290, Helsinki 29, Finland). N. Engl. J. Med. 304:867, 1981.

Gyrate atrophy of the choroid and retina is a disease characterized by progressive constriction of visual fields, a 10-fold to 20-fold elevation in plasma ornithine, and depressed activity of L-ornithine:2 oxoacid aminotransferase. Morphologically conspicuous but clinically unimportant atrophy of Type II muscle fibers progresses concomitantly with the eye disease. A pathogenic component of the disease may be deficient formation of creatine, caused by hyperornithinemia, which leads to a shortage of cellular phosphocreatine energy stores. To test the therapeutic value of replenishing the postulated deficiency of creatine, we supplemented the diet of seven patients with 1.5 g of creatine daily. During one year of this treatment the diameters of Type II muscle fibers increased from 34.1 ± 7.1 to 49.9 ± 7.0 μm (mean ± S.D.) ($P < .001$). There was no significant increase in the diameters of Type I fibers. The visual-field tests showed no further constriction during the therapy. Fundus photography revealed slow impairment at an age otherwise associated with rapid progression of the disease. (3 figures, 1 table, 14 references)—Authors' abstract

CONJUNCTIVITIS IN HUMAN BEINGS CAUSED BY INFLUENZA A VIRUS OF SEALS. Webster, R. G., Geraci, J., Petursson, G., and Skirnisson, K. (St. Jude Children's Res. Hosp., Memphis, TN 38101). N. Engl. J. Med. 304:911, 1981.

Harbor seals are susceptible to an influenza A virus which causes an acute hemorrhagic pneumonia. In December 1979, about 500 seals were found dead on Cape Cod of this disease. During the studies, four people involved in autopsies contracted purulent conjunctivitis. No viral cultures were obtained at that time but during experimental work with these harbor seals an investigator was exposed and developed a severe conjunctivitis and viral cultures were obtained which were identified as influenza A virus. The present case report indicates that influenza virus from seals also has the ability to replicate in human tissues. (3 references)—David Shoch

Plastic and Reconstructive Surgery

NUMBNESS AFTER BLEPHAROPLASTY: THE RELATION OF THE UPPER ORBITAL FAT TO SENSORY NERVES. Klatsky, S., and Manson, P. N. (Hilton Inn Plaza, 1726 Reisterstown Rd., Baltimore, MD

21208). Plast. Reconstr. Surg. 67:20, 1981.

About 1% of a group of 402 patients undergoing blepharoplasty complained of numbness after their surgery. The complaints were transient and had all resolved at the end of six months. The numbness apparently results from nerve injury to terminal branches of the ophthalmic division of the trigeminal nerve. These branches are found superior and superomedial to fat in the upper orbital fat compartments. Nerve injury has many mechanisms and can be avoided by recognition of the proximity of sensory nerve fibers to orbital fat. (1 figure, 18 references)—David Shoch

SEPARATE SKIN AND MUSCLE FLAPS IN LOWER-LID BLEPHAROPLASTY. Klatsky, S. A., and Manson, P. N. (Hilton Inn Plaza, 1726 Reisterstown Road, Baltimore, MD 21208). Plast. Reconstr. Surg. 67:151, 1981.

In most cases of repair of "bags" of the lower eyelids it is sufficient to excise the redundant skin and any herniated fat. However, the authors point out that in many cases there is also a thinning and elongation of the muscle fibers of the orbicularis and that a better result can be obtained if a separate flap is made of the orbicularis and shortened appropriately. The authors dissect the orbicularis free and excise a portion near the lateral canthus and stretch the fibers to fill this defect. This is combined with the usual skin and fat procedures. It appears to be especially effective in the management of atrophic wrinkled or deeply creased skin. (9 figures, 14 references)—David Shoch

A SIMPLE POSTOPERATIVE BLEPHAROPLASTY DRESSING. Gum, R. A., Shively, R. E., Northington, J. W., and Williamson, G. B. (Plast. Surg. Ser., William Beaumont Army Med. Ctr., El Paso, Texas 79920). Plast. Reconstr. Surg. 67:234, 1981.

Many surgeons used continuous iced saline compresses over the eyes after blepharoplasties. Many patients object to this because it obviously interferes with their ability to see and makes the postoperative period unpleasant. The authors have cut out the center of an ordinary eye pad and used the resultant ring as an iced saline compress. This allows the patient to see normally after surgery. (2 figures)—David Shoch

Quarterly Journal of Experimental Physiology

AXON REFLEX IN OCULAR INJURY: SENSORY MEDIATION OF THE RESPONSE OF THE RABBIT EYE TO LASER IRRADIATION OF THE IRIS. Butler, J. M., Unger, W. G., and Cole, D. F. (Dept. Exp. Ophthalmol., Inst. Ophthalmol., Judd St., London, WC1H 9QS, U.K.). Q. J. Exp. Physiol. 65:261, 1980.

Laser irradiation of the iris produces a prolonged miosis, uveal vasodilation, and a breakdown of the blood-aqueous barrier. This response has been attributed partly to prostaglandin mediation and partly to mediation by a noncholinergic nervous pathway thought to be sensory in function. In order to evaluate these possibilities more accurately, the authors irritated the rabbit iris with a laser at specific intervals after diathermic coagulation of the epigasserian nerve tract. The intensity of the pupillary constriction was unaltered after 90 minutes but progressively decreased until after four days there was essentially no response to high-energy laser irradiation in the denervated eye. It is evident that manifestation of the irritation response of the iris is largely dependent upon the presence of intact and functional sensory nerves. They suggest that endogenous prostaglandins exert some, if not all, of their effects via sensory nerve endings. Those terminals that are directly stimulated, whether by laser irradiation or by prostaglandins formed during the injury, release some mediator to cause pupillary constriction. From these terminals impulses pass orthrodromically and antidromically by axon reflex to release further mediator from terminals in the region of the ciliary vessels or the major arterial circle. In this way the response is propagated and augmented. (4 figures, 2 tables, 38 references)—David Shoch

Trends in Neuroscience

THE DEVELOPMENT OF VISUAL ACUITY IN HUMAN AND MONKEY INFANTS. Teller, D. Y. (Depts. Psychol., and Physiol./Bi-

ophysics, Child Develop. and Mental Retardation Ctr., and Regional Primate Res. Ctr., Univ. Washington, Seattle, WA 98195). Trends Neurosci. 4:21, 1981.

With the development of a new technique called preferential looking, it is possible to evaluate the visual acuity of infants shortly after birth. These studies have shown that infants can resolve gratings of about two cycles per degree and reach standard adult visual acuity (about 30 cycles per degree) at about 30 months. It is also possible to apply these same techniques to monkeys who respond just as well except that they develop adult visual acuity much more quickly, in about 30 weeks. Thus macaque monkey infants can provide an outstanding animal model for the development of human visual acuity. It should be possible to correlate behavorial with physiologic and anatomic development in infant monkeys under both normal and abnormal conditions and to use these data to evaluate models of the substrates of human vision. (3 figures, 12 references)—David Shoch

NEWS ITEMS

Send News Items double-spaced to News Editor, American Journal of Ophthalmology, Tribune Tower, Suite 1415, 435 N. Michigan Avenue, Chicago, Illinois 60611

For adequate publicity, notices of postgraduate courses, meetings, and lectures must be received at least six months before the date of occurrence.

VIII Instituto Barraquer International Course of Ophthalmology

The VIII Instituto Barraquer International Course of Ophthalmology will be held May 16–21, 1982, in Barcelona, Spain. For further information, write Instituto Barraquer, Laforja 88, Barcelona 21, Spain.

National Eye Institute: Fellowship in Experimental Ophthalmic Pathology

The National Eye Institute's Intramural Research Program announces a fellowship in experimental ophthalmic pathology aimed at applying various histologic and electron microscopic techniques to the understanding of disease. The NEI pathology staff includes Merlyn Rodrigues, Toichiro Kuwabara, and David G. Cogan. Preference will be given to applicants who have had training in ophthalmic pathology, histologic techniques, and electron microscopy. For further information, write Scientific Director, National Eye Institute, NIH, Bethesda, MD 20205.

Wayne State University Kresge Eye Institute: Symposium on Diagnosis and Therapy of Glaucomas

Kresge Eye Institute, Department of Ophthalmology, Wayne State University School of Medicine, will present a symposium on the diagnosis and therapy of glaucomas, Sept. 12, 1981. For further information, write Dong H. Shin, M.D., Kresge Eye Institute, 3994 John R, Detroit, MI 48201.

American College of Veterinary Ophthalmologists: Annual Scientific Meeting

The American College of Veterinary Ophthalmologists will hold its annual scientific meeting Nov. 1 and 2, 1981, in Atlanta, Georgia. For further information, write Dr. Charles L. Martin, Dept. of Small Animal Medicine, University of Georgia, Athens, GA 30602.

University of Pennsylvania-Scheie Eye Institute: Anterior Segment Ocutome/Fragmatome Workshop

The Scheie Eye Institute, Department of Ophthalmology, University of Pennsylvania, will sponsor an anterior segment Ocutome/Fragmatome system symposium and workshop, Nov. 19 and 20, 1981, in Philadelphia. The fee is $450 for physicians; $150 for lectures without workshop. For further information, write Alexander J. Brucker, M.D., Course Director, Scheie Eye Institute, 51 N. 39th St., Philadelphia, PA 19104.

United States Pharmacopeial Advisory Panel: New Members

Herbert E. Kaufman, M.D., chairman of the Ophthalmology Advisory Panel, announced the following new members of the United States Pharmacopeial Advisory Panel for the 1980–1985 term: Jules Baum, Bernard Becker, Robert P. Burns, W. Morton Grant, Howard M. Leibowitz, Irving H. Leopold, Thomas J. Mikkelson, Steven M. Podos, and Thomas J. Zimmerman.

INSTRUCTIONS TO AUTHORS

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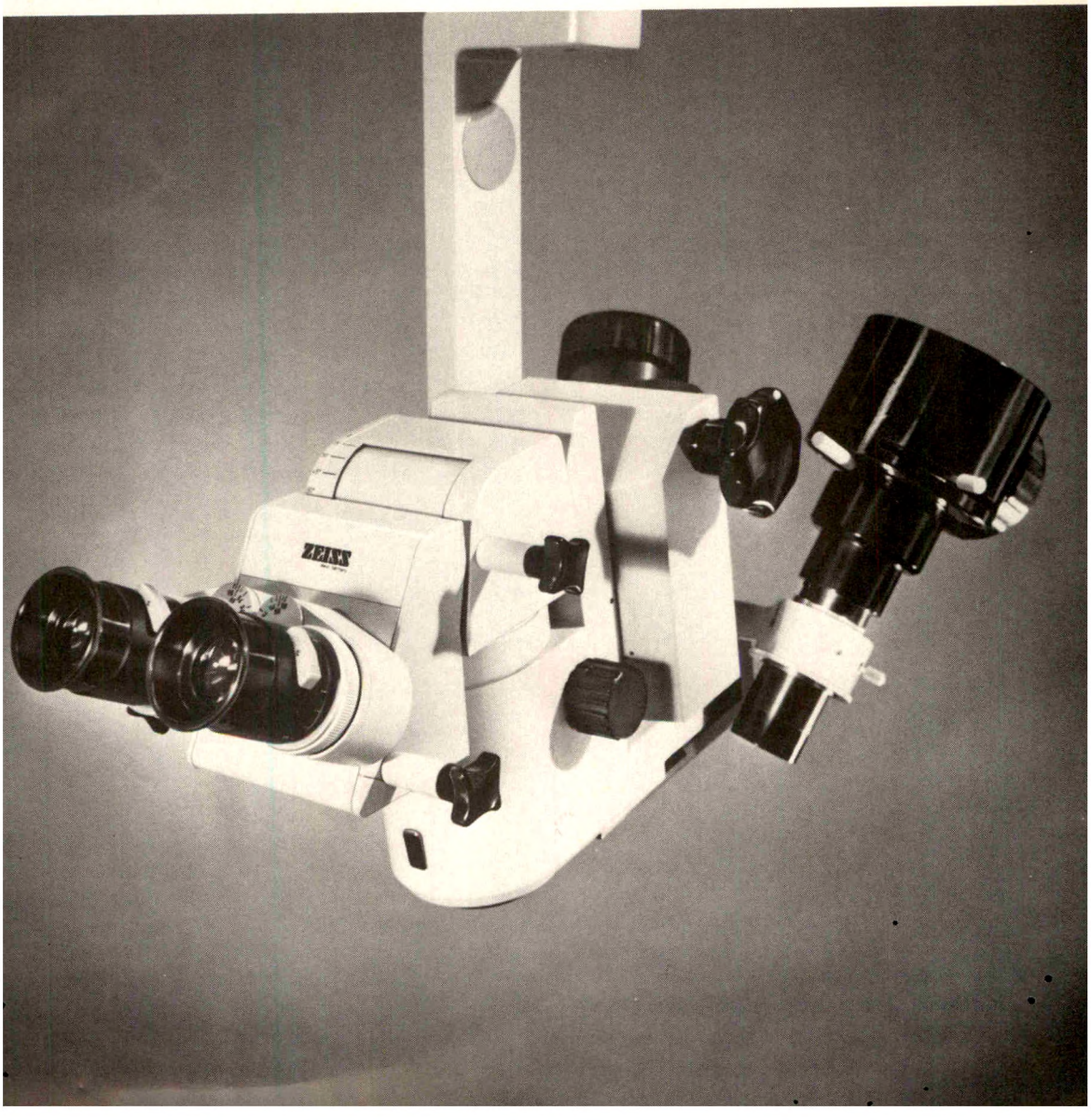
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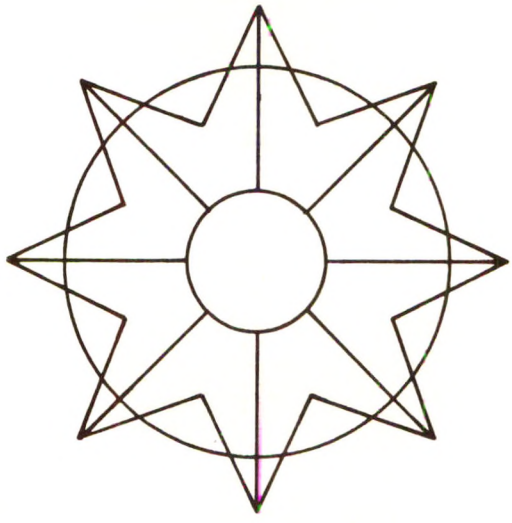
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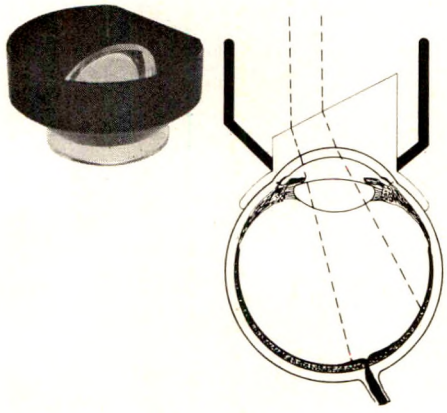
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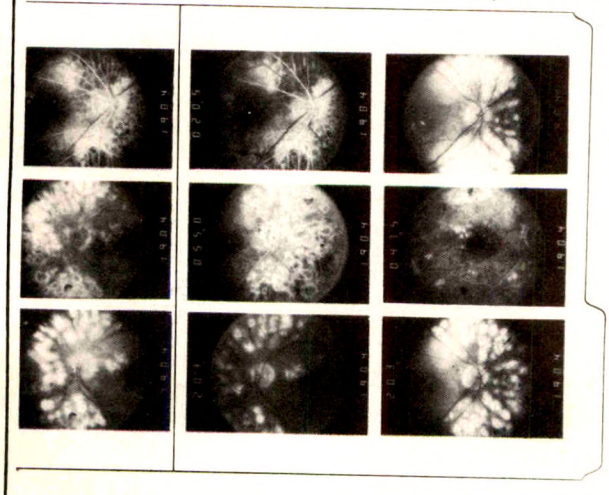
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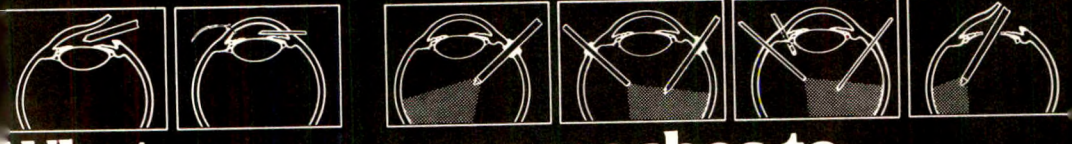
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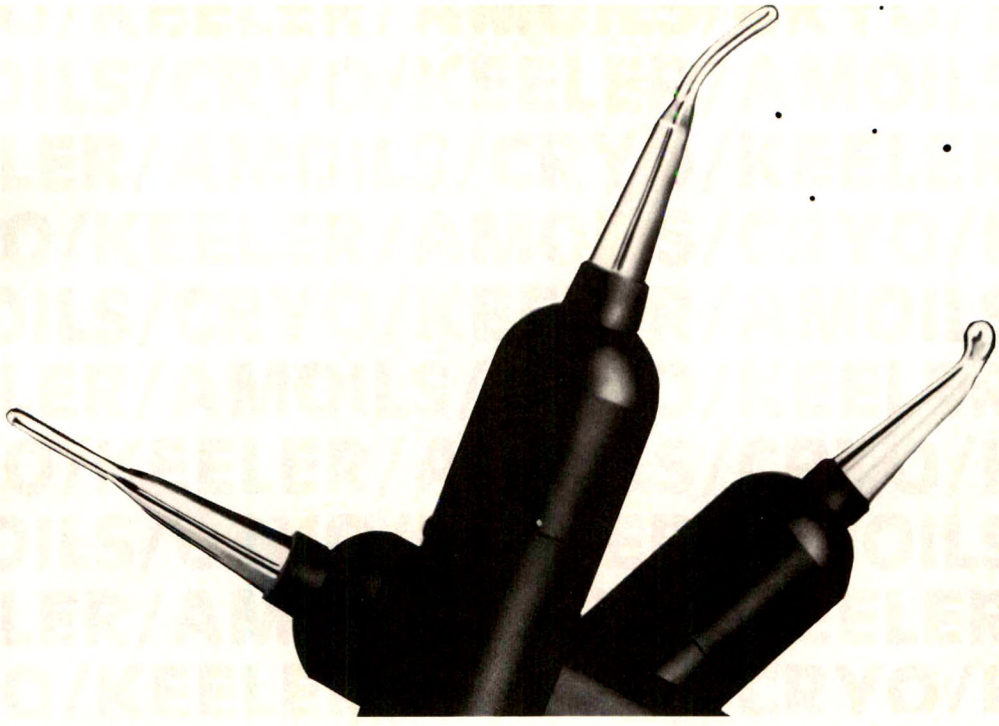
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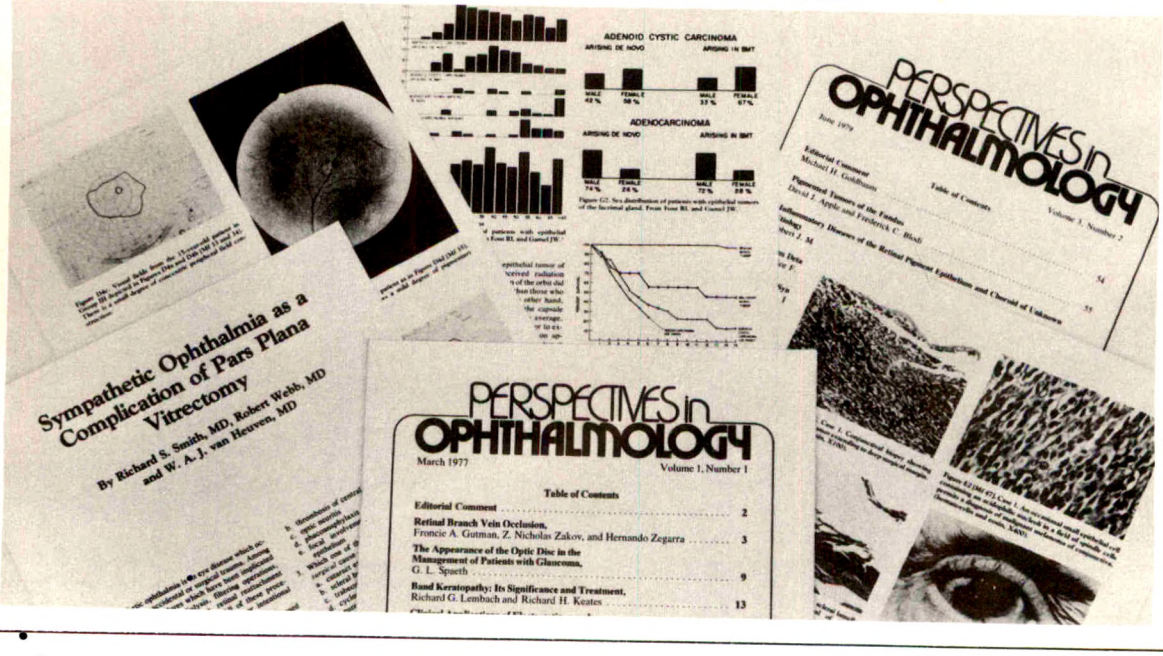
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